# Roberts and Hedges

# Chapter 1 - Basic Airway Management and Decision-Making

**Joseph E. Clinton** , **John W. McGill**

Airway management is widely preached as the first priority in the management of any seriously ill or injured patient. However, despite the lip service given to the importance of airway management, it is often overlooked and, consequently, can be a source of error in the care of the critically ill or injured patient. Although appropriate airway management is evident in all smoothly run resuscitations, inappropriate management often presages a cycle of patient deterioration and misguided therapeutic intervention.

Unfortunately, recognition of the need for airway management is only the first part of the problem in emergency resuscitation. Managing the airway may be one of the most difficult aspects of the entire resuscitation. Because of the sheer variety of airway difficulties possible, even the most skilled resuscitator can find the task challenging. Blood, loosened teeth, vomitus, swollen or distorted landmarks--all of these present formidable barriers to successful management. When obstruction occurs in conjunction with reflex clenching of the jaws and possible cervical spine injury, conventional airway management tools may be rendered useless. Time pressures imposed by the need to avoid cerebral anoxia force one to make difficult decisions concerning the use of risky maneuvers such as moving the neck, administering paralyzing agents, or using invasive procedures. Tools must be at hand, skills must be well practiced, and decision-making must be sharp if optimal emergency airway management is to occur.

Some solutions to the dilemmas faced in emergency airway management are presented in this and the following chapters. Minimally invasive approaches to airway establishment and emergency airway decision-making are described first in the current chapter with more advanced technique descriptions following in Chapters 2 through 4 . Decision algorithms are presented to help assemble the pieces of the airway management puzzle into a logical framework. Readers are encouraged to study the algorithms at their leisure to facilitate later decision-making when time is limited.

## DECISION-MAKING IN AIRWAY MANAGEMENT

The resuscitator must have many tools at hand to deal with the acutely compromised airway. Although proficiency may exist for all of the available maneuvers, the specific procedural choice often must be made in challenging circumstances. Rescuers should practice potential scenarios before facing actual airway management scenarios in the clinical situation. Failure to do so may lead to unnecessarily aggressive management in some situations or to irreversible hypoxic injury as a result of unnecessary hesitation in others.

Several parameters must be assessed quickly before an airway management choice can be made. The parameters to be considered are as follows:

1. Adequacy of current ventilation
2. Time of hypoxia
3. Patency of airway
4. Need for neuromuscular blockade (muscle tone, teeth clenching, severe obstructive pulmonary disease or asthma)
5. Cervical spine stability
6. Safety of technique and skill of operator

Consideration of these factors should guide a choice among those techniques described. This initial choice is often straightforward. Difficulty rises precipitously when the initial choice fails. Time becomes critical and safety of technique less important as the risk of irreversible hypoxic injury rises. Anxiety increases and error potential compounds under these circumstances. Forethought and practice are invaluable when making these decisions.

Schemata are offered in Figures 1-1 and 1-2 that outline the logic behind the airway choices. The first diagram (see Fig. 1-1) is the most complicated. It represents the choices among nonsurgical approaches to airway management that we have described. The end point of this diagram is either success or the decision to pursue surgical management. Note that time considerations may force a judgment in favor of surgical airway management when time is running out. The second diagram (see Fig. 1-2) is much simpler. Now that the decision to manage the airway surgically has been made, one need only choose among three available options. Consideration of patient condition, security of the airway approach, and invasive nature of the procedure are factors to be weighed in the final decision.

## ESTABLISHMENT OF AIRWAY PATENCY

The first concern in the management of a patient in critical condition is adequacy of the airway. Partial or complete airway obstruction must be overcome quickly. In some cases, such as an airway obstructed by a tongue, simple maneuvers will suffice. In other cases, particularly those in which myriads of obstructing agents are combining to block the airway, the task will be formidable. The tongue, dentures, swollen or distorted tissues, blood, and vomitus are common obstructing agents that make intubation difficult. Clearing obstructing agents may be made more difficult by muscular activity due to reflex stimulation or patient efforts to improve oxygenation. Moreover, the neck motion required for suction and intubation must be carefully managed in the face of potential cervical spine instability.

The wide availability of pulse oximetry monitors has greatly improved our ability to monitor oxygenation for patients at risk of airway or ventilatory compromise. [1] Clinically subtle deterioration is much more quickly and easily recognized using the monitors. They have become standard equipment in emergency departments, intensive care units, and operating rooms to allow early recognition of patient deterioration. Pulse oximetry is discussed in more detail in Chapter 6 .

**Airway Maneuvers**

Partial or complete airway obstruction resulting from *lax musculature* and *tongue occlusion* of the posterior pharynx may be overcome by a variety of maneuvers. The relative benefits of various airway-opening maneuvers have been examined. In a study of 120 anesthetized patients whose airways were obstructed by their tongues, Guildner compared the ease of performance of the neck-lift and head-tilt method, the jaw-thrust method, and the chin-lift method. He concluded that the chin lift was the easiest to perform and produced the greatest airway patency of the three methods tested. [2] Besides offering greater patency, the chin-lift method has the additional advantage that neck extension is unnecessary (Fig. 1-3 (Figure Not Available) , Tables 1-1 (Table Not Available) and 1-2) (Table Not Available) .

Partial airway obstruction in the patient with a decreased level of consciousness is commonly due to posterior displacement of the tongue. This may be recognized readily in the presence of snoring or stridor, but an apneic patient or one who is moving minimal air may not exhibit any audible evidence of airway obstruction. Some type of jaw-thrust or chin-lift maneuver should be performed on every unconscious patient to ensure airway patency. When uncertain about cervical spine status, the neck must be maintained in the neutral position. If the patient was found with a flexed or extended neck, the neck should first be restored to neutral position with gentle longitudinal traction. The chin-lift or jaw-thrust method is then performed. A combination of these maneuvers usually clears airways obstructed as a result of the position of the neck itself. The neck-lift and head-tilt maneuver, as described in cardiac life support courses, should not be used when cervical spine injury is suspected, because the extension of the spine produced during the maneuver endangers the spinal cord.

Clearing the airway of foreign material requires more than a simple jaw thrust. The occasional patient who presents with complete airway obstruction secondary to food aspiration may be treated with abdominal thrusts as described in basic cardiac life support. [3]

Partial or complete airway obstruction can be the result of upper airway hemorrhage, accumulation of the patient's own secretions, vomitus, or fractured dentition. When deciding on airway-clearing maneuvers, one must take these circumstances into account. Neck extension must be avoided or carefully minimized if the probability of a cervical spine injury is high. When stability of the spine is a concern, application of the abdominal thrust should be limited to the supine method described for unconscious victims. The abdominal thrust carries significant risks, compelling the rescuer to weigh the benefits of its application. [4]

**The Chin-Lift Maneuver**

The rescuer places the tips of the fingers, volar surface superiorly, beneath the patient's chin. The jaw is lifted gently forward. The patient's mouth is opened by drawing down on the lower lip with the thumb of the same hand. [2] Mouth-to-mouth resuscitation or other means of positive-pressure ventilation is provided if the patient is not ventilating

spontaneously.

**The Jaw-Thrust Maneuver**

The jaw-thrust maneuver is the second choice, again because neck extension is not necessary. Forward traction on the mandible is achieved by using two hands to grasp the mandibular rami and pull them forward.

**The Abdominal Thrust**

The abdominal thrust is a method to relieve a completely obstructed airway. The technique was popularized by Dr. Henry Heimlich and is commonly referred to as the *Heimlich maneuver*. The technique is most effective when a solid food bolus obstructs the larynx. Although a subject of controversy, a role for the maneuver has not been found for the resuscitation of near-drowning victims.

The conscious patient with an obstructed airway exhibits increased respiratory effort, anxiety, aphonia, and, occasionally, cyanosis. In the conscious patient, the maneuver is performed with the rescuer positioned behind the upright patient. The rescuer's arms are circled about the patient's midsection with the radial side of the clenched fist placed in the epigastrium of the patient. Care is exercised to position the fist midway between the umbilicus and the xiphoid of the patient. After proper positioning, the rescuer grasps the fist with the opposite hand and delivers an inward and upward thrust to the abdomen. A successful maneuver will cause the obstructing agent to be expelled from the patient's airway by the force of air exiting the lungs.

An unconscious, supine patient must be handled differently: the rescuer kneels next to the patient's pelvis facing cephalad. The palmar bases of the hands are placed in an overlapping fashion on the epigastrium at the same spot as that used in the upright patient. Inward, upward thrusts are delivered in this fashion with the same objective.

Abdominal thrusts are relatively contraindicated in pregnant patients and others with protuberant abdomens. A chest thrust similarly to that delivered in closed chest massage may be used instead. The upright patient may be delivered a chest thrust by placing the fist over the sternum. Experimental  primate models of infant airway obstruction show higher peak airway pressures with chest thrusts than with abdominal thrusts; a combined (simultaneous) chest and abdominal thrust produce even higher peak airway pressures. [6] Hence, a combined maneuver should be considered in the case of total airway obstruction that is unresponsive to simple abdominal thrusts.

Visceral injury can occur with the Heimlich maneuver. Excessive force may be responsible in such cases. In others, incorrect placement of the hands may play a role. Nonetheless, the technique can be life saving and should be used when needed. Attention to proper execution may limit complications.

# Positioning

Positioning the patient who has sustained multiple trauma can be a problem. Spinal injury and airway access priorities dictate that the patient should be kept in the supine position while immobilized on a backboard. Turning the patient on the side allows upper airway hemorrhage, secretions, and vomitus to drain externally rather than to collect in the patient's mouth, which can lead to aspiration and airway obstruction.

Guidelines for patient positioning must take into account the status of the patient's spine and the use of gravity to enable secretions to drain rather than accumulate in the airway. The following is a judicious approach to airway management in a patient *with spontaneous respiration*:

1. Initial airway maintenance accomplished by the chin-lift maneuver and the application of cervical stabilization (see Chapter 49) .
2. Immobilization of the patient on a spinal backboard.
3. With the position of the neck controlled, transportation of the patient on the side to facilitate airway drainage.

## Suctioning

Patient positioning and airway opening and clearing maneuvers are often inadequate to achieve the degree of airway patency desired. Ongoing hemorrhage, vomitus, and particulate debris often require suction to clear and maintain the respiratory passage. Three basic types of suctioning tips are available (Fig. 1-4) (Figure Not Available) . Each is suited to different types of airway obstruction problems.

*Dental tip* suction is most useful for clearing particulate debris from the upper airway. Vomitus is most readily cleared with this tip because it is least likely to become obstructed itself by particulate matter. The *tonsil tip (Yankauer)* suction device is used most effectively to clear upper airway hemorrhage and secretions. Its design is intended to prevent the obstruction of its tip by tissue and clot. The rounded tip is also less traumatic to soft tissues.

Unfortunately, the *catheter tip* suction device is the one most readily available in many hospitals. Often it is the only type of suction available. This device is inferior to the other catheter tips for suctioning before the patient has been intubated. After intubation, it works well for suctioning the trachea and bronchi through the tracheal tube. The dental tip device should be used during the resuscitation period and should be ready at the bedside. The dental tip allows rapid clearing of both particulate matter and hemorrhage, thereby expediting airway control.

Optimally, stabilization of the patient with multiple injuries will involve use of all three types of suction tips. The tonsil or dental tip should be attached to the suction source during the interval between patient evaluations because it is most likely to be the one needed on short notice. Both the tonsil tip and catheter tip should be stored next to the suction source so they can be attached when needed. It is essential that all physicians and nurses know the location of suction equipment and *know how to turn it on* during an

emergency. In the resuscitation rooms, the equipment should be connected and ready to operate and not kept in cabinets or wrapped in difficult-to-open packaging material (see Fig. 2-1) (Figure Not Available) . Interposition of a suction trap at the base of the dental tip suction device prevents clogging of the tubing with particulate debris. A trap that fits directly onto a tracheal tube has been described; use of this device allows effective suctioning during intubation (Fig. 1-5) (Figure Not Available) . [9]

Although no specific contraindications to airway suctioning exist, complications of incorrectly performed suctioning may be significant. Nasal suction is seldom required to improve oxygenation (except in infants), because most adult airway obstruction occurs in the mouth and oropharynx. Vigorous nasal suction can induce epistaxis and further complicate an already difficult situation. Suctioning that is prolonged may not be recognized during an emergency, but it should be avoided because it may lead to significant hypoxia, especially in children. *Suctioning should not exceed 15-second intervals*, and the provision of supplemental oxygen before and after suctioning should be routine (see also Chapter 5) . Basilar skull fractures can allow the inadvertent placement of nasal suction tubes in the brain (Fig. 1-6) (Figure Not Available) . Extreme care should be exercised when a basilar skull or facial fracture is suspected, because communication between nasal and intracranial cavities may exist.

Generally, it is best to perform suctioning *under direct visual inspection* or with the aid of the laryngoscope. Forcing a suction tip blindly into the posterior pharynx can injure tissue or convert a partial obstruction to a complete obstruction.

Complications may be avoided by anticipating problems and providing appropriate care during suctioning maneuvers. Epistaxis may be avoided by limiting the force applied during suctioning. Vasoconstrictor drops or spray, such as 0.25% phenylephrine, constrict the nasal mucosa and reduce the injury potential in patients who require repeated nasopharyngeal suctioning. The rescuer must be aware that the patient may develop transient pupillary dilation if the vasoconstrictor solution drips into the conjunctival space. [10] Naigow and Powasner found that suctioning induced hypoxia in dogs consistently and that it was best avoided by hyperventilating the animals before and after suctioning. [11]

## Artificial Airways

### Indications and Contraindications

Once the airway has been established through various maneuvers and suctioning, the patient may require further temporary support to maintain airway patency. The semiconscious patient who is breathing with an adequate rate and tidal volume at the time of the chin-lift maneuver may develop hypoxia because of recurrent obstruction if the maneuver is discontinued. Oxygen supplementation and an artificial airway may be all the support that is necessary. The use of an artificial airway also allows more efficient use of rescuer skills and relief from fatigue that is caused by the continuous application of chin-lift or jaw-thrust maneuvers.

Positive-pressure ventilation with a bag-valve-mask (BVM) device may be necessary to

bolster the patient's inadequate ventilatory effort or to provide total ventilation in cases of apnea. By maintaining airway patency, artificial airways facilitate spontaneous and bag-mask ventilation.

**Airway Placement Technique**

The simplest artificial airways are the oropharyngeal and nasopharyngeal airways (Fig. 1-7) (Figure Not Available) . Both are intended to prevent the tongue from obstructing the airway by falling back against the posterior pharyngeal wall. The oral airway may also prevent teeth clenching. The oropharyngeal airway may be inserted by either of two procedures. In the first procedure, the airway is inserted in an inverted position along the patient's hard palate. When it is well into the patient's mouth, the airway is rotated 180° and advanced to its final position along the patient's tongue, with the distal end of the airway lying in the hypopharynx. The second procedure involves the performance of a jaw-thrust maneuver, either manually or with a tongue blade, and the simple advancement of the airway into the mouth to its final position. No rotation is performed when the airway is placed in this manner. Once inserted, the oral airway may have to be taped in place to prevent expulsion by the patient's tongue.

The nasopharyngeal airway is placed by gently advancing the airway into a nostril, directing the tip along the floor of the nose toward the nasopharynx. When in final position, the flared external end of the airway should rest at the nasal orifice. Either of these two airways provides airway patency similar to that in a correctly performed chin-lift maneuver, but the nasal airway may be better tolerated by the semiconscious patient.

**Complications**

Few complications are encountered in the use of these airways. The oropharyngeal airway may cause obstruction if during its placement the tongue is pushed against the posterior pharyngeal wall. Care in placement will prevent this occurrence. In the patient whose reflexes are intact, the gag reflex may stimulate retching and emesis, and the semiconscious patient may not tolerate the oropharyngeal airway. If gagging is a persistent problem, the airway should be removed and a nasal airway or tracheal intubation should be considered. If the patient with airway compromise is comatose and lacks a gag reflex, the oropharyngeal airway *should not be used as a definitive airway*; tracheal intubation should be used instead. The oropharyngeal airway will keep the mouth partially open if an orogastric tube is placed for gastric lavage or suction, and it will prevent clenching of the teeth, which can obstruct an orotracheal tube.

The nasopharyngeal airway may offer an advantage over the oropharyngeal airway in that the nasopharyngeal airway is less likely to induce gagging. The same considerations that apply to nasal suctioning apply to placement of the nasopharyngeal airway. That is, care must be exercised not to induce epistaxis, and extreme caution is indicated in patients with a suspected basilar skull fracture or facial injury. All patients with oral or nasal pharyngeal airways should be observed constantly, because these devices are temporary measures and cannot substitute for tracheal intubation.

# BAG-VALVE-MASK VENTILATION

## Indications and Contraindications

Correctly performed, the BVM method of ventilation appears to be simple and effective. Still, it is fraught with difficulty and therefore deserves special mention. Bag-valve-mask ventilation should be used by experienced individuals who are able to ensure a tight mask seal in situations requiring positive-pressure ventilation. The BVM is often used with an oropharyngeal or nasopharyngeal airway in place. [3]

Inexperience is a relative contraindication to the use of a BVM. A rescuer who is not skilled with the BVM will achieve much better ventilation with mouth-to-mouth or mouth-to-mask breathing than with a BVM. However, concern regarding transmission of infectious diseases has reduced the willingness of the lay public and health professionals to perform mouth-to-mouth ventilations. [12] Although BVM ventilation may provide excellent respiratory support in the anesthetized, paralyzed patient in the operating room, the device frequently is of marginal value during cardiopulmonary resuscitation (CPR), during an ambulance run, or in the combative patient. A tight mask seal is mandatory to prevent loss of air volume during ventilation. Another hazard of BVM ventilation occurs when vomitus, blood, or other debris is present in the mouth or pharynx. The foreign material may be insufflated down the trachea if it is not cleared before ventilation. The three major problems encountered with BVM ventilation are inadequate tidal volumes, inadequate oxygen delivery, and gastric distention.

## Ventilation Technique

Achieving adequate tidal volume with BVM ventilation requires a tight mask seal and adequate compression of the bag. Even trained paramedics practicing on manikins have difficulty delivering tidal volumes above 650 mL, which is well below the 10-15 mL/kg recommended by the American Heart Association. A variety of mask configurations are available to facilitate a tight seal, but none substitutes for the practiced skill of the rescuer. For the single rescuer, only one hand can be used to achieve the seal because the other must squeeze the bag. The rescuer's hand must be large enough

to apply pressure anteriorly while simultaneously lifting the jaw forward. The thumb and index finger provide anterior pressure while the fifth and fourth fingers lift the jaw. Care must be exercised to deliver an adequate tidal volume by full compression of the bag. *Dentures generally should be left in place to help ensure a better seal with the mask.*

It has been suggested that effective BVM ventilation during CPR requires two hands and, therefore, two rescuers. [13] We suggest using the two-rescuer technique (Fig. 1-8 (Figure Not Available) *B*) whenever it is practical. The presence on the BVM device of a pop-off valve may further frustrate ventilation efforts in the patient with reduced compliance.

All BVM devices should be attached to a supplemental oxygen source (with a flow rate of 15 L/min) to avoid hypoxia. A significant problem with the BVM method is the low oxygen saturation achieved with various reservoirs. The amount of delivered oxygen is dependent on the ventilatory rate, the volumes delivered during each breath, the oxygen flow rate into the ventilating bag, the filling time for reservoir bags, and the type of reservoir used. The commonly used corrugated tube reservoir is the least effective of those examined by Campbell and colleagues. [14] It is too sensitive to ventilatory technique and does not alert the clinician to changes in oxygen flow. A 2.5-L bag reservoir and a demand valve are the preferred supplementation technique during BVM ventilation. [14]

Pediatric BVM devices should have a minimum volume of 450 mL. Pediatric and larger bags may be used for ventilation of infants with the proper mask size, but care should be taken to administer only the volume necessary to effectively ventilate the infant. Pop-off valves should be avoided because airway pressure under emergency conditions may often exceed the pressure of the valve. [3]

### Complications

Hypoventilation often occurs because of the difficulty of carrying out the technique properly. Three mechanisms can result in complications: poor mask seal, failure to achieve airway patency, and low tidal volume. Practiced skill development is necessary to avoid these errors. Gastric distention can also result from poor airway patency. Air is insufflated down the esophagus, which inflates the stomach. Consequently, the risk of regurgitation and aspiration increases. When assistance is available, the application of firm posterior pressure on the cricoid ring helps reduce gastric inflation during BVM ventilation. [15] [16] The technique must be used carefully in infants, whose airway is more pliable and subject to obstruction with excessive cricoid pressure. Even with proper BVM technique, aspiration can occur. The rescuer must be vigilant to recognize complications early and take corrective action.

## INTERMEDIATE AIRWAYS

*Intermediate airways* are those interventions that go beyond the maintenance of a patent airway. They represent a midpoint between airway establishment and true airway control. Airway control is secured by maneuvers such as tracheal intubation and tracheotomy, in which an endotracheal cuff isolates the trachea. The devices described in this section occlude the esophagus and allow ventilation across the larynx. The devices discussed are the esophageal obturator airway (EOA), the esophageal gastric tube airway (EGTA), the laryngeal mask airway (LMA) and the esophageal-tracheal Combitube (ETC) airway (Sheridan Catheter Corp., Argyle, NY). Two are designed to occlude only the esophagus (EOA and EGTA), one (LMA) seals the larynx at the hypopharynx level, and one ETC offers the versatility of use whether placed into the esophagus or the trachea. Each is designed for use in the unconscious patient who requires positive-pressure ventilation. The esophageal cuff or seal built into these devices reduces gastric content aspiration. The EOA and EGTA have fallen into general disfavor in recent years due to the gravity of errors in placement. Complications

including esophageal rupture and tracheal intubation have led many to prefer the ETC or LMA as an intermediate airway.

## Esophageal Obturator Airway and Esophageal Gastric Tube Airway

The EOA and the EGTA maintain airway patency in ways similar to the oral and nasal airways, but they also protect

the airway by occluding the esophagus to reduce gastric distention and regurgitation. The face mask permits use of these airways as positive-pressure ventilating devices. Air insufflated through the airway traverses the upper airway before crossing the larynx and entering the trachea. Ventilation from the EOA exits the airway through numerous ports in its hypopharyngeal portion (Fig. 1-9 (Figure Not Available) *A* and *B*). Ventilation from the EGTA is identical to mask ventilation, with the addition of esophageal occlusion. A port is available on the EGTA to vent the stomach. The attractiveness of the EOA and the EGTA for use in the apneic patient stems from their retention of much of the simplicity of the artificial airway with the addition of an important feature of more complicated airways--some protection against regurgitation and reduction of gastric distention.

### Indications and Contraindications

Speed and simplicity are advantages of the esophageal airway over tracheal intubation. Trained individuals can successfully place an esophageal airway in an average of 5 seconds, whereas the same individuals may need 20 seconds to perform a tracheal intubation. [17] [18] In one out-of-hospital study, failure to intubate was much higher with the endotracheal tube (19.4%) than with the EOA (1.7%). [19] Neck motion is not as necessary with the esophageal airway as it is with tracheal intubation. For these reasons, the EOA may be an effective adjunct in the management of the unconscious injured patient who requires respiratory assistance. Hypercarbia may occur more commonly with EOA ventilation as compared with endotracheal ventilation. [20] [21] The most difficult aspect of this form of ventilation is securing a tight fit with the mask. Dentures should be left in place to give support to the lips. Adequate tidal volume must be delivered to ventilate the lungs. [22]

There are various contraindications to the use of the EOA and the EGTA. Because the airway is not protected from pharyngeal secretions, the presence of active oropharyngeal bleeding and excessive secretions represent a relative contraindication to EOA and EGTA use. Because of attendant discomfort, the devices cannot be used in the awake patient. Size specifications preclude their use in the pediatric patient; 16 years is the age usually cited as the lower limit for EOA and EGTA use. The actual limiting factors are the size of the esophagus and the face; an adult-sized 14-year-old would certainly tolerate an EOA or EGTA if necessary. However, a small adult may not receive an appropriate fit. Other contraindications include esophageal injury or conditions predisposing to perforation. A patient who has ingested a caustic agent or one with a known esophageal stricture should not undergo esophageal intubation. As a precaution against pressure-related complications, it is recommended that the device not be left in place for longer than 2 hours. It must be recognized that the EOA and the

EGTA are *temporary* forms of airway control. This form of airway control is most often used in out-of-hospital care.

**Placement of EOA/EGTA**

The head is in the neutral position during placement of the EOA and the EGTA. Neck motion is unnecessary. The rescuer grasps and pulls the jaw forward. At this point, the rescuer inserts the assembled airway with the mask attached. The obturator tip is directed into the patient's posterior pharynx with gentle, steady pressure. The obturator is advanced down the esophagus until the mask rests flush against the face of the patient. Figure 1-9 (Figure Not Available) *A* illustrates the correct position at placement. The cuff should lie in the esophagus just distal to the carina of the trachea. The rescuer postpones inflation of the balloon until proper position is confirmed. The patient is ventilated with a tight mask seal on the face, and the lungs are auscultated. For effective ventilation, the mask seal must be tight. Breath sounds should be audible bilaterally. Unilateral breath sounds or failure of auscultation should lead the rescuer to reassess the airway placement. Pneumothorax or hemothorax may explain unilateral sounds, as may inadvertent main stem bronchus intubation. Tracheal intubation will result in the absence of breath sounds. The possibility of bronchial or tracheal intubation requires removal and replacement of the airway. Once satisfactorily placed, the esophageal balloon is inflated to 20 to 25 mL.

**Complications**

A 5% incidence of inadvertent tracheal intubation has been reported by Don Michael in experience with 29,000 placements. [18] In a subsequent smaller sample, a 2.9% (5 of 170) incidence was reported with a 100% mortality among the 5 patients. [23] One study comparing out-of-hospital EOA placement with endotracheal tube placement found that the occurrence rate for complications of the EOA that prevented resuscitation (tracheal placement, tube kinking) was nearly three times higher for the EOA (8.7% vs 2.6%). [24] If not quickly rectified, tracheal intubation with the EOA or tube kinking are disastrous complications that produce occlusion of the patient's airway. Disciplined examination for bilateral breath sounds is critical.

Esophageal lacerations of undetermined depth were found in 8.5% of autopsies of patients in whom the EOA was used. [19] Esophageal rupture has been found and reported in case histories. [25] [26] Since Scholl and Tsai first reported esophageal ruptures in 1977, [26] the recommended balloon inflation volume was reduced from 35 to 20 mL. No further ruptures or leakage around the cuff have been reported. However, factors other than balloon inflation volume that theoretically can contribute to rupture include careless balloon removal without deflation and forceful attempts at placement when obstruction is met.

*Tracheal intubation should be performed BEFORE removal of the EOA*, because vomiting often occurs following deflation of the balloon and EOA removal (see Chapter 2) . If the EOA cuff has been overinflated, it may partially occlude the trachea and make intubation difficult. In such cases, the balloon is partially deflated to facilitate tracheal

intubation.

## The Laryngeal-Mask Airway

The laryngeal-mask airway (LMA) (Intavent International SA, Henley-on-Thames, England) functions intermediately between an oropharyngeal airway and an endotracheal tube. It was developed for use in the operating room as an alternative for endotracheal intubation, but it has also been recommended for use in difficult intubations. [27] It consists of a tube fitted with an oval mask, rimmed with an inflatable cuff (Fig. 1-10 (Figure Not Available) *A*). Contrary to usual mask design, the mask is intended to reside in the hypopharynx rather than on the face. It is inserted digitally until its tip meets resistance in the upper esophageal sphincter. The cuff is then inflated, forming a seal around the glottic opening (see Fig. 1-10 (Figure Not Available) *B*). The result is a relatively secure airway. However, it cannot be considered to protect against gastric regurgitation. Leakage of the hypopharyngeal mask allows aspiration of emesis and gastric distention may occur with misplacement. Although the device may be used for prolonged periods under appropriate conditions, it is usually considered a temporary adjunct until tracheal intubation is established. [27]

### Indications and Contraindications

The LMA is indicated for patients requiring an airway who cannot be endotracheally intubated. The most frequently cited example is a patient whose anatomy prevents visualization of the larynx. Contraindications include the inability to open the patient's mouth and vomiting.

### Placement of LMA

The LMA is first checked for possible air leaks by inflating and deflating the cuff. If the patient has a gag reflex, deep oropharyngeal topical anesthesia or conscious sedation must be administered. With the patient's head in the sniffing position, the mask is lubricated and oriented so the mask opening is facing the tongue. With the index finger of the dominant hand placed on the proximal aspect of the mask, the mask is inserted into the mouth, firmly against the hard palate (see Fig. 1-10 (Figure Not Available) *C*). The index finger (or thumb) may also be used as a guide during advancement (see Fig. 1-10 (Figure Not Available) *D*). With one smooth motion, the mask is advanced until resistance is encountered. With the tip of the mask thus seated in the upper esophageal sphincter, the cuff is inflated. The lungs are auscultated to confirm correct placement.

While the sniffing position is desirable, it has been shown that LMA placement was 95% successful when the patient was placed in the neutral position with in-line immobilization, simulating a trauma setting. [28]

After successful placement of the LMA, several methods are available to achieve subsequent endotracheal intubation. The first method is simply to pass an appropriately sized endotracheal tube down through the lumen of the LMA, rotate the tube 90° so that the tip easily passes through the fenestrations, and advance it through the larynx to the

trachea (Fig. 1-11) (Figure Not Available) . This has been found to be successful in 90% of attempted cases. [29] The second method involves the use of a tracheal tube exchanger. The exchanger is passed blindly down the lumen of the LMA and into the trachea. The LMA is then removed and an endotracheal tube is passed over the tracheal tube exchanger. This method of tube placement must be combined with confirmation of exchanger location, because it has been shown to pass into the esophagus in up to 70% of attempts. [30] Confirmation of endotracheal tube location should be made (see Chapter 2) . The third and most dependable method of intubation with an LMA in place is via a fiberoptic scope. A lubricated, appropriately sized endotracheal tube is mounted over a fiberoptic scope, and this combination is advanced through the lumen of the LMA out through the mask and through the larynx. The scope is then removed, but the LMA may be left in place with the cuff deflated. If the LMA must be removed after a tracheal tube has been successfully placed through it, pass a tracheal tube exchanger down the tube, remove the tracheal tube/LMA combination, and replace it with a tracheal tube.

### Complications

Although the LMA works well in most cases, this airway has several significant drawbacks. Aspiration is always a possibility, because the cuff does not provide a watertight seal. Laryngospasm can occur if adequate anesthesia is not achieved. A significant air leak around the cuff may occur when high airway pressures exist, leading to poor ventilation. Finally, success rates in the operating room range from 94 to 98%; success rates in difficult emergency airway management are unknown, but they are undoubtedly lower.

### Conclusion

The LMA is a blindly placed intermediate airway that should be considered in patients who require establishment of an emergency airway but cannot receive endotracheal intubation. The technique is quick and simple, requires a minimum amount of training, and appears effective in the hands of paramedics, nurses, and respiratory therapists. [31] [32]

## The Esophageal-Tracheal Combitube

The ETC is a noninvasive airway device that is placed blindly. It allows for effective ventilation and oxygenation when placed in either the esophagus or the trachea. The device has two lumina running parallel to each other. One is perforated at the level of the pharynx and occluded at the distal end, similar to the EOA. The second lumen is open at the distal end, resembling an endotracheal tube. The device has two balloons: a proximal pharyngeal balloon that occludes the oropharynx by filling the space between the base of the tongue and the soft palate and a smaller, distal cuff that serves as a seal in either the esophagus or trachea (Fig. 1-12) (Figure Not Available) . [33] [34] The Combitube has compared favorably with the endotracheal tube with respect to ventilation and oxygenation in cardiac arrest situations. [35] [36] It is also placed more rapidly. [35]

**Indications and Contraindications**

The ETC is superior to other intermediate airways, because no face mask seal is necessary. It may be preferable to tracheal intubation in certain situations, because it can be placed blindly and is also effective in the esophageal or tracheal position. It is, therefore, more easily placed than an endotracheal tube and is indicated in situations in which tracheal intubation is difficult, neck motion is impossible, or the rescuers are not skilled in tracheal intubation.

The ETC should not be used in patients with an intact gag reflex and is not recommended in patients younger than 16 years or less than 5 feet in height. It is contraindicated in suspected caustic poisonings or proximal esophageal disorders.

**Placement of ETC**

The device is held in the dominant hand and gently placed caudally into the pharynx while the nondominant hand grasps the tongue and jaw between the thumb and index finger. The tube is passed blindly to a depth where the printed rings on the proximal end of the tube lie between the patient's teeth or alveolar ridge. The pharyngeal balloon is then filled with 100 mL of air, and the distal cuff is subsequently filled with 10 to 15 mL of air. The large pharyngeal balloon serves to both securely seat the ETC in the oropharynx and to create a closed system in the case of esophageal placement. Because approximately three-quarters of placements are esophageal, ventilation is begun through the longer (blue plastic) connector associated with the esophageal lumen. Chest rise and good breath sounds without gastric insufflation confirms effective placement in the esophagus. However, gastric insufflation without breath sounds and chest rise indicate a tracheal positioning of the tube and require changing the ventilation to the shorter (clear plastic) tracheal lumen. Auscultation of breath sounds over the lateral lung fields confirms endotracheal placement of the Combitube. If the tube is in the esophageal position, gastric suctioning can be accomplished by passing a catheter through the open lumen into the stomach while the patient is being ventilated via the other port.

An alternative method to identify position is to attach an aspirating device (see Chapter 2) to the tracheal or clear plastic shorter tube. The inability to easily aspirate air confirms esophageal placement necessitating ventilation via the longer blue esophageal tube. In the patient with ventilatory effort, $CO_2$ detector devices also may be useful.

A patient who has been successfully resuscitated with an ETC positioned in the esophagus should ultimately receive a definitive airway. The steps required to place a tracheal tube in this setting are detailed in Chapter 2 but consist generally of deflating the large pharyngeal balloon and, with the distal balloon still inflated, intubating around the ETC.

**Complications**

Inappropriate balloon inflation and incorrect ETC placement can lead to air leaks during ventilation. The most common placement error is an improper insertion angle. A more caudal, longitudinal direction is recommended, as opposed to an anteroposterior direction of insertion. Another caveat is that the ETC must be maintained in the true midline position during insertion to avoid blind pockets in the supraglottic area, which prevent passage of the tube. Attention to the ring markings on the tube at the level of the incisors ensures proper positioning of the tube. One must remember to first inflate the oropharyngeal balloon before inflating the distal balloon. Although unlikely, esophageal injury is theoretically possible with the overinflation of the distal balloon.

## SPECIAL CONSIDERATIONS

### Cardiac Arrest

Mouth-to-mouth and BVM ventilation may suffice for out-of-hospital care with short transport times or for the initial few minutes of ventilation in cardiac arrest. However, optimal BVM ventilation during CPR is impossible. Mouth-to-mouth and BVM ventilation are adequate and effective in the anesthetized or paralyzed patient with an empty stomach in the absence of chest compression, but they are *inadequate* for prolonged ventilation in the patient in cardiac arrest.

Proper BVM ventilation is probably harder to master than tracheal intubation, and prolonged attempts during CPR usually only distend the stomach and give the uninitiated a false sense of security. *Patients in cardiac arrest should be orotracheally intubated.* Most cardiopulmonary arrests are not associated with cervical spine injury. When there is suspicion of cervical injury, the following precautions should be followed.

### Potential Cervical Spine Injury

Any patient who has sustained a significant injury has the potential for cervical spine injury. Approximately 1.5 to 3.0% of initial survivors of all types of major trauma seen in emergency departments have significant cervical spine injury. [37] [38] It is interesting to note that this prevalence is not increased in the setting of significant head injury. Falls from heights and motor vehicle crashes are also common causes of spinal instability.

In patients with multiple injuries, the possibility of cervical spine injury warrants caution when considering tracheal intubation involving the use of the laryngoscope. It is prudent to provide adequate oxygenation while limiting neck extension until cervical spine injury is disproved. If the patient is severely hypoxic or apneic, immediate tracheal intubation may be necessary with in-line manual stabilization of the neck (without axial traction) by an assistant. When done cautiously, oral intubation of the unconscious spinal cord injured patient may be as safe as other techniques, including intubation with fiberoptic guidance. [39]

Note that mouth-to-mouth and BVM ventilation frequently require some degree of neck extension to open the airway. A cadaver study demonstrated increased neck motion

with BVM ventilation when compared to various intubation techniques, including oral intubation, lighted stylet guided oral intubation, and nasotracheal intubation. [40] BVM techniques may, therefore, be less desirable than the other methods of securing the airway and ventilating the patient.

Many institutions and some out-of-hospital systems use pharmacologic adjuncts, in-line cervical stabilization, and orotracheal intubation before cervical spine films are initiated. In the patient who is comatose, combative, or in severe respiratory distress without definite evidence of spinal cord injury, this approach is advocated, because it may be life saving. Precautions during intubation of the patient with known cervical spine fracture or its potential should include in-line stabilization of the cervical spine with attempts to minimize traction or lateral neck motion during the intubation procedure. Clinical experience is accumulating that supports the safety of this approach.

**Potential Epiglottitis/Supraglottitis**

Epiglottitis is often considered a disease of children between the ages of 2 and 8 years, but it is being recognized in adults with increasing frequency. [49] The typical presenting picture is that of an adult or child sitting upright, drooling, or spitting up oral secretions rather than swallowing. The voice may sound muffled. There is a history of a relatively abrupt onset of a sore throat that rapidly becomes more painful. Children commonly present with a high temperature, but adults usually are only mildly febrile. The disease is especially treacherous in children because of their small airways and their tendency to panic when an oral examination or insertion of an IV line is attempted.

Small children are most calm when allowed to sit on a parent's lap. An oxygen mask with oxygen flowing at 10 L/min can be held by the parent several centimeters from the child's face. If the child is using accessory muscles to breathe, every attempt should be made to keep the child calm. If a lateral radiograph of the neck taken on inspiration can be obtained without disturbing the child, it will often establish the diagnosis. On radiography, the inflamed epiglottis often appears thickened and rounded. The hypopharynx is dilated above the obstruction.

In cases of respiratory compromise, an epiglottitis protocol should be implemented rapidly. A preestablished protocol can save many minutes of time otherwise spent trying to reach all of the personnel needed to manage this critical emergency. When a child is suspected of having epiglottitis based on history and clinical presentation, the safest course of action to establish the airway should be pursued. The emergency physician should accompany the child at all times until the airway is secure and be prepared to intervene. Otolaryngologist notification should be included in the protocol because a tracheostomy may be necessary. [50] When operating room space or personnel are not available immediately, emergency department personnel must be prepared to manage the airway.

If the child lapses into a coma or stops making ventilatory efforts, the first step is to attempt to force oxygen past the obstruction by using mouth-to-mouth respiration or a BVM apparatus. Because the obstruction is edematous supraglottic tissue and epiglottis, positive-pressure ventilation often can displace the edema enough to allow

adequate ventilation. If this effort is unsuccessful, the emergency physician should attempt oral intubation. However, a normal larynx will not be visible because of the edema. The operator should attempt to pass an endotracheal tube through the slit-like opening that remains for the supraglottic airway. An assistant can compress the chest to force bubbles through the airway, as a means of locating the airway. [51] The assistant can also palpate the larynx and the trachea to detect the tube's entry into the trachea. If orotracheal intubation fails, the intubator should go directly to transtracheal needle ventilation (TTNV) (see Chapter 4) . The obstruction of epiglottitis is mainly inspiratory, so there should be no difficulty with chest hyperinflation with intermittent TTNV. This method should ease subsequent orotracheal intubation, because the path of the airway should be readily apparent as exhaled gases pass through it.

It is recommended that all children with acute epiglottitis receive tracheal intubation. [52] If the child is not in distress, an IV line can be established before intubation for appropriate drug administration, although some operators prefer to delay IV placement until after inhalation anesthesia.

Adults and cooperative older children with suspected epiglottitis can be examined directly. It is good practice to visualize the epiglottis and the vocal cords of the stable older patient with laryngeal tenderness who is complaining of a severe sore throat or difficulty swallowing. A mirror, fiberoptic scope, or a right-angle scope can be used to do this (see Chapter 68) . In epiglottitis, the pharynx and tonsils usually do not appear inflamed, a finding that might otherwise explain the symptoms. Adults with epiglottitis do not always

need to be intubated if rigorous monitoring can be accomplished, a skilled intubator is immediately available, and the patient is not in distress. [49] Orotracheal intubation for epiglottitis is not as difficult in adults as it is in small children. Transtracheal needle ventilation can also be used in adults who are difficult to intubate.

**Jaw Clenching**

Hypertonus induced by neurologic dysfunction is a common complicating factor of airway management, especially in the patient with multiple injuries, drug overdose, or seizures. Jaw clenching may be a lethal complication when it prevents clearing of blood, vomitus, or foreign bodies in the airway. No more difficult airway problem exists than occlusion of the nasal and oral passages by vomitus while the patient's teeth are tightly clenched. Respiratory efforts may lead to severe aspiration, and although the hypertonus gradually gives way as the brainstem becomes progressively hypoxic, the cerebrocortical hypoxic insult sustained in the process may be irreversible. Various disease states can lead to a similar scenario in which the jaws are clenched in the presence of upper airway hemorrhage or the accumulation of secretions.

Jaw clenching and cervical spine injury can, of course, occur together. At times, the blind nasotracheal route of intubation may be adequate for airway management while minimizing the risk of further spine injury. However, at least a small degree of spontaneous air movement should be present for the blind nasotracheal approach to be successful. Although a serendipitous success may occur in the apneic patient, it is

recommended that time not be wasted on this approach in the completely apneic patient.

Neuromuscular blocking agents are generally an effective means to overcome jaw clenching in the breathing patient. Both neuromuscular depolarizing and nondepolarizing agents may be administered IV to induce paralysis and allow orotracheal intubation. Neuromuscular blocking agents and their administration for intubation are discussed in Chapter 3 .

**Apnea with Airway Obstruction**

Despite the many nonsurgical approaches to tracheal intubation discussed in this chapter, the patient who is apneic secondary to deep airway obstruction may be served best by a surgical airway. When maneuvers to relieve airway obstruction are unsuccessful and direct laryngoscopy is not possible or cannot rapidly alleviate the obstruction and permit ventilation, the operator should rapidly move to a surgical airway approach (see Chapter 4) .

## CONCLUSION

Airway management is the most fundamental aspect of emergency care. Every rescuer must know basic airway maneuvers and be able to use them instinctively. When basic maneuvers fail, airway management rapidly becomes more complex. Familiarity with the ingenious intermediate airway devices can often reverse a deteriorating situation and provide the rescuer with a temporary solution to an airway dilemma. When basic and intermediate maneuvers fail, complexity, risk, and exigency mount. Choices become more critical and complications more likely. Advance consideration of situations represented in the airway management algorithms is a wise practice for the emergency physician. It may hasten accurate decision-making when time becomes critical. In this chapter we have described basic and intermediate airway techniques and offered a logical schema for their use in the patient with an acutely compromised airway. Subsequent chapters deal with the more advanced airway techniques of tracheal intubation and cricothyrotomy.

# Chapter 2 - Tracheal Intubation

**John W. McGill** , **Joseph E. Clinton**

Tracheal intubation is generally considered the most definitive means of airway control. As discussed in Chapter 1 , the decision to tracheally intubate must consider the patient's physiologic status, anticipated patient care needs, operator experience, and features related to preparation for the procedure. This chapter discusses the indications for tracheal intubation in greater detail as well as the preparation for intubation and the key steps and modifications of the actual procedure. Chapter 3 discusses the pharmacologic adjuncts (including pharmacologic paralyzing agents) for facilitating tracheal intubation and their administration. Chapter 4 discusses more invasive means of establishing an airway and ventilating the patient. The general decision-making that is relevant to these techniques has been presented in Chapter 1 and should be reviewed prior to proceeding with this chapter.

## GENERAL PREPARATION

Preparation is the key to successful airway management. Two general areas of preparation should be addressed before undertaking the first attempt at definitive airway management in a clinical setting. The first is mental and physical preparedness. The second is the assembly of essential intubation equipment.

Mental and physical preparation comes from reading about the procedures, discussing the principles and details with instructors, practicing the techniques on intubation mannequins or in the animal laboratory, and finally performing the technique under supervision in a controlled clinical setting. Studies addressing various approaches to tracheal intubation are generally performed under optimal conditions (i.e., with equipment available and appropriate preparatory training). Also, often hidden within the study findings are individual learning curves. Therefore, it is overly optimistic to expect to match the success reported in the literature when first attempting a new intubation technique. However, the goal of preparation is to be as high on the learning curve as possible prior to the first clinical application of a new intubation technique. Further, continued rehearsal and application of the techniques that have been learned are important for skill maintenance.

Each approach to tracheal intubation has a preferred training format. Orotracheal intubation, for example, may be simulated with a mannequin, whereas retrograde intubation is best learned using an animal or cadaver model. Orotracheal intubation is likely to be successful on the first attempt, whereas considerable practice is required for facile use of the scope for fiberoptic intubation. In preparation for managing critical airway problems, maximal hands-on training is desirable.

The second general area of preparation is material preparedness (i.e., the immediate availability of all essential equipment required to optimally perform the airway maneuvers that are within the capabilities of the care provider). This may be accomplished by the wall-mounting of essential resuscitation equipment. [1] Alternatively,

dedicated adult and pediatric airway carts may be used for placement of the equipment in an open, organized, and labeled manner that can be regularly checked (Fig. 2-1) (Figure Not Available) . [2] The worst moment to realize that a vital piece of equipment is missing is when a patient's life depends on it. The importance of this concept cannot be overstated. *Technical expertise cannot substitute for the lack of essential equipment.*

In airway management, failure has ominous consequences. Mental, physical, and material preparation maximizes the chances of success.

## AIRWAY ANATOMY

Requisite for a discussion of procedures in airway management is a common understanding of airway anatomy and its terminology (Fig. 2-2 *A* and *B*). The following terms are used frequently in this chapter:

*Arytenoid cartilages*

> the paired cartilages forming the posterior aspect of the laryngeal inlet nasal cavity, from the external nares to the choana.

*Nasopharynx*

> from the end of the nasal cavity (choana) to the level of the soft palate.

*Oropharynx*

> soft palate to the upper border of the epiglottis.

*Hypopharynx (laryngopharynx)*

> epiglottis to the lower border of the cricoid cartilage.

*Vallecula*

> the space at the base of the tongue formed posteriorly by the epiglottis and anteriorly by the anterior pharyngeal wall.

*Laryngeal inlet*

> the opening to the larynx bounded anterosuperiorly by the epiglottis, laterally by the aryepiglottic folds, and posteriorly by the arytenoid cartilages.

*Piriform fossae (recesses)*

> the pockets on both sides of the laryngeal inlet separated from the larynx by the

aryepiglottic folds.

*Corniculate cartilage*

the posteromedial portion of the arytenoid cartilage.

*Cuneiform cartilage*

the anterolateral prominence of the arytenoid cartilage.

*Glottis*

the vocal apparatus, including the true and false cords and the glottic opening.

*Glottic opening (rima glottidis)*

the opening into the trachea as seen from above through the vocal cords.

## OROTRACHEAL INTUBATION

### Indications and Contraindications

Any clinical situation in which a definitive airway is necessary and limited neck motion is permissible is an indication for orotracheal intubation. Many of these situations, including cardiac arrest, airway compromise in infection and trauma, and airway obstruction are discussed in detail in Chapter 1 . Most orotracheal intubations are accomplished using a direct laryngoscope. An unstable cervical spine injury is a relative contraindication to direct laryngoscopy.

### Equipment

#### Laryngoscope

Facility in the use of the direct laryngoscope is a prerequisite for orotracheal intubation. Various adult and pediatric blade sizes are available. There are two basic blade designs-- curved (MacIntosh) and straight (Miller and Wisconsin). Slight variations in laryngoscopic technique follow from one's choice of blade design, which is often a matter of personal preference. The tip of the straight blade goes under the epiglottis and lifts it directly, whereas the curved blade fits into the vallecula and indirectly lifts the epiglottis via the hyoepiglottic ligament to expose the larynx. Special blades designed for the anterior larynx include the Siker and the Belscope (Avulunga Pty Ltd, New South Wales, Australia).

Each blade type has advantages and disadvantages. The straight blade is usually a better choice in pediatric patients, in patients with an anterior larynx or a long floppy

epiglottis, and in individuals whose larynx is fixed by scar tissue. It is less effective, however, in patients with prominent upper teeth, and it is more likely to break teeth. Use of the straight blade is also more often associated with laryngospasm due to its stimulation of the superior laryngeal nerve, which innervates the undersurface of the epiglottis. A straight blade may inadvertently be advanced into the esophagus and initially present one with unfamiliar anatomy until it is withdrawn. The blade has a light bulb at the tip that may slightly hamper vision. The wider, curved blades are helpful in keeping the tongue retracted from the field of vision, allowing for more room in passing the tube in the oropharynx, and they are generally preferred in uncomplicated adult intubations. Aside from patient considerations, some clinicians prefer the curved blade because they find it requires less forearm strength than the straight blade.

**Tracheal Tubes**

The standard endotracheal tube is plastic and measures approximately 30 cm. Tube sizing is based on internal diameter (ID), measured in millimeters, and ranges from a 2.0 to a 20.0 mm tube, increasing in increments of 0.5 mm. The outer tube diameter is 2 and 4 mm larger than the internal diameter. [3] Tube size is printed on the tube. There is also a scale, in centimeters, for determining the distance along the tube from the tip.

Adult men will generally accept a 7.5 to 9.0 mm orotracheal tube, whereas women can usually be intubated with a 7.0 to 8.0 mm tube. In most circumstances, tubes smaller than these should not be used, especially in patients with chronic obstructive lung disease who may be difficult to wean from the respirator due to excessive airway resistance from a small tube. However, in emergency intubations, particularly if a difficult intubation is anticipated, many clinicians

choose a smaller tube and change to a larger tube later if necessary. One exception is in the burn patient, in whom one places as large a tube as possible on the initial attempt because swelling may prohibit subsequent tube placement. For nasal intubation, a slightly smaller tube (by 0.5 to 1.0 mm) is chosen.

The cuff of a standard tracheal tube is high-volume and low-pressure. A clinical test for determining correct cuff inflation is to slowly inject air until no air leak is audible while the patient is receiving bag-tube ventilation. This usually occurs with 5 to 8 mL of air if the proper-sized tracheal tube has been selected. Many clinicians use the tension of the pilot balloon as a guide to cuff inflation; slight compressibility with gentle external pressure indicates adequate inflation for most clinical situations. For long-term use, cuff pressure should be measured and maintained at 20 to 25 mm Hg. Capillary blood flow is compromised in the tracheal mucosa when the cuff pressure exceeds 30 mm Hg.

In *infants and children*, the following formula is a highly accurate method for determining correct tracheal tube size:

Tube size = [4 + age (years)]/4

For most clinical situations, however, using the width of the nail of the little finger as a

guide is sufficiently accurate and has been shown to be more precise than finger diameter 45911 (Fig. 2-3) . [4]

Correct tube size is especially important in the pediatric population (Table 2-1) (Table Not Available) , because most patients younger than 8 years are intubated with an uncuffed tube; adequate tube size is necessary to provide a good seal between the tube and the upper trachea and to prevent aspiration. A cuffed tube is used in children with decreased lung compliance who may require prolonged mechanical ventilation. In a child, the smallest airway diameter is at the cricoid ring rather than at the vocal cords, as in adults. Hence, a tube may pass the cords but go no farther. Should this occur, the next smaller sized tube should be passed after reoxygenation.

Adult endotracheal tubes will accept a standard adaptor on which the ventilator tubing will fit. Pediatric tubes require a special adaptor with a distal end small enough to accommodate the small tube size.

**Preparing for Intubation**

Before beginning intubation, a number of issues should be addressed. In chronologic order, they are (1) confirm that
the required intubation equipment is available and functioning; (2) position the patient correctly; (3) assess the patient for difficult airway; (4) establish intravenous (IV) access, time permitting; (5) draw up essential drugs, and; (6) attach the necessary monitoring devices. In the haste of the moment, it is a common error to fail to position the patient properly or to proceed with the procedure before the proper equipment is assembled and checked. Simple omissions, such as failing to restrain the patient's hands, removing dentures, or misplacing the suction device, can seriously hamper the performance of the procedure. A suggested pre-intubation checklist is presented in Table 2-2 .

In addition to the preparation necessary for optimum patient care, the operator should also minimize exposure to potentially infectious materials (see Chapter 75) . Generally, the operator should be gloved and should wear eye and mouth protection to guard against exposure to patient secretions.

The endotracheal tube cuff should be checked for leaks by inflating the balloon before attempting intubation. The tube is prepared for placement by passing a flexible stylet down the tube to increase its stiffness and enhance control of the tip of the tube. The stylet should not extend beyond the end of the tube. The tube is then bent in a gradual curve with a more acute angling in the distal one-third to more easily access the anterior larynx. The tip and cuff of the tube are lubricated with viscous lidocaine or another water-soluble gel.

The patient should be positioned to optimally align the oral, pharyngeal, and laryngeal axes (Fig. 2-4) . The desired position was aptly described by Magill to make the patient appear to be "sniffing the morning air," with the head extended on the neck and the neck *slightly flexed* relative to the torso. A small towel under the occiput (to raise it 7 to 10 cm) may facilitate positioning. Positioning of the head and neck is a critical step;

nonoptimal head positioning may be the sole reason for some intubation failures.

| | TABLE 2-2 -- Suggested Preintubation Checklist |
|---|---|
| 1. | An assistant should be in the room watching the cardiac monitor, blood pressure, and $O_2$ saturation while observing the patient for signs of decompensation. The assistant should be instructed to inform the operator if more than 30 sec have elapsed without ventilation. |
| 2. | An IV infusion should be running properly. Oxygen should be administered to the patient. |
| 3. | Draw the necessary drugs (e.g., atropine, lidocaine, paralyzing agent, induction agent). |
| 4. | Attach the bag-valve-mask to an oxygen source (rate of 15 L/min). |
| 5. | If used, a stylet should be inserted properly into the tracheal tube. |
| 6. | Check the integrity of the balloon on the tracheal tube. |
| 7. | Have tape or commercial tube stabilizer available. |
| 8. | Check laryngoscope light source. Have a second light source, a selection of blades, and additional endotracheal tubes available. |
| 9. | Turn on the oral suction device and place the suction tip under the mattress to the left of the patient's head. Prepare the catheter suction for postintubation use. |

| 10. | Place the syringe to inflate the endotracheal tube balloon on the stretcher to the right of the patient's head. An option is to attach a syringe to the pilot balloon of the endotracheal tube. |
|---|---|
| 11. | If the patient is awake, restrain the hands. |
| 12. | Remove the patient's dentures (delay this action until immediately before intubation if patient is being bag-mask ventilated). |
| 13. | Check the cardiac monitor leads and the rhythm strip immediately before the intubation attempt. |
| 14. | Check for optimal head positioning: neck slightly flexed and head extended on the neck (conditions permitting). May be facilitated by placing a towel under the patient's occiput to raise it 10 cm. |
| 15. | Have an aspiration device for esophageal detection or an end-tidal $CO_2$ detector at the bedside. |
| 16. | Radiology department should be alerted for the postintubation chest radiograph. |

**The Difficult Airway**

The majority of difficult intubations are predictable. Perhaps the most frequently encountered condition associated with a difficult intubation is the agitated or combative patient. Fortunately, this condition can be readily eliminated through pharmacologic intervention (see Chapter 3) . The classic parameters that predict a difficult intubation include a history of previous difficult intubation, prominent upper incisors, limited ability to extend at the atlanto-occipital joint, [5] poor visibility of pharyngeal structures when the patient extends the tongue (Mallampati's classification, or the tongue/pharyngeal ratio) (Fig. 2-5 A), [6] limited ability to open the mouth, [7] a limited direct laryngoscopic view of the laryngeal inlet, [7] and a short distance from the thyroid notch to the chin with the neck in extension (Fig. 2-5 B). [8] Radiographic indicators of the ease of intubation include the mandibular length-to-height ratio [9] and the distance from the spine of the atlas to the occiput. [10] In emergency airway management, many of these predictors are

not obtainable. An extensive history is rarely available, the patients are frequently uncooperative, and the presence of trauma limits movement of the neck. Fortunately, some of the key predictors are apparent simply by observing the external appearance of the patient's head and neck.

Patients with neck tumors, thermal or chemical burns, traumatic injuries to the face and anterior neck, angioedema and infection of the pharyngeal and laryngeal soft tissues, or previous operations in or around the airway suggest a difficult intubation because distorted anatomy or secretions may compromise visualization of the vocal cords. Facial or skull fractures may further limit airway options by precluding nasotracheal intubation. Patients with ankylosing arthritis or developmental abnormalities, such as a hypoplastic mandible or the large tongue of Down's syndrome, are difficult to intubate because neck rigidity and problems of tongue displacement can obscure visualization of the glottis.

Besides these obvious congenital and pathologic conditions, the short, thick neck poses the greatest difficulty for performing orotracheal intubation. In such individuals, the larynx is anatomically higher and more anterior, which makes it harder to visualize the vocal cords. These individuals are easily identified by observing the head and neck in profile. In such patients, apply laryngeal pressure and consider using the straight blade. Use of other options, including nasotracheal intubation, may be required.

It should be emphasized that some patients, despite normal-appearing anatomy and the absence of a complicating history, are unexpectedly difficult to intubate. One must be prepared for this rare but inevitable occurrence.

## Procedure

### Adults

**Direct laryngoscopy.**

The operator is stationed at the patient's head (Fig. 2-6) . The patient is generally supine with the head at the level of the operator's lower sternum. To maintain the best mechanical advantage, the operator keeps his or her back straight and does not hunch over the patient; any bending should occur in the knees. The left elbow is kept relatively close to the body and flexed to provide better support. In the severely dyspneic patient who cannot tolerate lying down, direct laryngoscopy can be performed with the patient seated semi-erect and the laryngoscopist on a stepstool behind the patient. [11]

The laryngoscope is grasped in the *left* hand with the blade directed toward the patient from the hypothenar aspect of the operator's hand. The patient's lower lip is drawn down with the right thumb, and the tip of the laryngoscope is introduced into the *right* side of the mouth. The blade is slid along the right side of the tongue, gradually displacing the tongue toward the left as the blade is moved to the center of the mouth. If the blade is initially placed in the middle of the tongue, the tongue will fold over the lateral edge of the blade and obscure the airway. Placing the blade in the middle of the tongue and failure to move the tongue to the left are two common errors preventing visualization of

the vocal cords (Fig. 2-7 *A-C*).

As the blade tip approaches the base of the tongue, the operator exerts a force along the axis of the laryngoscope handle, lifting upward and forward at a 45° angle. The epiglottis should come into view with this maneuver. It may help to have an assistant retract the cheek laterally to further expose the laryngeal structures. Do not bend the wrist; bending the wrist can result in dental injury because the teeth may be used as a fulcrum for the blade.

The step following visualization of the epiglottis depends on which laryngoscope blade is used. With the *curved* blade, the tip is placed into the vallecula, the space between the base of the tongue and the epiglottis. Continued anterior elevation of the base of the tongue and the epiglottis will expose the vocal cords. If the blade tip is inserted too deeply into the vallecula, the epiglottis may be pushed down to obscure the glottis. [6] When using the *straight* blade, the tip is inserted under and slightly beyond the epiglottis, directly lifting this structure. The jaw and larynx are literally suspended by the blade. If the straight blade is placed too deeply, the entire larynx may be elevated anteriorly and out of the field of vision. Gradual withdrawal of the blade should allow the laryngeal inlet to drop down into view. If the blade is deep and posterior, the lack of recognizable structures indicates esophageal passage; gradual withdrawal should permit the laryngeal inlet to come into view. The use of the curved and straight laryngoscope blades is illustrated in Figure 2-7 *D* and *E*.

Proper neck positioning and pressure (cephalad, dorsally, and rightward) on the larynx by an assistant will facilitate visualization and intubation of an anterior larynx. If needed, suctioning is performed at this point. If the vocal cords are still not seen, consider using a tracheal tube introducer (Smiths Industries Medical Systems, Keene, NH). This device, also known as the "elastic gum bougie," is a long, semirigid introducer that is placed, using the laryngoscope, through the laryngeal inlet and into the trachea. [12A] The tracheal tube is then passed over the introducer and the introducer is withdrawn. If resistance is met in passing the tracheal tube, rotate the tube 90° counterclockwise and advance the tube.

**Tube passage.**

Once the vocal cords have been visualized, the final and most important step, tube passage under direct vision through the vocal cords and into the trachea, is performed. The tube is held in the operator's right hand and introduced from the right side of the patient's mouth. The tube is advanced toward the patient's larynx at an angle, not parallel with or down the slot of the laryngoscope blade. This way, the operator's view of the larynx is not obstructed by the hand or the tube until the last possible moment before the tube enters the larynx. The tube should be passed during inspiration, when the vocal cords are maximally open. It enters the trachea when the cuff disappears through the vocal cords. The tube is advanced 3 to 4 cm beyond this point. It is not enough to see the tube and the cords; the tube must be seen *passing through* the vocal cords to ensure tracheal placement.

When the vocal cords are stimulated, laryngospasm-- the persistent contraction of the

adductor muscles of the vocal cords--may prevent passage of the tube. Inadequate anesthesia is often the cause. Pretreatment with topical lidocaine decreases the likelihood of this occurring. Two percent or 4lidocaine is sprayed directly on the cords. An infrequent but effective route for achieving tracheal anesthesia is via transtracheal puncture, injecting a bolus of 3 to 4 mL of lidocaine through the cricothyroid membrane. Laryngospasm is usually brief and is often followed by a gasp. The operator should be ready to pass the tube at this moment. Occasionally, the spasm is prolonged and needs to be broken with sustained anterior traction applied at the angles of the mandible--the jaw lift. At no time should the tube be forced, because permanent damage to the vocal cords may result. Consideration should be given to using a smaller tube. Prolonged, intense spasm may ultimately require muscle relaxation with a paralyzing drug (see Chapter 3) . The pediatric patient is far more prone to laryngospasm than is an adult. [12] In a child, if vocal cord spasm prevents tube passage, a chest thrust maneuver may momentarily open the passage and permit intubation.

**Positioning and securing the tube.**

The endotracheal tube should be secured in a position that minimizes both the chance of inadvertent endobronchial intubation and the risk of extubation. The tip should lie in the midtrachea with room to accommodate neck movement. Because tube movement with both neck flexion and extension averages 2 cm, the desired range of tip location is between 3 and 7 cm above the carina. [14]

On a radiograph, the tip of the tube should ideally be 5 ± 2 cm above the carina when the head and neck are in a neutral position. On a portable radiograph, the adult carina overlies the fifth, sixth, or seventh thoracic vertebral body. If the carina is not visible, it can be assumed that the tip of the tube is properly positioned if it is aligned with the T3 or T4 vertebra. In children, the carina is more cephalad than in the adult, but it is consistently situated between T3 and T5. In children, T1 is used as the reference point for the tip of the endotracheal tube. 44 [15]

An estimate of the proper depth of tube placement can be derived from the following formulas, the lengths representing the distance from the tube tip to the upper incisors in children  and from the upper incisors [18] or the corner of the mouth [19] in adults:

Adults: Tracheal tube depth (cm) = 21 cm (women)
Tracheal tube depth (cm) = 23 cm (men)

In adults, this method has been shown to be more reliable than auscultation in determining the correct depth of placement. [18]

The cuff is inflated to the point of minimal air leak with positive-pressure ventilation. In an emergency intubation, 10 mL of air is placed in the cuff, and inflation volume is adjusted after the patient's condition is stabilized.

After tracheal tube placement, both lungs are auscultated under positive-pressure ventilation. Care is taken to auscultate laterally because midline auscultation may lead

to an erroneous impression of tracheal placement when the tube is actually in the esophagus. With the tube in position and the cuff inflated, the tube is secured in place. Commercial endotracheal tube holders, adhesive tape, or umbilical (nonadhesive cloth) tape can be attached securely to the tube and around the patient's head (Figs. 2-8 and 2-9) (Figure Not Available) . The tube should be positioned in the corner of the mouth, where the tongue cannot expel it. This position is also more comfortable for the patient and allows for suctioning. A bite-block or oral airway to prevent endotracheal tube crimping or damage from biting is commonly incorporated into the system used to secure the tube.

### Infants and Children

Appreciation of the anatomic differences between children and adults is helpful when intubating the pediatric patient (Table 2-3 and Fig. 2-10) (Figure Not Available) . Infants' proportionately larger head naturally places them in the "sniffing position," so a towel under the occiput is rarely necessary. The large head can even result in a posterior positioning of the larynx that prevents visualization of the vocal cords; a small towel under the child's shoulders should correct this problem. The head also may be floppy, and it can be stabilized by an assistant during intubation. The child's increased tongue-to-oropharynx ratio and shorter neck hinder forward displacement of the tongue and, coupled with a long U-shaped epiglottis, can make visualization of the glottis difficult.

Consequently, direct laryngoscopy in the infant and young child is generally best performed with a straight blade: Miller size 0 for premature infants, size 1 for normal-sized infants, and size 2 for older children. The infant's larynx lies higher and relatively more anterior. One can have an assistant lightly apply laryngeal pressure, or the operator can use the little finger of the hand holding the laryngoscope blade for this purpose (Fig. 2-11) . If no laryngeal structures are visible after laryngeal pressure, the blade should be gradually withdrawn, because inadvertent advancement of the blade into the esophagus is a common error.

## Confirmation of Tracheal Intubation

### Clinical Assessment

The best assurance of tracheal placement is for the operator to see the tube pass through the vocal cords (Table 2-4) . Absent or diminished breath sounds, vocalization, increased abdominal size, and gurgling sounds during ventilation are clinical signs of esophageal placement. However, *esophageal placement is not always obvious*. One may hear "normal" breath sounds if only the midline of the thorax is auscultated. One way to clinically assess tracheal placement after a ventilation or during spontaneous respiration is to note whether air is felt or heard to exit through the tube following cuff inflation. If the tidal volume is adequate, the exit of air should be obvious. It is important to note that when an appropriately sized tube is placed in the trachea, the patient cannot groan, moan, or speak. Any vocalization suggests esophageal placement.

Asymmetrical breath sounds indicate probable main stem bronchus intubation. Due to

the angles of takeoff of the main bronchi and the fact that the carina lies to the left of the midline in adults, right main stem intubation is most common and is indicated by decreased breath sounds on the left side. When asymmetrical sounds are heard, the cuff should be deflated and the tube withdrawn until equal breath sounds are present. Bloch and colleagues report accurate pediatric tracheal positioning if after noting asymmetrical breath sounds, the tube is withdrawn a defined distance beyond the point at which equal breath sounds are first heard--2 cm in children younger than 5 years and 3 cm in older children. [20]

**Esophageal Detector Device**

An aspiration technique used to determine endotracheal tube location was first described by Wee in 1988. [21] The technique takes advantage of the difference in tracheal and esophageal resistance to collapse during aspiration to determine location of the tip of the tracheal tube. Following intubation, a large syringe is attached to the end of the endotracheal tube and the syringe plunger is withdrawn. If the tube is correctly placed in the trachea, the plunger will pull back without resistance as air is aspirated from the lungs. However, if the tracheal tube is in the esophagus, resistance is felt when the plunger is withdrawn, because the pliable walls of the esophagus collapse under the negative pressure and occlude the end of the tube. Another device using the same principle as syringe aspiration is the self-inflating bulb (e.g., Ellick's device).

Wee first reported use of an esophageal detector device in the operating room. [21] The tube was correctly identified in 99 of 100 cases (51 esophageal, 48 tracheal). The device result was considered equivocal in the remaining tracheal tube. The tube was removed and found to be nearly totally occluded with purulent secretions. Slight resistance was noted in one patient with a right main stem intubation; resistance decreased when the tube was pulled back. Before use, the esophageal detector device must always be checked for air leaks. If any connections are loose, the leak may allow the syringe to be easily withdrawn, mimicking tracheal location of the tube.

Wee recommends the following guidelines in using the aspiration technique: apply constant, slow aspiration to avoid tube occlusion from tracheal mucosa drawn up under high negative pressure. If the tracheal tube is correctly placed, 30 to 40 mL of air can be aspirated without resistance. If air was initially aspirated and then some resistance is encountered, the tracheal tube should be pulled back between 0.5 and 1.0 cm and partially rotated. This takes the tube out of the bronchus, if it has been placed too deeply, and changes the orientation of the bevel if the tube has been temporarily occluded with tracheal mucosa. Air is easily aspirated if the tube was in the trachea, but repositioning will make no difference if the tube was in the esophagus. The syringe aspiration technique can be used before or after ventilation of the patient. Continuous cricoid pressure should be applied pending tube confirmation. Inflation of the tube cuff will have no effect on the reliability of the test. [22] This device is reliable, rapid, inexpensive, and easy to use. Jenkins reported good success with physician use of the aspiration technique to confirm placement of emergency department and out-of-hospital intubations. [23]

A squeeze-bulb aspirator can be used as an alternative to the syringe technique. [24] [25]

The bulb is attached to the endotracheal tube and squeezed; if the tube is in the esophagus, it is often accompanied by a flatus-like sound followed by absent or markedly delayed refilling. Insufflation of a tube in the trachea is silent, with instantaneous refill. An early study with the Ellick's evacuator bulb device reported that 87% of esophageal tubes were identified. [24] A later study using a slightly different bulb device (Respironics, Murrysville,

| | | TABLE 2-3 -- Comparison of the Airway in the Adult and Child | |
|---|---|---|---|
| **Comparison** | **Child** | **Adult** | **Clinical Consequences or Adjustments for Child** |
| Head | Proportionately larger (up to about age 10 yr) | Proportionately smaller | Child naturally in sniffing position when supine; do not place towel under occiput; may benefit from elevation of shoulders; large head may be "floppy," requiring assistant to hold head still during intubation |
| Teeth | Easily knocked out | Stable unless decay or trauma is a factor | Teeth may be knocked out and aspirated or forced into trachea |
| Tonsils or adenoids | Large and friable | Generally not a problem | Nasotracheal intubation in child may cause excessive bleeding: adenoid or tonsil tissue may plug endotracheal tube or cause airway obstruction from aspiration |
| Tongue | Relatively large | Relatively smaller | Difficult to displace the tongue anteriorly in child: should consider using straight blade |

| | | | |
|---|---|---|---|
| Larynx | Opposite C2, C3 | Opposite C4-C6 | The more superiorly located larynx, the "anterior" larynx, is more difficult to visualize: consider using straight blade |
| Epiglottis | U-shaped, shorter, stiffer | Flatter, more flexible | Epiglottis more difficult to manipulate in child, may fold down and obstruct view with curved blade: consider using straight blade |
| Vocal cords | Concave upward. Anterior attachment of cords lower than posterior, creating a slant | Horizontal | Concave shape does not affect intubation, but it may affect ventilation: for partial airway obstruction or to break laryngospasm: consider positive pressure ventilation with jaw lift to open arytenoids; anterior superior slant of the vocal cords may cause the endotracheal tube to hang up on anterior commissure as it passes into larynx: rotate tube 90° counterclockwise Overextension of the neck may cause partial airway obstruction due to airway collapse |
| Length of trachea | Relatively short | Relatively longer | Short trachea increases likelihood of main stem bronchus intubation: follow the formula for correct depth of placement (cm depth = 0.5 age (yr) + 12) measured from the corner of the mouth; double black line on endotracheal tube should pass just beyond the cords |

| Airway diameter | Relatively smaller; smallest diameter is at the cricoid ring | Relatively larger; smallest diameter is between vocal cords | Laryngoscope-induced trauma, edema, and foreign material will significantly alter airway diameter: be gentle, extremes of flexion or extension may kink airway; if trouble with bag-valve-mask ventilation, reassess the degree of head flexion or extension; cricoid pressure may cause complete airway obstruction; endotracheal tube may pass through cords but be too small to pass through cricoid ring: if unable to pass into trachea, use the next smaller tube |
|---|---|---|---|
| Residual lung capacity | Relatively smaller | Relatively larger | Child may become hypoxic more quickly than adult. Closely monitor $O_2$ saturation and avoid prolonged periods without ventilation |

| **TABLE 2-4** -- Assessing Proper Tube Placement | |
|---|---|
| **Test** | **Interpretation** |
| Observe tube pass through the vocal cords | Accurate way to ensure placement; if in doubt, look again after intubation |
| Auscultation of breath sounds over the chest | May be misleading, especially if only midline is examined; listen in both axillae |
| Auscultation over the stomach | Gurgling indicates esophageal placement |

| | |
|---|---|
| Condensation (fog) forms inside tube with each breath | Quite reliable |
| Observe chest rise with positive pressure and fall with release | Generally reliable but may be absent in patients with small tidal volume or severe bronchospasm |
| Feel air exiting from end of tube following inflation | Reliable |
| Air remains in lung after end of tube is occluded and exits when occlusion is removed | Reliable but one may "ventilate" a closed area of the esophagus |
| Ask patient to speak; listen for moaning or other sounds | If tube is in proper place, no sound is possible |
| Chest radiograph | Generally reliable but can be misleading |
| End-tidal carbon dioxide measurements | Carbon dioxide not persistently detected if esophagus is intubated |
| Aspiration technique | Tracheal location with patent tube if 30-40 mL air is aspirated without resistance; probable esophageal location if unable to aspirate syringe easily or delayed bulb refill |
| Fiberoptic bronchoscope | Reliable if tracheal rings are seen down endotracheal tube |

| Lighted stylet down endotracheal tube | Reliable if transillumination seen in low midline neck |
| --- | --- |

Pa) found that all 45 esophageal tubes were detected. [25] The device is cheap and easy to use and can be operated single-handedly in <5 seconds.

Confusion may occur if the esophageal tube is tested more than once, because subsequent inflations may be silent. On repeated assessments, a false-positive refilling of the bulb may occur due to instillation of air during the first attempt. This observation has led to a recommendation that the bulb be compressed *before* it is attached to the endotracheal tube. Delayed though complete refilling may occur with bronchial tube placement or in the more pliable pediatric airway. The bulb suction modification of the aspiration technique has not been studied as thoroughly as the syringe technique.

**End-Tidal $CO_2$ Detector Devices**

A high level of $CO_2$ in exhaled gas is the physiologic basis for capnography and the principle on which end-tidal $CO_2$ ($ET_{CO_2}$) detectors was developed (see Chapters 6 and 16) . The most commonly available devices for emergency use are colorimetric indicators responding to $CO_2$ levels of gas flowing through the device when placed on the tracheal tube adapter. The typical device displays two extreme colors indicating a low level of $CO_2$ in esophageal intubation and another color in tracheal intubation. An intermediate color is indeterminate. Hand-held quantitative or semiquantitative electronic $CO_2$ monitors are also available.

A multicenter study of a colorimetric device demonstrated an overall sensitivity of 80% and a specificity of 96%. [26] In patients with spontaneous circulation and the tracheal tube cuff inflated, the sensitivity and specificity rose to 100%. The poor sensitivity seen in cardiac arrest (69%) is due to the fact that low exhaled $CO_2$ levels are seen in both very-low-flow states and in esophageal intubation. The device must therefore be used with caution in the cardiac arrest victim. Levels of $CO_2$ return to normal after return of spontaneous circulation in these patients. Further, colorimetric changes may be difficult to discern in reduced lighting situations, and secretions can interfere with the color change. Regardless of the monitoring device, patients in cardiac arrest should be ventilated for a minimum of 6 breaths prior to taking a reading, because recent ingestion of carbonated beverages can result in spuriously high $CO_2$ levels with esophageal intubation. [27]

**Comparison of Detector Devices**

In the setting of *spontaneous circulation*, both syringe aspiration and $ET_{CO_2}$ detection are highly reliable means of excluding esophageal intubation. A comparison of the techniques with clinical assessment was carried out in the animal laboratory, with measurement of the speed and accuracy of determination of tube placement. [28] Both

the syringe esophageal detector device and ET$co_2$ detection were highly accurate, approaching 100%. The esophageal detector device was more rapid with determination in 13.8 seconds vs 31.5 seconds for ET$co_2$ detection. The detector device remained accurate when air was insufflated into the esophagus for 1 minute, simulating unrecognized esophageal placement. Clinical assessment alone yielded an alarming 30% rate of misidentifying an esophageal tube as being in the trachea. In the setting of *cardiac arrest*, the aspiration method is more reliable than $CO_2$ detection, because its accuracy is not dependent on the presence of blood flow.

## Complications

Prolonged efforts to intubate may result not only in hypoxia but also in cardiac decompensation. Pharyngeal stimulation can produce profound bradycardia or asystole; when it is feasible, an assistant should view the cardiac monitor during intubation of a patient who has not suffered cardiac arrest. Atropine should be available to reverse vagal-induced bradycardia that may occur secondary to suctioning or laryngoscopy. Prolonged pharyngeal stimulation also may result in laryngospasm, bronchospasm, and apnea.

The maximum interval allowable for routine intubation of the apneic patient is 30 seconds. As a guide, one should limit the time of an intubation attempt to the amount of time a single deep breath can be held. This is especially important in a child, because the functional residual capacity of a child's lungs is less than that of an adult. Failure to achieve control within this time frame demands an interval of bag-valve-mask ventilation before intubation is attempted again. The use of preoxygenation to minimize hypoxia is strongly recommended. An oxygen saturation monitor can also be used to monitor explicitly for hypoxia. Assuming optimal preoxygenation of the patient to >98% $O_2$ saturation, attempts at intubation should be halted until the patient is reoxygenated whenever the $O_2$ saturation drops below 92%, equal to a P$O_2$ of about 60 to 65 mm Hg. When ventilation is not achievable, irreversible brain damage can result within minutes. Therefore, the maximum interval allowable for conservative airway management maneuvers is about 3 minutes; one must then choose alternative methods (see Chapter 1 and 4) .

One should check for loose or missing teeth before and after orotracheal intubation. Any avulsed teeth not found in the oral cavity warrant a postlaryngoscopy chest film to rule out aspiration of a tooth. Swallowed teeth are of no consequence. In a study of 366 patients, McGovern and coworkers found broken teeth to be the most common complication of laryngoscopy. [29] Laceration of the mucosa of the lips, especially the lower lip, may occur if adequate care is not taken. Tracheal or bronchial injuries are rare but serious, usually occurring in infants and the elderly as a result of decreased tissue elasticity. [30] Vomiting with aspiration of gastric contents is another serious complication that can occur during intubation.

The most devastating complication of tracheal intubation is unrecognized esophageal intubation. Assessment of tube position should be the first step in the emergency department evaluation of patients who have undergone out-of-hospital intubation. The best assurance of tracheal placement is for the operator to see the tube pass through

the vocal cords. Techniques to assess tube placement are discussed earlier. Another method of reliably determining tracheal tube location uses the fiberoptic scope. Passage of the scope through the tube with visualization of tracheal rings confirms endotracheal placement as well as the position within the trachea. The placement of a lighted stylet down the tracheal tube and successful transtracheal illumination also reliably predicts tracheal positioning. [31]

A chest radiograph should be taken shortly after the intubation to confirm tube placement and position. Bissinger and coworkers noted that endobronchial intubation was clinically unrecognized without a chest film in 7% of out-of-hospital intubations. [32] In addition to hypoxia, delayed tube repositioning can lead to unilateral pulmonary edema. [33] Persistent asymmetrical breath sounds after appropriate tube positioning suggests unilateral pulmonary pathology (e.g., main stem bronchus obstruction, pneumothorax, or hemothorax).

If an endotracheal tube is removed from the esophagus, vomiting may occur. This should be anticipated and suction readied. Cricoid pressure should be applied during tube removal and maintained until intubation is successful. Alternatively, the first tube can be left in the esophagus to serve as temporary gastric venting until tracheal intubation is achieved.

A persistent air leak during ventilation usually means one of three things: (1) the cuff is leaking because of damage to the balloon, (2) the cuff is positioned above or between the vocal cords, or (3) the pilot balloon is leaking. If the cuff is leaking, the tracheal tube must be replaced (see Changing Tracheal Tubes). If the pilot balloon is determined to be leaking, however, this can usually be remedied without changing the tube. [34] An incompetent 1-way balloon valve can be fixed by placing a stopcock into the inflating valve. Reinflation of the cuff followed by shutting off the stopcock should solve the problem. If the leak involves the pilot balloon itself, or if the distal inflation tube has been inadvertently severed, cut off the defective part and slide a 20-ga catheter into the inflation tube. Then connect the stopcock to the catheter, inflate the cuff, and close the stopcock.

Tracheal stricture used to be a significant late complication of long-term intubation with low-volume high-pressure cuffs. The standard use of high-volume low-pressure cuffs has markedly decreased the incidence of this complication. [35] Tubes with high-pressure cuffs are obsolete and should be avoided.

**Summary**

Orotracheal intubation is the mainstay of definitive airway management. In the comatose patient, it is usually accomplished rapidly and without difficulty. The easy intubation is frequently successful in the hands of the novice; the difficult intubation often proves challenging even for the experienced operator. Rapid-sequence intubation has increased the use of orotracheal intubation as the first-line approach in a variety of clinical situations and settings (see Chapter 3) . Once the patient's breathing and protective reflexes are removed, however, the operator has the supreme responsibility of safely reestablishing them. A mastery of the technique of orotracheal intubation is

essential.

# MODIFIED OROTRACHEAL INTUBATION

## Intubation with an Intermediate Airway in Place

### Esophageal Obturator/Gastric Tube Airway in Place

The unconscious patient who requires ventilatory assistance may benefit from the temporary use of the esophageal obturator

airway (EOA) or similar device, as described in Chapter 1 . Although this may be an effective means of ventilation, it is at best a temporary measure. The patient experiencing upper airway hemorrhage with the EOA in place may have oropharyngeal blood insufflated into the trachea. Also, an endotracheal tube is the preferred airway, because with endotracheal intubation the airway is more secure and ventilation more convenient. Although the EOA may allow rapid airway support until cervical spine injury can be ruled out, it is recommended that the EOA not be left in place for more than 2 hours.

Replacement of the EOA with an orotracheal tube requires appropriate care. Removal of the esophageal cuff before placement of the endotracheal tube is fraught with danger. Spontaneous gastric regurgitation often occurs on EOA removal. The rescuer must therefore learn to perform endotracheal intubation around the EOA to protect the patient from aspiration.

The patient is hyperventilated through the EOA before intubation is attempted around it. The EOA mask is then removed, and the EOA tube is moved to the *left* side of the patient's mouth. Laryngoscopy and intubation are then performed in the usual fashion. If resistance to passage of the tracheal tube is met, the volume of the EOA balloon should be reduced, because the balloon may be producing distortion of the larynx. Next, the operator deflates the EOA balloon completely and slides it out of the patient's esophagus. If resistance is met, the operator must be sure that the esophageal cuff has been deflated completely.

### Esophageal-Tracheal Combitube (ETC) in Place

Combitubes placed in the esophagus will generally require replacement with a tracheal tube. The inflated pharyngeal balloon prevents tracheal intubation around this airway. This proximal balloon must be deflated before attempting tracheal intubation. If intubation is still not possible, the ETC may need to be removed; the stomach should first be emptied via a gastric tube placed through the esophageal port of the airway. Suction is readied, the distal balloon is deflated, and the patient is quickly intubated. This maneuver poses an added risk over that associated with the esophageal obturator intermediate airway placement (i.e., EOA and EGTA--see Chapter 1) .

**Laryngeal Mask Airway in Place**

The trachea can often be intubated with the laryngeal mask airway (LMA) left in place. The various approaches to intubation with an LMA in place are discussed in Chapter 1 (see Figs. 1-10 (Figure Not Available) and 1-11) (Figure Not Available) .

## Bullard Laryngoscope Use

A recent development for intubating the difficult airway is the Bullard laryngoscope, an anatomically shaped rigid fiberoptic laryngoscope that provides an indirect view of the larynx (Fig. 2-12) (Figure Not Available) . It was design`ed to aid in intubating the difficult airway; and because no manipulation of the neck is necessary, it is especially well suited for the patient with potential cervical spine injury. Indeed, in the anesthetized patient, the Bullard laryngoscope has been found to cause less head extension and cervical spine extension than conventional laryngoscopes do. [36] The recent addition of an intubating stylet attached to the laryngoscope has resulted in increased ease and speed of intubation, and the technique appears to be effective regardless of the patient's head and neck anatomy. [37] Because alignment of the oropharyngeal and laryngeal axes is not required, the Bullard laryngoscope offers the advantage provided by a conventional fiberoptic scope but requires less training to gain proficiency in its use. [38]

**Indications and Contraindications**

The Bullard laryngoscope is indicated in patients with anticipated difficult airways who require definitive airway control. It can be used in awake as well as unresponsive patients. [39] The total inability to open the mouth is a contraindication to the use of this laryngoscope. However, because the Bullard laryngoscope follows the contour of the mouth and hypopharynx, only 2 cm of occlusal opening is necessary for the introduction of the scope plus endotracheal tube for intubation.

**Procedure**

The technique for introducing the Bullard laryngoscope blade is similar to that for direct laryngoscopy. The operator, who is at the head of the patient, opens the mouth with the left thumb while holding the head stable. As the scope blade is introduced into the oropharynx, the handle is rotated to follow the curve of the hypopharynx until the handle is fully vertical. The tip of the blade can be used to lift the epiglottis, but visualization of the larynx is usually possible without this maneuver. Only minimal force is exerted along the axis of the handle. Intubation of the larynx can be accomplished using a styletted endotracheal tube or an endotracheal tube with a directional tip (Endotrol; Mallinckrodt, Critical Care, Glens Falls, NY). The technique is generally successful when using the new Bullard intubating stylet. [37]

Awake intubation using the Bullard laryngoscope can be performed comfortably using topical anesthesia and light IV sedation. [39] Adult and pediatric Bullard laryngoscopes

are available, and the scope has been used successfully in neonates. [38] The Bullard scope can also be used in conjunction with nasotracheal intubation and has the advantage of requiring only 6 mm of mouth opening through which to insert the blade. [40]

### Complications

The major difficulty in using the Bullard laryngoscope is the inability to visualize the larynx because of blood, emesis, or secretions. Another reason for failure is the inability to place the blade tip under the epiglottis. [37]

### Summary

The Bullard scope is useful in the difficult airway uncomplicated by blood and excessive secretions. In the all-too-common setting of blood and secretions, however, the inability to visualize the vocal cords significantly limits the utility of this device in emergency airway management.

## NASOTRACHEAL INTUBATION

Nasotracheal intubation was first described by Magill in the 1920s and the basic technique has changed little over the years. Modifications have been described that increase the success rate and limit complications. The tube may be placed blindly or with the aid of a laryngoscope or bronchoscope. Blind nasotracheal intubation can be one of the more technically demanding airway approaches, with the outcome being heavily dependent on the skill and experience of the operator. The primary advantage of blind nasotracheal intubation is that it minimizes neck movement and does not require opening the mouth.

### General Indications and Contraindications

Nasotracheal intubation is technically more difficult than oral intubation, but it has definite advantages. It is especially suitable for the patient with a short, thick neck or other anatomic characteristics that would make orotracheal intubation difficult. Patients with clenched teeth or suspected cervical spine injury can be intubated with minimal preparation. Cervical spine films, jaw spreading, or paralyzing agents as preliminaries to airway control are unnecessary.

Blind nasotracheal intubation is possible with the patient in the sitting position, a distinct advantage when intubating the patient with congestive heart failure who cannot tolerate lying flat. In fact, patients in respiratory distress are the easiest to intubate blindly because their air hunger results in increased abduction of the vocal cords, which facilitates tube entry into the trachea. The drug overdose patient with a decreased level of consciousness is a candidate for nasotracheal intubation. These patients are often intubated before gastric lavage and may be sufficiently awake to make orotracheal intubation difficult without paralyzing agents.

A nasotracheal tube has advantages that extend beyond the immediate difficulties of airway control. The patient cannot bite the tube or manipulate it with the tongue. Oral injuries may be cared for without interference by the tube. A nasotracheal tube is more easily stabilized and generally easier to care for than an orotracheal tube. It is better tolerated by the patient, permitting easier movement in bed, and produces less reflex salivation than do oral tubes.

Nasal intubation should be avoided in patients with severe nasal or midface trauma. In the presence of a basilar skull fracture, a nasotracheal tube may inadvertently enter the brain through a basilar skull fracture. [41] The technique should be avoided in patients in whom thrombolytic therapy is being considered. Nasal intubation is relatively contraindicated if the patient is taking anticoagulants or is known to have a coagulopathy.

## Blind Placement

Blind nasotracheal intubation is the most common form of nasotracheal intubation in the emergency setting. Danzl and Thomas reported a success rate of 92% in a large series of emergency department patients. [42]

### Indications and Contraindications

Any patient requiring airway control who has spontaneous respirations is a candidate for blind nasotracheal intubation. Specific indications that favor this approach over others are (1) short, thick neck, (2) inability to open the mouth, (3) inability to move the neck, (4) gagging or resisting the use of the laryngoscope, and (5) oral injuries.

Apnea is the major contraindication to blind nasotracheal intubation. Attempts to place the tube without respirations as a guide are futile. Relative contraindications include basilar skull fracture and nasal injury. [41] [43] Furthermore, significant bleeding may occur if the patient is receiving anticoagulants or has a coagulopathy. Blind nasotracheal intubation should be avoided in patients with expanding neck hematomas. Patient combativeness, if not controlled with sedation, is also a contraindication.

Some would argue that the inability to open the mouth is a relative contraindication, because emesis may be induced that could not be cleared. The operator must exercise judgment in the individual case and be prepared to use neuromuscular blocking agents or to bypass the upper airway with a surgical technique if such a complication develops.

### Procedure

The patient is placed in the "sniffing" position with the proximal neck slightly flexed and the head extended on the neck. In preparation for intubation, the operator constricts the nasal mucosa of both nares, using either 0.25 to 1.0% phenylephrine drops, oxymetazoline (Afrin) spray, or 4% cocaine spray. Topical anesthesia of the nares, oropharynx, and hypopharynx with lidocaine spray (10%) is also indicated if time

permits. If available, cocaine is ideal because it is both a vasoconstrictor and an anesthetic; caution is necessary in hypertensive patients. The most patent nostril is chosen. In the cooperative patient, this can be determined simply by occluding each nostril and asking the patient which one is easier to breathe through. The most patent nostril can also be identified by direct vision, or by gently inserting a gloved finger lubricated with viscous lidocaine, full length into the nostrils. If time is not an issue, an effective method to dilate the nasal cavity and administer the anesthetic is to pass a lidocaine gel-lubricated nasopharyngeal airway (nasal trumpet) into the selected nostril. This

airway is left in place for several minutes, and progressively larger trumpets are introduced.

After preparation of the nostril, a well-lubricated endotracheal tube with a 7.0 or 7.5 mm ID is inserted along the floor of the nasal cavity. The tube is *not* directed cephalad, as one might expect from the external nasal anatomy, but rather is directed straight back toward the occiput, corresponding with the nasal floor. Twisting the tube may help bypass soft tissue obstruction in the nasal cavity. It is sometimes recommended that the bevel of the tube be oriented toward the septum to avoid injury to the inferior turbinate. However, such an event is rare. At 6 to 7 cm, one usually feels a "give" as the tube passes the nasal choana and negotiates the abrupt 90° curve required to enter the nasopharynx. This is the most painful and traumatic part of the procedure and must be done gently. If resistance is encountered that persists despite continued gentle pressure and twisting of the tube, the passage of a suction catheter down the tube and into the oropharynx may allow for successful passage of the tube over the catheter. [44] If this fails, the other nostril should be tried. In an attempt to avoid this difficulty from the outset, a controllable-tip tracheal tube (Endotrol, Mallinckrodt Medical Inc, St Louis) may be used that allows the operator to increase the flexion of the tube and facilitates passage past this tight curve. One study found the Endotrol tube to enhance first attempt success with blind nasotracheal intubation. [45]

As the tube is advanced through the oropharynx and hypopharynx and approaches the vocal cords, breath sounds from the tube become louder, and fogging of the tube may occur. At the point of maximal breath sounds, the tube is lying immediately in front of the laryngeal inlet. The tube is most easily advanced into the trachea during inspiration because that is when the vocal cords are maximally open. As the patient begins to breathe in, the tube is advanced in one smooth motion. If a gag reflex is present, the patient usually coughs and becomes stridulous during this maneuver, suggesting successful tracheal intubation. The absence of such a response should alert the operator to probable esophageal passage. If there is a delay in advancing the tube, oxygen can be added to the end of the tube to increase inspired oxygen. Once the tube is in the trachea, moaning and groaning should cease. If they continue, esophageal intubation is likely. Breath sounds coming from the tube and tube fogging are other signs of endotracheal placement. Reflex swallowing during blind nasotracheal intubation may direct the tube posteriorly toward the esophagus. If this occurs, the conscious patient should be directed to stick out the tongue to inhibit swallowing and prevent consequent movement of the larynx. Application of laryngeal pressure may also help

avoid esophageal passage.

Following intubation, both lungs are auscultated while positive-pressure ventilation is applied. If only one lung is being ventilated, the tube is withdrawn until breath sounds are heard bilaterally. The optimum distance from the external nares to the tube tip is about 28 cm in males and 26 cm in females. [45A] After verification of tracheal placement, the cuff is inflated and the tube is secured.

**Technical Difficulties**

The nasotracheal tube may slide smoothly through the hypopharynx and into the trachea on the first pass. Unfortunately, this is not always the case; in the operating room, the first attempt was successful in <50% of cases. [46] When the initial pass is unsuccessful, there are 4 potential locations of the tip of the tube: (1) anterior to the epiglottis in the vallecula, (2) on the arytenoid or vocal cord, (3) in the piriform sinuses, or (4) in the esophagus.

Observation and palpation of the soft tissues of the neck during attempted passage of the nasotracheal tube are helpful in determining the location of the misplaced tube. This is ideally done by the operator but may also be performed by an experienced attendant. Before reattempting placement, the tube is withdrawn slightly; it is not removed from the nose, because this will create additional trauma to the nasal soft tissues. The possibility of cervical spine injury must be kept in mind when considering corrective maneuvers. Any maneuver that moves the neck significantly should not be used if alternatives are available. Methods for achieving success when difficulties with tube placement are encountered include the following:

**Anterior to the epiglottis.**

Difficulty advancing the tube beyond 15 cm or palpation of the tube tip anteriorly at the level of the hyoid bone suggests an impasse anterior to the epiglottis in the vallecula. Withdrawing the tube 2 cm, decreasing the degree of neck extension, and readvancing the tube will frequently remedy this problem.

**Arytenoid cartilage and vocal cord.**

Contrary to the classic teaching, [47] recent studies have demonstrated a propensity for a nasotracheal tube, when placed through the right nares, to lie posteriorly and to the right as it approaches the larynx. [40] [48] It is not surprising, then, that the most common obstacles to advancement of the nasotracheal tube are the right arytenoid and vocal cord. No data are available on the common obstacles encountered if the tube is placed in the left nares. If the tube appears to be hanging up on firm, cartilaginous tissue, withdraw the tube 2 cm, rotate it 90° counterclockwise, and readvance the tube. This maneuver orients the bevel of the tube posteriorly and frequently results in successful passage (Fig. 2-13) (Figure Not Available) . Another technique is to pass a suction catheter down the tube; it often will pass through the larynx without difficulty and the

tube can then be advanced over the catheter (Fig. 2-14) (Figure Not Available) . [49] [50]

**Piriform sinus.**

Bulging of the neck lateral and superior to the larynx indicates tube location in a piriform sinus. The tube should be withdrawn 2 cm, rotated slightly away from the bulge, and readvanced. An alternate method is to tilt the patient's head toward the side of the misplacement and reattempt placement. [51]

**Esophageal placement.**

Esophageal placement is indicated by a smooth passage of the tube with the loss of breath sounds. The larynx may be seen or felt to elevate as the tube passes under it. Assisted ventilation will usually produce gurgling sounds when the epigastrium is auscultated. The tube should be withdrawn until breath sounds are clearly heard, and passage should be reattempted while pressure is applied to the cricoid. Increased extension of the head on the neck during placement may help. If attempts continue to result in esophageal misplacement, the following maneuver may result in successful tracheal intubation: from the precise point at which breath sounds are lost, the endotracheal tube is withdrawn 1 cm. The cuff is inflated with 15 mm of air, resulting in an elevation of the tube off the posterior pharyngeal wall and angling it toward the larynx. The tube is then advanced 2 cm; continued breath sounds indicate probable intralaryngeal location. At this point, the cuff is deflated and the endotracheal tube is advanced into the trachea (Fig. 2-15) (Figure Not Available) . This technique may be particularly useful in the patient with cervical spine injury, because it requires no manipulation of the head or neck. [52] This maneuver, when used on the *first* pass in 20 patients in the operating room, was successful in 75% of cases. [46] One should bear in mind, however, that these patients were paralyzed and thus did not experience the laryngospasm that may be encountered in a breathing patient. The use of topical anesthesia is recommended. Alternatively, if a controllable-tip endotracheal tube (Endotrol) is used, the tip can be flexed anteriorly to help avoid esophageal placement. [45] Remember that the tip is very responsive to pulling on the ring. A common mistake is to exert too much force on the ring, resulting in the tube curling up short of the larynx, thus preventing tube advancement.

**Laryngospasm.**

Laryngospasm is common when attempting nasotracheal intubation. It is usually transient. The tube is withdrawn slightly and the operator should wait for the patient's first gasp; advancement of the tube at this precise moment is frequently successful, as the vocal cords are widely abducted during forced inhalation. Laryngeal anesthesia should also be assessed, and if IV and nebulized lidocaine have already been administered without success, transcricothyroid anesthesia (e.g., 2 mL of 4% lidocaine) should be considered. [53] Occasionally, a jaw lift is necessary to break prolonged spasm. Another option is to use a smaller tube.

**Placement Under Direct Vision**

This technique combines elements of oral and nasotracheal intubation. The indications and precautions are similar, and the importance of considering cervical spine injury is identical. Likewise, the need for jaw opening by physical or pharmacologic means is unchanged. This method is preferred to orotracheal intubation if the presence of an orotracheal tube might interfere with the repair of an oral injury. It is also useful when blind nasotracheal intubation has failed.

Preparation of the nose and nasopharynx and passage of the tube into the oropharynx are the same as described for blind nasotracheal intubation. It is with the introduction of the laryngoscope that the technique changes.

Laryngoscopy, as described with orotracheal intubation, is used to visualize the vocal cords and the tip of the endotracheal tube. With the Magill forceps in the right hand, the endotracheal tube is grasped proximal to the cuff (to avoid damage to the balloon) and directed toward the larynx (Fig. 2-16) . An assistant advances the tube gently while the operator directs the tip into the larynx and trachea. Cricoid pressure may facilitate the passage. Often the larynx can be manipulated sufficiently with the laryngoscope so that the physician can advance the tube with the right hand and guide it between the cords without using the Magill forceps. Occasionally, the natural curve of the tracheal tube guides it through the cords without any manipulation. The cuff is inflated, and both lungs are auscultated to ensure ventilation. When placement is satisfactory, the tube is secured.

**Complications**

Epistaxis is the most common complication of nasotracheal intubation. However *severe* epistaxis was encountered in only 5 of 300 cases reported by Danzl and Thomas. [42] Tintinalli and Claffey reported severe bleeding in 1 of 71 cases and less serious bleeding in 12 others. [54] Bleeding is usually not a problem unless it provokes vomiting or aspiration, a serious potential problem in obtunded patients with a clenched jaw or a decreased gag reflex. Other immediate complications include turbinate fracture, intracranial placement through basilar skull fracture, retropharyngeal laceration or dissection, and delayed or unsuccessful placement.  Unsuccessful placement may be minimized by selection of a smaller tube and by gentle technique.

Sinusitis in patients with nasotracheal tubes is common and can be an unrecognized cause of sepsis. [58] Rare but potentially fatal delayed complications include mediastinitis following retropharyngeal abscess [59] and massive pneumocephalus. [60]

Because most of the complications occur during tube advancement through the nasal passage and proximal nasopharynx, the complications of blind nasotracheal intubation and placement under direct vision are largely the same. However, retropharyngeal laceration and esophageal intubation are more of a threat in blind placement techniques because they are more likely to go unrecognized. [54] One unique problem associated

with nasotracheal intubation is damage of the tube cuff with the Magill forceps.

Delayed nasotracheal placement under direct vision deserves special discussion. Manipulation of the endotracheal tube through the nose and with the Magill forceps during the direct vision technique involves additional steps that require time. Because time is of the essence in the resuscitation of the critically ill patient, orotracheal intubation may be preferable.

**Summary**

Nasotracheal intubation is being used less frequently than in the past, because practitioners are increasingly comfortable using oral intubation in the patient with potential cervical spine injury. In addition, emergency physicians frequently use paralytics to facilitate orotracheal intubation. Nevertheless, nasotracheal intubation remains an effective and potentially life saving approach to the difficult airway and should be a dependable part of the armamentarium of all providers who are active in emergency airway management.

# GUIDED INTUBATION TECHNIQUES

## Digital Intubation

Digital intubation is a technique that uses the index and middle finger to blindly direct the endotracheal tube into the larynx. It is particularly well adapted to the out-of-hospital situation in which a trapped victim cannot be positioned for intubation. An out-of-hospital series of 66 digitally intubated patients demonstrated an 89% success rate. [61]

**Indications and Contraindications**

Digital intubation is indicated in the deeply comatose patient whose larynx cannot be visualized and who has a contraindication to nasotracheal intubation. Advantages include speed and ease of placement, immunity to anatomic constraints and other difficulties visualizing the larynx, and little neck movement. Contraindications are primarily precautions to protect the operator: digital intubation should not be attempted on any patient who presents a significant risk of biting. This includes the calm and awake patient as well as the agitated patient.

**Procedure**

The patient's head and neck are placed in neutral position. The operator stands at the patient's right side, facing the patient. The operator's left index and middle fingers are introduced into the right angle of the patient's mouth and are slid along the surface of the tongue until the epiglottis is palpated. The tip of epiglottis is palpated at 8 to 10 cm from the corner of the mouth in the average adult. The use of a stylet in the tube is optional; the largest reported series had good success without a stylet. [61] For the operator with short fingers or a patient with an anterior larynx, a stylet is advantageous. If a stylet is used, it is placed in the tube and bent into the form of an open "J" with the

distal end terminating in a gentle hook. A lubricated tube is introduced from the patient's left between the tongue and the rescuer's 2 fingers (Fig. 2-17) (Figure Not Available) . The tube is cradled between 2 fingers and the tip is guided beneath the epiglottis. Gentle anterior pressure directs the tube into the larynx. If the operator has sufficiently long fingers, they can be placed posterior to the arytenoids, acting as a "backstop" for the tube to both avoid esophageal passage and to assist in laryngeal placement. [62] If a stylet has been used, it is withdrawn at this time while simultaneously advancing the tube. An alternative to using a stylet for directing the tube anteriorly is to select an endotracheal tube with a controllable tip (Endotrol, Mallinckrodt Medical Inc, St Louis).

A variation on the technique of digital intubation has been described for intubating the newborn. [63] Only the index finger is used to guide the tube intraorally into the larynx. The end of the tube is bent and both the tube and the finger are moistened with sterile water. The index finger of the nondominant hand follows the tongue posteriorly to easily palpate the epiglottis and paired arytenoid. The thumb of the same hand may be used to apply cricoid pressure to steady the larynx. The endotracheal tube is held in the dominant hand and advanced using the nondominant index finger as a guide (Fig. 2-18) (Figure Not Available) . The tube snugs up (encounters subtle resistance) as it enters the trachea, and palpation of the tube through the trachea provides further confirmation of correct placement. A styletted tube, shaped in the form of a J, is usually desired until familiarity with the procedure is achieved.

### Complications

The risk of esophageal intubation is always present and, being a blind procedure in deeply comatose or cardiac arrested patients, the potential for esophageal misplacement is increased. If used in patients with a gag, induction of emesis with aspiration is a possibility. A high incidence of left main stem intubations was noted in a cadaveric study, [64] but clinical confirmation is lacking. The greatest risk seems to be to the operator, whose fingers may be bitten.

### Summary

While most of the recent experience with digital intubation in adults has been out of hospital, there is no reason why it should be confined to this setting. The majority of moribund emergency department patients who defy orotracheal intubation are never given a trial of digital intubation. This omission undoubtedly deprives some patients of expeditious airway management.

## Lighted Stylet Intubation

This technique uses a battery-operated lighted stylet that is placed in an endotracheal tube and used to guide the tube into the trachea by transilluminating the soft tissues of the neck. First described in 1957 by MacIntosh and Richards, [65] it was designed to aid in intubating the difficult airway. It has also been shown to be a useful means of determining the position of the tracheal tube. [31]

In the operating room, the Tube Stat lighted stylet (Concept Corp, Clearwater, Fla) has been 99 to 100% successful. [66] [67] The requirement that the overhead lights be dimmed during the procedure has limited its use in most emergency settings. In a small out-of-hospital study, 88% of patients were successfully intubated by physicians using a lighted stylet. [68] The majority of the failures occurred in the setting of bright sunlight and in patients who had vomited. A new device (Trachlight, Laerdal, Inc, Starger, Norway) with a brighter light source and adjustable length, appears to have solved this problem. In a series of 96 patients, many with a history of difficult intubation, all but 1 were successfully intubated in ambient light with this device using either the oral or nasotracheal route. [69] Consistent with other series, the only failure was in a morbidly obese patient.

**Indications and Contraindications**

The patient with a difficult airway in whom direct laryngoscopy has failed is a candidate for light-guided tracheal intubation. A multiple trauma patient with airway bleeding is a prime example. The patient who has been pharmacologically paralyzed and cannot be intubated with direct laryngoscopy is another example. The lighted stylet may also be helpful in successfully completing a difficult nasotracheal intubation. One advantage of this technique over nasotracheal intubation is that it can be used in the apneic patient.

Because lighted stylet intubation is a blind approach, it should be avoided in patients with expanding neck masses and patients with airway compromise presumed due to a foreign body. Massive obesity has been shown to be the most common cause for failure with this technique because of the impossibility of transilluminating through the generous soft tissue.

**Preparation**

The function of the bulb of the lighted stylet should be checked before use. The patient's head should be placed in a neutral or, if cervical spine injury is not a concern, the sniffing position. The awake patient should have the oropharynx and hypopharynx sprayed with lidocaine and sedation should be administered as indicated.

**Procedure**

The lubricated lighted stylet is inserted into a tracheal tube (5.5 mm or larger) until the bulb lies just distal to the side port, not protruding from the end of the tube. This unit is bent in the shape of a hockey stick that approximates a 90° curve beginning just proximal to the tube cuff. The operator stands at the head of the patient. When this is not possible, the patient can be approached from the right or the left side. The tongue is grasped with gauze and pulled forward. Another means of exposing the oropharynx is to grasp the jaw between the thumb and the fingers (Fig. 2-19) (Figure Not Available) . The light is turned on and the unit is inserted into the mouth, following the curve of the tongue into the oropharynx. A transilluminating glow indicates the location of the tube tip. Application of cricoid pressure may enhance transillumination. [31] The overhead light

should routinely be dimmed if feasible. Positioning is optimal when the glow emanates from the midline at the level of the hyoid bone. Holding the lighted stylet steady, the tube is slid off and advanced into the trachea. If the glow is located elsewhere, the unit should be withdrawn 2 cm or cocked back and repositioned as indicated by the light. If no light is seen, the tube is in the esophagus and should be pulled back, laryngeal pressure applied, and, if necessary, the head extended slightly. After passage, the tube should be checked for correct positioning and then secured.

**Complications**

Earlier reports noted complications resulting from an equipment failure and lost bulbs, but these problems have been corrected. No complications have been noted in the recent literature, but this may only reflect the limited use of this technique in uncontrolled settings.

**Summary**

Lighted stylet intubation is a safe, rapid, and highly successful method that has a definite place in the management of the difficult airway. Recent improvements in the device will increase its applicability to most settings in which emergency airway control is required.

## Intubation over a Fiberoptic Bronchoscope

The use of the flexible fiberoptic bronchoscope as an aid to tracheal intubation is a recent addition in airway management in the emergency department. In this setting, success approximates 80%, with the most common cause of failure being the inability to visualize the glottis secondary to blood and secretions. [70] [71]

Flexible fiberoptic endoscopy is the best method for intubating the awake patient with a difficult airway. It can be accomplished using the nasal or oral route and is better tolerated than direct laryngoscopy. It also may be effective in the comatose patient when more conventional methods have failed. It provides excellent visualization of the airway and permits the evaluation of the airway prior to tube placement. The greatest obstacle to success is the inability to see through the scope secondary to blood, secretions, or fogging. The expense of the equipment, its fragility, and the time required to achieve technical proficiency are three other drawbacks.

**Indications and Contraindications**

Common indications for emergency fiberoptic intubation include the unstable cervical spine, expanding neck masses, upper airway infection, facial and airway burns, and anticipation of a difficult intubation due to anatomic constraints. It may also be helpful in guiding blind nasotracheal intubation that is initially unsuccessful.

Contraindications to fiberoptic nasotracheal intubation are those ascribed to nasotracheal intubation in general: severe midface trauma and coagulopathy. Although

there are no clear contraindications to fiberoptic orotracheal intubation, active airway bleeding and vomiting are relative contraindications because successful fiberoptic intubation is rarely achieved in this setting. If the operator is inexperienced in fiberoptic intubation, apnea is another relative contraindication to its use.

**Equipment**

Fiberoptic scopes are graded according to their external diameter (in millimeters). A convenient intubating scope is 3.5 mm. Although it is physically possible to pass a 4.0 mm (0.5 mm larger) tracheal tube over the scope, the fit is quite tight. As a rule, the tracheal tube should be at least 1 mm larger than the intubating scope. The size of the working channel--the port to which suction or oxygen is applied and through which fluid or catheters may be passed--is another important dimension when evaluating fiberoptic scopes. Large working channels are desirable.

**Procedure**

The optimal positioning of the neck is in extension, as opposed to the cervical flexion desired when using direct laryngoscopy. Extension allows for better visualization of the

glottis by elevating the epiglottis off the posterior pharyngeal wall. This is especially pertinent in the comatose patient who lacks the muscle tone necessary to maintain an open airway. Problems with the tongue and soft tissues falling back and obscuring fiberoptic scope view are effectively managed by applying a jaw lift or pulling the tongue forward and away from the soft palate and posterior pharyngeal wall. This maneuver also moves the epiglottis away from the posterior pharyngeal wall facilitating exposure of the cords. Extending the head on the neck may accomplish the same objective.

**Nasotracheal approach.**

The nasal approach is preferred to the oral approach because the angle of insertion allows for easier visualization of the larynx and because patient cooperation is not as critical. Also, in the unconscious patient, the tip of the scope is less likely to impinge on the base of the tongue with a nasal approach.

The nose is prepared using a vasoconstrictor and topical anesthetic agent as described for nasotracheal intubation. Using an aerosolized anesthetic agent, it is important to obtain sufficient hypopharyngeal anesthesia to minimize gagging and laryngospasm once the procedure begins. The well-lubricated endotracheal tube may be placed in the nostril first, and the scope passed through it, or the endotracheal tube can be mounted over the scope and the scope first passed through the nostril. The advantage of the former is that it avoids the possibility of nasal secretions covering the scope and obscuring the view. The disadvantage is that nasotracheal placement may cause bleeding as well as that in some patients, the tube may not make the bend into the nasopharynx.

The most patent nostril is prepped and the endotracheal tube is advanced until it makes

the bend into the nasopharynx in the manner described under nasotracheal intubation. If negotiating this bend is difficult, a well-lubricated fiberoptic scope can be placed through the tube and into the oropharynx to serve as a guide for the endotracheal tube. Once the tracheal tube is in the oropharynx, thorough oropharyngeal suctioning should be performed prior to introduction of the scope into the endotracheal tube. The fiberoptic scope is then advanced toward the larynx; the epiglottis and vocal cords are seen with little or no manipulation of the tip of the fiberoptic scope in 90% of patients. [72] As the scope is advanced, the cords are kept in view by frequent minor adjustments of the scope tip.

In the comatose or obtunded patient, the tongue and other soft tissue may obscure the view of the larynx; this can be alleviated by having an assistant pull the tongue forward or apply a chin or jaw lift. The scope is advanced through the larynx to the level of the midtrachea and the endotracheal tube is passed over the firmly held fiberoptic scope into the trachea (Fig. 2-20) (Figure Not Available) . It is helpful to remember that in adults the average distance from the naris to the epiglottis is 16 to 17 cm; if the scope has been advanced much beyond this distance and the glottis is still not seen, the scope is probably in the esophagus. [73] If the scope meets resistance at about this same level and only a pink blur is visible, the scope tip is probably in a piriform sinus; transillumination of the soft tissues may be present to confirm this as well as to indicate what corrective maneuvers are necessary.

The greatest impediment to successful fiberoptic intubation is the inability to visualize the larynx because blood or secretions have covered the optical element and cannot be removed. The best time to suction is *before* introducing the fiberoptic scope, actively suctioning the oropharynx just prior to scope insertion. Once the scope is in place, minor secretions can be suctioned through the fiberoptic suction port. Significant blood and secretions, however, are best removed by insufflation of oxygen through the suction port and out the tip of the scope, serving simultaneously to remove blood and secretions, defog the tip, and increase the inspired $O_2$ content. The set-up required for insufflation should be immediately available, if not already attached to the suction port prior to scope insertion. Once the scope has entered the trachea, difficulty may be encountered in advancing the endotracheal tube into the trachea. The tip of the tube most commonly catches on the right arytenoid cartilage or vocal cord; withdrawing the tube 2 cm, rotating it counterclockwise 90°, and readvancing the tube should result in successful tracheal intubation (see Fig. 2-13) (Figure Not Available) .

**Orotracheal approach.**

Oral fiberoptically guided intubation is indicated when contraindications to nasal intubation are present, the most common being severe midface trauma, or when the operator is more comfortable with this approach. The oral approach is more difficult than the nasal approach because the path of the scope is less defined by the surrounding soft tissue and the tip of the scope is more likely to impinge on the base of the tongue or vallecula. Attention to keeping the scope in the midline and elevating the soft tissue by pulling the tongue forward or applying the jaw lift will minimize this difficulty. Another disadvantage of the oral approach is that the oropharyngeal axis is not as well aligned with the laryngeal axis as in the nasal approach and thus requires more scope

manipulation to visualize the larynx.

The drawbacks of the oral approach can be minimized by using an oral intubating airway. This adjunct resembles an oropharyngeal airway but is longer and has a cylindrical passage through which the fiberoptic scope and tracheal tube are passed. The tip of this airway lies just cephalad to the epiglottis and assures midline positioning and a predictable place from which to advance the scope (Fig. 2-21) (Figure Not Available) .

The patient must be adequately anesthetized or obtunded to minimize the gag reflex. Topical anesthesia is achieved by spraying a 4 or 10% solution of lidocaine into the oropharynx. A degree of laryngeal and tracheal anesthesia may be achieved by a transoral spray using the laryngeal tracheal anesthetic (LTA) set. A well-lubricated fiberoptic scope, premounted with an endotracheal tube, is placed through the oral intubating airway and the trachea is fiberoptically intubated. The endotracheal tube is advanced over the scope into the trachea, frequently requiring the same counterclockwise manipulation as described with the nasal approach. After successful intubation, the intubating device can be left in place as a bite block, or it can be removed over the endotracheal tube after removal of the tube adapter. Some oral intubating airways can be removed from the mouth without disconnecting the endotracheal tube adapter.

### Complications

Complications of fiberoptic orotracheal intubation include prolonged intubation attempts and vomiting and laryngospasm in the underanesthetized patient. Oxygen saturation monitoring should alert the operator to hypoxemia from prolonged intubation attempts. The majority of complications seen with fiberoptically guided nasotracheal intubation are associated with the passage of the endotracheal tube through the nasopharynx. Epistaxis is most common, followed by other nasopharyngeal injuries seen with nasotracheal intubation in general. A rare but potentially significant complication may result if on blind advancement of the fiberoptic scope through the endotracheal tube, the tip of the scope inadvertently exits out through the distal side port (Murphy's eyes) as it is being advanced through the larynx into the trachea. [74] Attempts at passing the endotracheal tube through the larynx will fail because the tube tip, now extending off the midline, will catch on the laryngeal structures. This complication is avoided if the scope is introduced prior to tracheal tube placement.

### Summary

The primary advantage of fiberoptic intubation is its ability to negotiate difficult airway anatomy. It is noninvasive and well tolerated. Its major limitation in the emergency setting is lack of visibility in the presence of blood and secretions. The fiberoptic scope requires more practice than other methods of airway management; the first experience using the scope should not be in the setting of an emergency airway problem. Once familiarity and facility with the scope are acquired, fiberoptic intubation can be used early in the management of the difficult airway rather than as a last resort after repeated

failed attempts using conventional techniques.

## Retrograde Intubation

Retrograde orotracheal intubation is a technique of guided endotracheal intubation using a wire or catheter placed percutaneously through the cricothyroid membrane or high trachea and exiting through the mouth or nose. An endotracheal tube is then passed over this guide and advanced through the vocal cords into the trachea. Introduced by Butler and Cirillo in 1960, [75] the technique has undergone several recent modifications that have enhanced its value as a means of establishing a definitive airway when more conventional techniques have failed.

### Indications and Contraindications

Retrograde intubation is indicated when definitive airway control is required and less invasive methods have failed. Indications include trismus, ankylosis of the jaw or cervical spine, upper airway masses, unstable cervical spine injuries, and maxillofacial trauma. It can be used to convert transtracheal needle ventilation (see Chapter 4) into a definitive airway. It has been described in a 1-month-old with developmental abnormalities. [76] It is particularly helpful in the trauma patient with airway bleeding that prevents visualization of the glottis. A striking example of the efficacy of this technique is presented in an article by Barriot and Riou describing successful out-of-hospital retrograde intubation in a series of trauma patients in whom attempts at conventional intubation failed. [77]

Contraindications to this procedure include the ability to control the airway by less invasive means. The inability to open the mouth is another contraindication. A relative contraindication is the apneic patient who cannot be effectively ventilated using the bag-valve-mask; in this setting it is advisable to first establish transtracheal needle ventilation (see Chapter 4) before attempting retrograde intubation or to go directly to cricothyrotomy.

### Equipment

Needed materials include the following: (1) local anesthetic and skin preparation materials, (2) 18-ga needle, (3) 60 cm epidural catheter-needle combination or 80 cm (0.88 mm diameter) spring guide wire (J-tip preferred), (4) hemostat, (5) long forceps (e.g., Magill) for grasping wire in pharynx, (6) endotracheal tube of appropriate size, (7) syringe for tube cuff, and (8) materials for securing tube. A prepackaged alternative is the Cook Retrograde Intubation Set (Cook Critical Care, Bloomington, Ind), which also contains a sheath.

### Procedure

Three anatomic landmarks must be located by palpation: the hyoid bone, thyroid cartilage, and cricoid cartilage. The skin overlying the cricothyroid membrane is prepped and anesthetized. Next, the lower half of the cricothyroid membrane is punctured with a

needle directed slightly cephalad. The bevel should also face cephalad. Air is aspirated to confirm needle tip position within the lumen of the larynx (see also Chapter 4) . An alternative entry point is the high trachea, usually through the subcricoid space, using the same steps as described for the cricothyroid membrane.

The syringe is removed and the wire is then passed through the needle and advanced until it is seen in the patient's mouth, with the help of the laryngoscope, or until it exits out the nose. If the wire is found in the hypopharynx, it is grasped with the Magill forceps and drawn out through the mouth. The needle is removed from the neck and the end of the wire is secured at the puncture site with a hemostat. The oral end of the wire is then threaded in through the endotracheal tube side port--not the end of the tube--and advanced up the tube until it can be grasped by a second hemostat. Threading the wire through the side port allows the tube tip to protrude 1 cm beyond the point at which the wire enters the larynx. The wire is then pulled taut and moved back and forth to ensure that no slack remains.

The endotracheal tube is then advanced over the wire until resistance is met. This is the most critical point in the procedure; because this is a blind technique, it may be difficult to determine whether the tube has entered the trachea or is hung up on more proximal structures. If the endotracheal tube has successfully passed through the vocal cords and it is being restricted by the guide wire as it traverses the anterior laryngeal wall, one should feel some caudally directed tension on the wire at its laryngeal insertion point. If this does not occur, the tip of the endotracheal tube may be proximal to the vocal cords, either in the vallecula, the piriform sinus, or abutting the narrow anterior aspect of the vocal cords. If in doubt, pull the tube back 2 cm, rotate it 90° counterclockwise, and readvance the tube. This will usually result in successful passage through the larynx. [73] When satisfied that the tube has entered the trachea, the tube should be stabilized and the guide wire pulled out through the mouth. The tube is then advanced further into the trachea.

The classic method of retrograde intubation, as described above, has undergone modifications that facilitate the passage of the endotracheal tube through the glottis. A significant advance has been the addition of a plastic sheath that is passed antegrade over the wire until it meets resistance at the point at which the wire penetrates the laryngeal mucosa (Fig. 2-22) (Figure Not Available) . [78] This sheath needs to be stiff enough to effectively guide an endotracheal tube, yet small enough to easily pass through the vocal cords without impinging on supraglottic or glottic structures. Once the sheath comes to rest against the anterior laryngeal wall, the wire is withdrawn from the mouth and the sheath is advanced. With the sheath well within the trachea, the endotracheal tube is passed over the sheath. Any resistance that may be encountered at the arytenoids or vocal cords can usually be remedied by pulling the tube back 1 to 2 cm and rotating it counterclockwise 90°. One advantage of the antegrade sheath is that it lies freely in the larynx, allowing for a more posterior passage through the widest distance between the cords. The wire, however, pulls the endotracheal tube anteriorly toward the narrow commissure of the vocal cords and is more likely to result in impingement of the tube on the cords. Also, the use of the sheath permits unrestricted advancement of the endotracheal tube, whereas a wire entering the larynx 1.0 to 1.5 cm below the vocal cords prevents the tube from advancing more than this distance prior to

removal of the wire.

If no sheath is available, one should consider placing the needle inferiorly in the subcricoid space, thereby increasing the distance the endotracheal tube can be advanced before being stopped by the wire. [79] This will decrease the likelihood of dislodging the endotracheal tube tip when the guide wire is withdrawn.

Up to this point, blind retrograde intubation has been described. A further modification of the technique allows for visualization using a fiberoptic scope. [80] In addition to the scope, an extra long guide wire (e.g., 125 cm, 0.025 cm Teflon-coated J-wire) is also required. The procedure is the same as previously described up to the point at which the wire is withdrawn from the mouth. At that point, with a endotracheal tube mounted on a lubricated fiberoptic scope, the long guide wire is passed retrograde up through the end of the fiberoptic scope and out the suction port. The fiberoptic scope is then advanced over the guide wire and through the cords, coming to rest against the anterior laryngeal wall (Fig. 2-23) (Figure Not Available) . The wire is withdrawn from the suction port and the scope is advanced into the trachea. The endotracheal tube is then passed over the fiberoptic scope, and visualization guarantees correct endotracheal placement. The scope is then withdrawn and the lungs are auscultated.

### Complications

The complications of retrograde intubation are largely related to cricothyroid membrane puncture (see Chapter 4) . Hemorrhage is minimized by taking care to puncture the cricothyroid membrane in its lower half (to avoid the cricothyroid artery). Subcutaneous emphysema may occur, but it is of no clinical significance because no air is insufflated during this technique. A small incidence of soft tissue infection is reported with translaryngeal needle procedures, but this can be minimized by ensuring that the wire is withdrawn from the mouth rather than the neck.

The final complication, the failure to achieve intubation, has been mitigated by the addition of the antegrade sheath over the wire.

### Summary

Retrograde intubation is an underused technique for achieving endotracheal intubation in a patient who cannot be intubated by less aggressive means. It is more invasive than fiberoptic intubation but requires less skill. Whereas retrograde intubation usually takes several minutes to complete, [81] the patient can undergo bag-mask ventilation through much of the procedure. Recent modifications in the technique guarantee this method a prominent place in the management of the difficult airway, particularly when active bleeding compromises the airway.

## CHANGING TRACHEAL TUBES

The tracheal tube with a leaking cuff is a vexing problem, especially if the original intubation was difficult. A method of replacing the tube without losing control of the

tracheal lumen is preferred. This can be achieved by passing a guide down the defective tube, withdrawing the tube while leaving the guide in place, and introducing a new tube over the guide and into the trachea.

A number of different guides have been described (e.g., simple nasogastric tubes, 18 Fr Salem sump tubes [Table 2-5] , feeding tubes), but they are poor substitutes for a designated tube exchanger such as the TTX "tracheal tube exchanger" (Sheridan Catheter Corporation, Argyle, NY) or a similar commercially available device. The advantages of the designated tube exchanger are that it is stiff enough to prevent dislodgment when the endotracheal tube is introduced, it is ready to use without modification, it has a printed scale to aid in determining depth of placement, and if replacement is prolonged, the patient may be oxygenated using the exchanger and wall oxygen.

**Procedure**

Prior to the procedure, the patient is properly sedated or restrained. The patient is hyperventilated before placing the guide through the existing tube. The guide is lubricated and advanced into the defective tube so that it is well within the tracheal lumen (adults, 30 cm). While applying cricoid pressure (Sellick maneuver), the defective tube is withdrawn over the guide, and care is taken not to dislodge the guide when removing the tube. The replacement tube is then slid over the guide and is gently advanced into the trachea (Fig. 2-24) (Figure Not Available) . At this juncture, it may be helpful to perform a jaw thrust or chin lift to facilitate passage through the pharynx. Resistance may be encountered at the laryngeal inlet or vocal cords; if this occurs, withdraw the tube 1 to 2 cm, rotate it 90° counterclockwise, and readvance it. With the tube clearly in the trachea, remove the guide, inflate the cuff, and ventilate the patient. After correct placement has been verified, the new tube can be secured.

Complications are related to the time required to change the tube. A successfully performed procedure can be accomplished within 30 seconds. Laryngeal injury from forcing the guide or the tube is a possibility to consider when replacing a tube

## CONCLUSION

Airway management in the critically ill or injured patient with acute airway compromise is a most demanding task for the emergency physician. Mastery of oral and nasotracheal intubation and a facility with various guided intubation techniques, plus a competence with the surgical approaches, is

---

**TABLE 2-5** -- Procedure for Changing an Orotracheal Tube Using a Salem Sump Nasogastric Tube as a Guide

---

| | |
|---|---|
| 1. | Cut off the proximal 6 to 8-cm flared end of an 18 Fr Salem sump nasogastric tube. |
| 2. | Test the balloon or cuff of the new tracheal tube. An 8.0-mm tracheal tube should be used. Remove the proximal adapter from the new tracheal tube. |
| 3. | Sedate the patient and restrain the hands as necessary. Preoxygenate as much as possible. |
| 4. | Lubricate the entire length of the nasogastric tube with water-soluble (K-Y, Lubrifax) lubricant. |
| 5. | Advance the nasogastric tube as far as possible into the trachea through the existing tracheal tube. |
| 6. | Deflate the balloon of the existing tracheal tube and remove it. *Now only the nasogastric tube remains in the trachea, to be used as a guiding stylet.* |
| 7. | Thread the proximal end of the nasogastric tube through the distal end of the new tracheal tube. This is facilitated by the use of a hemostat. |
| 8. | Advance the new tracheal tube over the guide until the nasogastric tube exits through the proximal opening in the new tracheal tube. |
| 9. | Grasp the exiting nasogastric tube, have an assistant *lift the patient's jaw*, and advance the tracheal tube over the guide into the trachea. If resistance is encountered, rotate the tube 90° counterclockwise. |
| 10. | Remove the guide. |

| 11. | Replace the adapter on the new tracheal tube, inflate the cuff, and ventilate the patient. Confirm proper placement of the new tube. |

necessary to meet all emergency situations that might occur. Preparation (including mastery of technique), advance preparation of equipment, and experience in clinical decision-making are essential. Scenario visualization is one means of practicing the difficult decisions that will afford the patient the most expeditious and safest means of airway control available under the circumstances.

# Chapter 3 - Pharmacologic Adjuncts to Intubation

**Steven C. Dronen**

Endotracheal intubation in the acute care setting presents a challenge distinct from that associated with intubation of the fasted, premedicated patient in the operating room. The emergency department (ED) patient is frequently uncooperative and unstable and may have medical problems that are completely unknown to the treating physician. Often within a matter of minutes, the physician is expected to assess and control the airway, as well as to diagnose and manage other life-threatening problems.

In 1979, Taryle and colleagues reported that complications occurred in 24 of 43 patients intubated in a university hospital ED. They called for improved house officer training in endotracheal intubation, as well as "more liberal use of the procedures used in the operating room, such as sedation and muscle relaxation." [1] Since the report of Taryle and others, training programs in both critical care and emergency medicine have been greatly expanded, resulting in significant improvement in the expertise of physicians who provide acute airway management. Simultaneously, the use of established pharmacologic adjuncts to intubation previously available only in the operating room has increased. In addition, new drugs have been developed that are potent, rapid acting, and relatively safe, giving the physician greater ability to tailor therapy to specific clinical problems. [9] [10] Because of these developments, physicians now may not only concentrate on the manual skill of intubation, but also skillfully use drugs to achieve specific objectives. These objectives may include (1) immediate airway control necessitating induction of anesthesia and muscle relaxation; (2) provision of analgesia and sedation to the awake patient; and (3) minimization of intubation adverse effects, including systemic and intracranial hypertension.

This chapter reviews the pharmacology and use of the drugs that are currently available to facilitate intubation in the acute care setting.

## RAPID-SEQUENCE INDUCTION OVERVIEW

In the critically ill patient who may be hypoxic, hemodynamically unstable, agitated, uncooperative, and at risk of further deterioration, it is frequently necessary to gain immediate control of the airway. In recent years the technique of rapidly inducing anesthesia (rapid-sequence induction [RSI]) as a means of airway control has gained broad acceptance among emergency physicians. RSI as practiced in the ED is a modification of the process initially described in the anesthesia literature and used to minimize the risk of aspiration in patients with full stomachs who require anesthesia. There is also a subtle but significant modification of the intent of RSI as practiced in the ED. Whereas anesthesiologists have used RSI to intubate patients requiring anesthesia, emergency physicians commonly use RSI to induce anesthesia in patients requiring intubation.

As described by Stept and Safar, [11] RSI includes 13 steps, several of which are not practical or relevant to the ED setting. In the ED, RSI is begun by placing the patient on

100% oxygen, for at least 2 to 3, and ideally 5, minutes. The intent is to denitrogenize the lungs and build an oxygen reserve that will last several minutes should intubation prove to be difficult. Under optimal conditions, breathing 100% oxygen for 3 minutes has been demonstrated to maintain acceptable oxygen saturation for 8 minutes. [12] The same study demonstrated that 4 maximal breaths of 100% oxygen from a face mask maintained acceptable saturation for 6 minutes. Comparable results should not be expected in the ED setting because of differences in the underlying health and cooperation of the patient population. Therefore, preoxygenation should be maintained for the longest period practical prior to beginning intubation.

During the period of preoxygenation, the airway should be assessed to determine the likelihood of a difficult intubation while establishing an IV line, placing the patient on cardiac and pulse oximetry monitors and assembling all necessary equipment for oral intubation and potentially cricothyrotomy. A defasciculating dose of a nondepolarizing neuromuscular blocking agent is commonly recommended 2 to 3 minutes prior to administration of succinylcholine, although the advantages of this time-consuming step are generally overstated. General anesthesia is then rapidly induced with one of the agents discussed below, followed immediately by muscle paralysis with succinylcholine at a dose of 1.5 to 2.0 mg/kg. Cricoid pressure is applied by an assistant as consciousness is lost. After the onset of adequate relaxation, orotracheal intubation is performed, and correct tube placement is verified. Cricoid pressure can then be released and ventilation with 100% oxygen begun. Mask ventilation prior to intubation is unnecessary if adequate preoxygenation occurs and in fact should be avoided because of potential gastric distention and passive regurgitation. Mask ventilation should be used only if adequate oxygenation cannot be ensured and then should be performed gently in association with cricoid pressure. In the event of failure to obtain orotracheal intubation, cricothyrotomy generally should be performed. In some cases, bag-mask ventilation with cricoid pressure may temporize until a repeat orotracheal attempt or alternative approach is made. The protocol for RSI is described in Table 3-1 .

The two general types of drugs used most commonly as part of RSI protocols are anesthetic agents and neuromuscular blocking agents. These are discussed in greater detail below.

## ANESTHETIC AGENTS

A number of diverse drugs are routinely used in the ED to induce anesthesia prior to intubation. These include thiopental, methohexital, ketamine, etomidate, and propofol. Midazolam and fentanyl may also be used; these two agents are more commonly used at low doses as conscious sedation agents (see Analgesia and Sedation). The choice of a particular anesthetic agent depends to a great extent on the experience and training of the physician and to a

**TABLE 3-1** -- Rapid-Sequence Induction Protocol

1. Preoxygenate (denitrogenize) the lungs by providing 100% oxygen by mask. If ventilatory assistance is necessary, bag gently while applying cricoid pressure.

2. Assemble required equipment:

Bag-valve-mask connected to an oxygen delivery system

Suction with Yankauer tip

Endotracheal tube with intact cuff, stylette, syringe, tape

Laryngoscope and blades, in working order

Cricothyrotomy tray

3. Check to be sure that a functioning, secure IV line is in place.

4. Continuously monitor the cardiac rhythm and oxygen saturation.

5. Premedicate as appropriate:

*Fentanyl:* 2 to 3 mug/kg given at a rate of 1 to 2 mug/kg/min IV for analgesia in awake patients

| |
|---|
| *Atropine:* 0.01 mg/kg IV push for children or adolescents (minimum dose of 0.1 mg recommended) |
| *Lidocaine:* 1.5 to 2.0 mg/kg IV over 30 to 60 seconds |
| 6. Induce anesthesia with one of the following agents administered intravenously: thiopental, methohexital, fentanyl, ketamine, etomidate, or propofol. |
| 7. Give succinylcholine 1.5 mg/kg IV push (use 2.0 mg/kg for infants and small children). |
| 8. Apnea, jaw relaxation, and decreased resistance to bag/mask ventilations indicate that the patient is sufficiently relaxed to proceed with intubation. |
| 9. Perform endotracheal intubation. If unable to intubate during the first 20-second attempt, stop and ventilate the patient with the bag-mask for 30 to 60 seconds. Follow pulse oxymetry readings as a guide. |
| 10. Treat bradycardia occurring during intubation with atropine 0.5 mg IV push (smaller dose for children; see item 5). |
| 11. Once intubation is completed, inflate the cuff and confirm endotracheal tube placement by auscultating for bilateral breath sounds and checking pulse oxymetry and capnography readings. |
| 12. Release cricoid pressure and secure endotracheal tube. |

certain extent on institutional protocols governing use of these agents. Drugs commonly used and their doses are listed in Table 3-2 .

**Barbiturates: Thiopental and Methohexital**

The barbiturates, in particular thiopental, have been the traditional agents used for RSI in the operating room. These agents are used less often in the ED setting because of their

| TABLE 3-2 -- Recommended Anesthetic Doses for Rapid-Sequence Induction | |
| --- | --- |
| **Drug** * | **Dose** |
| Thiopental | 3-5 mg/kg IV |
| Methohexital | 1-3 mg/kg IV |
| Fentanyl | 5-15 mug/kg IV |
| Ketamine | 1-2 mg/kg IV |
| Etomidate | 0.3 mg/kg IV |
| Propofol | 2.0 mg/kg IV |

reputation as cardiorespiratory depressants. Of these two agents, methohexital is used more commonly in the ED because of its extremely rapid onset and short duration of action.

**Pharmacology**

Following IV injection, the ultrashort-acting barbiturates bind rapidly to plasma proteins, particularly albumin. Unbound barbiturate quickly accumulates in highly vascular

organs, reaching peak brain levels in as short a time as 50 seconds. The drugs then diffuse from the brain, ultimately reaching equilibrium between the intracerebral and plasma concentrations. Degradation occurs primarily in the liver, producing inactive metabolites that are excreted in the urine or gut, depending on the drug used. Single-pass hepatic clearance is substantially higher for methohexital than for thiopental, which accounts for the former drug's shorter duration of action. The period of anesthesia following a single IV dose of methohexital is 4 to 6 minutes, compared with 5 to 10 minutes for thiopental. [13] [14]

The barbiturates are central nervous system (CNS) depressants that are capable of producing mild sedation to deep coma. They do not block afferent sensory impulses to a significant extent and therefore should be used in conjunction with an analgesic agent such as fentanyl if a painful procedure is to be performed. However, it is common practice to intubate patients who have received only barbiturates. [14]

The advantages of barbiturates as adjuncts to intubation include their high potency, rapid onset, and short duration of action, traits they share with fentanyl and midazolam. The barbiturates are also known to reduce cerebral metabolism and oxygen consumption and, secondarily, cerebral blood flow and intracranial pressure (ICP). [15] [16] For this reason, thiopental is considered the agent of choice for anesthesia induction and maintenance in patients with elevated ICP. Some have stated that thiopental is the drug of choice to temporarily anesthetize the patient with a head injury before intubation. It has not been proved, however, that barbiturates exert a protective effect on the CNS when used for a short period of time during RSI. Moreover, their use in trauma patients may lead to systemic hypotension and impaired cerebral perfusion pressure that may offset the theoretic advantages of barbiturate therapy.

**Dose**

The recommended dose of thiopental is 3 to 5 mg/kg IV administered as a 2.5% solution over 60 seconds. Normal saline should be used as a diluent. Methohexital is given at 1 to 3 mg/kg IV over 30 to 60 seconds.

**Adverse Effects**

It has been stated that barbiturates are "fatally easy" to use. [14] This is an overstatement that reflects improper use of the drugs more than an inherent danger associated with their proper use. The most significant complication of barbiturate therapy is depression of the vasomotor center and myocardial contractility leading to significant hypotension. This may be particularly pronounced in the presence of hypovolemia or cardiovascular disease.

Barbiturates also depress the brainstem respiratory centers when given rapidly or in large doses. This effect may be accelerated by simultaneous treatment with opioids. Patients

with asthma or chronic bronchitis may experience bronchospasm. Laryngospasm may

occur in patients who were anesthetized lightly with barbiturates during manipulation of the upper airway. Laryngospasm usually responds to positive-pressure ventilation or paralysis with succinylcholine. In addition, the high pH of the barbiturate solution can cause tissue necrosis following extravascular administration and severe pain, vessel spasm, and thrombosis following intraarterial infusion.

## Etomidate

Etomidate is an ultrashort-acting nonbarbiturate hypnotic agent that has been used successfully as an anesthesia induction agent in Europe since the mid-1970s and in the United States since 1983. Only recently has it been used as an adjunct to intubation in the ED. A potentially significant benefit of etomidate in the emergency setting is its lack of cardiodepressant effects. [17] Although experience in this setting is limited, etomidate's potency, rapid onset, short duration of action, and minimal side effects suggest that it will become much more widely used.

### Pharmacology

Etomidate is a carboxylated imidazole that is both water and lipid soluble. The drug rapidly accumulates in vascular organs, reaching peak brain concentrations within 1 minute of IV infusion. [18] Sleep is produced within 1 arm-brain circulation time and lasts less than 10 minutes following a single bolus infusion. [19] Redistribution of the drug is quite rapid (distribution half-life, 2.6 minutes), which accounts for the short duration of action. Etomidate is rapidly hydrolyzed in the liver and plasma, forming an inactive metabolite excreted primarily in the urine. [18]

Etomidate acts on the CNS to stimulate gamma-aminobutyric acid receptors and depress the reticular activating system. After IV infusion, etomidate produces electroencephalographic changes similar to those produced by barbiturates as patients pass rapidly through light to deep levels of surgical anesthesia. Because etomidate has no analgesic activity, [18] it should be used in conjunction with an analgesic such as fentanyl when painful conditions are being treated. Etomidate decreases cerebral oxygen consumption, cerebral blood flow, and ICP but appears to have minimal effects on cerebral perfusion pressure. [20]

### Dose

The recommended dose is 0.3 mg/kg IV. There is virtually no accumulation of the drug, and anesthesia may be maintained through repeated or continuous dosing. [21]

### Adverse Effects

The most common side effects of etomidate are nausea and vomiting, pain on injection, and myoclonic jerks. [22] Pain on injection occurs in up to two thirds of cases. Use of a large vein, simultaneous saline infusion, and opioid premedication are reported to reduce this side effect. [23] Myoclonic activity has been reported in about one third of cases and is believed to be caused by disinhibition of subcortical activity rather than

CNS stimulation. [18]

**Ketamine**

**Pharmacology**

Unique among anesthetic agents currently in use, ketamine produces a dissociative anesthesia characterized by excellent analgesia and amnesia despite the appearance of wakefulness. As a drug that is potent and relatively safe and possesses a rapid onset and brief duration of action, ketamine fits the profile of a drug that could be used effectively to facilitate intubation. It does, however, possess a number of pharmacologic properties that limit its use to selected circumstances.

Ketamine is a water- and lipid-soluble drug with rapid penetration into the CNS. Like the barbiturates, ketamine accumulates rapidly in highly vascular organs and then undergoes redistribution. The half-life of redistribution from plasma to peripheral tissues is 7 to 11 minutes, and the half-life of elimination is 2 to 3 hours. Degradation occurs primarily in the liver. [24]

Unlike other anesthetic agents that depress the reticular activating system, ketamine acts by interrupting association pathways between the thalamocortical and limbic systems. Characteristically, the eyes remain open, and patients exhibit spontaneous, although not purposeful, movements. Increases in blood pressure, heart rate, cardiac output, and myocardial oxygen consumption are seen--effects that are most likely mediated through the CNS. In vitro studies indicate that ketamine is a myocardial depressant, but the CNS-mediated pressor effects generally mask the direct cardiac effects. [25] [26] Respirations are initially rapid and shallow after ketamine administration, but they soon return to normal.

Other features of ketamine anesthesia include increased skeletal muscle tone, preservation of laryngeal and pharyngeal reflexes, hypersalivation, and relaxation of bronchial smooth muscle. ICP is increased, most likely as a consequence of increased cerebral blood flow. [24]

Ketamine has been recommended for anesthesia induction in children because of its relative safety and infrequency of postanesthesia emergence reactions in this group. There is no evidence, however, that it offers any advantage over commonly used agents. Ketamine also has been recommended for the unstable critically ill patient, because it does not depress the cardiovascular system. This recommendation is too vague to be useful to the clinician, and it ignores the fact that ketamine is potentially harmful in patients with cardiac ischemia (because it can increase myocardial oxygen consumption) or acute intracranial pathology (because it can increase ICP). Ketamine may be useful during hemorrhagic shock because of its cardiostimulatory effect. Its administration to patients in shock has been reported to cause a fall in blood pressure only when the shock state has been prolonged. [29] [30]

The most promising use of ketamine as an intubation adjunct has been in the setting of

acute bronchospastic disease. Ketamine relaxes bronchial smooth muscle either directly, through the enhancement of sympathomimetic effects, or through the inhibition of vagal effects. Ketamine also increases bronchial secretions, which may decrease the incidence of mucus plugging commonly reported in autopsies of asthmatic patients. [31] Clinical studies have demonstrated a reduction in airway resistance and an increase in pulmonary compliance that occur within minutes of ketamine administration. L'Hommedieu and Arens [8] reported

prompt improvement in respiratory acidosis in 5 asthmatics intubated with ketamine and succinylcholine.

**Dose**

The recommended dose of ketamine before intubation is 1 to 2 mg/kg administered IV over 1 minute. Anesthesia occurs within 1 minute of completing the infusion and lasts approximately 5 minutes. A small dose (0.5 to 1.0 mg/kg) may be given 5 minutes after the initial dose if there is a need to maintain anesthesia. The simultaneous administration of succinylcholine and midazolam is recommended to provide adequate muscle relaxation and to decrease the incidence of postanesthesia emergence reactions.

**Adverse Effects**

A side effect that has greatly limited the use of ketamine is its tendency to produce postanesthesia emergence reactions, a characteristic that it has in common with the structurally similar drug phencyclidine. The reactions may be marked and distressing to the patient; symptoms include floating sensations, dizziness, blurred vision, out-of-body experiences, and vivid dreams or nightmares. The reported incidence of these reactions varies from 5 to 30%. They are less common in children than in adults.

Of the drugs that have been evaluated for their ability to suppress postanesthesia emergence reactions, the benzodiazepines show the most promise. Both diazepam and lorazepam are useful, but the latter is more effective, most likely owing to its enhanced amnestic effect. Midazolam has not been evaluated as thoroughly as have the other benzodiazepines, but it has potent amnestic effects and offers the advantage of a short duration of action. White [40] reported a 55% incidence of postemergence dreaming in patients receiving ketamine and complete suppression of dreaming with the addition of midazolam. Evidence also suggests that midazolam may inhibit the cardiostimulatory effects of ketamine.

Although ketamine produces excellent analgesia and is relatively safe, its use as an agent to facilitate intubation is somewhat limited. The widely held belief that aspiration does not occur with ketamine because of preservation of pharyngeal and laryngeal reflexes is incorrect. Moreover, ketamine does not relax skeletal muscle. The production of desired intubation conditions often requires the simultaneous administration of a paralytic agent, thereby removing any upper airway reflexes.

**Propofol**

Emergency department experience with propofol is limited, and it is uncertain whether it will have a significant role as an adjunct to intubation in this setting.

**Pharmacology**

Propofol is an alkylphenol sedative-hypnotic recently introduced for induction and maintenance of general anesthesia. The drug has no analgesic activity, but it does have an amnestic effect. It produces dose-dependent depression of consciousness ranging from light sedation to coma. Propofol is a highly lipophilic, water-insoluble compound that undergoes rapid uptake by vascular tissues, including the brain, followed soon afterward by redistribution to the muscle and fat. The drug is metabolized by the liver and excreted in the urine.

**Dose**

Following an induction dose of 2 mg/kg IV, hypnosis occurs within 1 minute and lasts for 5 to 10 minutes. A smaller dose (1.0 to 1.5 mg/kg) is recommended in the elderly and when simultaneously administering other CNS depressants. Because propofol has a short duration of action and patients rapidly regain consciousness, repeat bolusing is not a practical way to maintain a desired level of anesthesia or sedation.  A slow drip infusion of 3 to 5 mg/kg/hour titrated to effect is the preferred technique. Conscious sedation may be achieved using a drip infusion beginning at 6 mg/kg/hour and decreasing the rate as the desired level of sedation is obtained.

**Adverse Effects**

Side effects of propofol include direct myocardial depression causing a moderate fall in blood pressure, particularly in the elderly, in hypovolemic patients, and when administered simultaneously with opioids. Propofol reduces cerebral blood flow and may cause mild CNS excitation activity (e.g., myoclonus, tremors, hiccups) during anesthesia induction. Pain on injection occurs commonly, even when the drug is infused slowly.

# NEUROMUSCULAR BLOCKING AGENTS

## Overview

Neuromuscular blocking agents (NMBs) are the standard agents used to achieve muscle relaxation for intubation, because they permit complete airway control and greatly simplify visualization of the vocal cords. This is particularly important when intubation must be performed quickly under less than ideal circumstances. NMBs are used routinely for RSI, as well as in conjunction with sedatives during "awake" intubation. Sedatives may be used to provide some muscle relaxation, but this requires that they be administered rapidly and in large doses, risking depression of the

cardiovascular system. The combination of a paralytic agent and a sedative or an analgesic agent is generally superior to the use of either agent alone.

The only absolute contraindication to the use of NMBs is the inability to manage the airway once the patient becomes apneic. Although not absolutely contraindicated, it is relatively inhumane to paralyze and intubate an alert patient. A sedative or an analgesic agent should always be administered simultaneously if the patient is able to perceive pain.

Controversy surrounds the use of NMBs to facilitate intubation in the patient who may have a cervical spine injury. Despite claims to the contrary, there is no evidence that orotracheal intubation performed during in-line stabilization, with or without paralysis, is dangerous. Cadaver studies purporting to demonstrate the dangers inherent in this practice simply do not simulate the condition of a living patient accurately. [51] Conversely, no studies prove conclusively that orotracheal intubation following the use of NMBs is a safe practice, but it has been performed for a number of years in operating rooms without reported detrimental effects, even in patients with unstable cervical spine fractures. [52] [53]

NMBs are classified as either depolarizing or nondepolarizing. Depolarizing agents mimic the action of acetylcholine

(ACh), producing a sustained depolarization of the neuromuscular junction during which muscle contraction cannot occur. Nondepolarizing agents competitively block the action of acetylcholine at the neuromuscular junction and prevent depolarization. NMBs in common use and their dosages, onset, and duration of action are listed in Table 3-3 .

## Depolarizing Agents

### Succinylcholine

**Pharmacology.**

The standard depolarizing agent currently in use is succinylcholine, which was introduced in 1952. It has a chemical structure similar to that of acetylcholine and is therefore able to depolarize the postjunctional neuromuscular membrane. Administration is followed by a brief period of muscle fasciculation that corresponds to the initial membrane depolarization. Unlike acetylcholine, which is released in minute amounts and hydrolyzed in milliseconds, succinylcholine requires several minutes for significant hydrolysis to occur. During this time, the neuromuscular membrane remains depolarized, but the muscles relax and will not contract until the neuromuscular end plate and adjacent sarcoplasmic reticulum return to the resting state and are again depolarized. Relaxation proceeds from the small, distal, rapidly moving muscles to the proximal, slowly moving muscles. The diaphragm is one of the last muscles to relax. [54]

Succinylcholine is rapidly hydrolyzed in the serum by the enzyme pseudocholinesterase. Only a small amount ever reaches the neuromuscular junction,

and that portion is quickly drawn back into the serum by a concentration gradient produced by serum clearance. The duration of action of a single dose is 3 to 5 minutes. Relaxation may be maintained by repeated IV injections or a constant infusion. Prolonged or repeated use of the drug, however, may enhance its effects at either the vagal or sympathetic ganglia. Vagal stimulation may result in bradycardia and hypotension, as well as other muscarinic effects. These effects may be seen even at normal doses, particularly in children. [55] For this reason atropine pretreatment is recommended in all children and in adults receiving multiple doses. [56] Prolonged neuromuscular blockade is uncommon but may occur in patients with reduced pseudocholinesterase levels.

Repeat dosing may also produce desensitization blockade in which the neuromuscular membrane returns to the resting state and becomes resistant to further depolarization and succinylcholine.  Clinically this is indicated by an unsustained contraction in response to a tetanic stimulus and response to a test dose of edrophonium. [59] In general,

| | TABLE 3-3 -- Commonly Used Neuromuscular Blocking Agents | | |
|---|---|---|---|
| **Agent** | **Dose (mg/kg)** | **Onset (min)** | **Duration (min)** |
| Succinylcholine | 1.5 | 1 | 3-5 |
| Pancuronium | 0.1 | 2-5 | 40-60 |
| Vecuronium | 0.1 | 3 | 30-35 |
| | 0.25 | 1 | 60-120 |
| Atracurium | 0.5 | 3 | 25-35 |
| Mivacurium | 0.15 | 2-3 | 15-20 |

| | | | |
|---|---|---|---|
| Rocuronium | 1.0 | 1-1.5 | 30-110 |

there is little need for repeated doses of succinylcholine. If paralysis in excess of 3 to 5 minutes is desired, agents such as vecuronium or pancuronium should be used.

**Dose.**

The recommended dose of succinylcholine is 1.0 to 1.5 mg/kg given IV. It is better to err on the side of too large a dose, thereby guaranteeing complete relaxation and avoiding the need for repeat dosing.

**Adverse Effects.**

There are a number of potential adverse effects of succinylcholine use. These include muscle fasciculations and their side effects, hyperkalemia, stimulation of autonomic ganglia, malignant hyperthermia, prolonged apnea, histamine release, and elevation of ICP.

As noted previously, muscle fasciculations accompany the initial depolarization of the neuromuscular membrane. They may be prevented by preadministration of a subparalytic dose (0.01 mg/kg) of pancuronium. Fasciculations are most prominent in muscular adolescents but are uncommon in children. Their most frequent side effect is deep, aching muscle pain that may last for several days. [60] Fasciculations of the abdominal wall may elevate intragastric pressure and cause regurgitation of stomach contents. This is an uncommon complication that most often follows overzealous bag-mask ventilation before intubation. Distention of the stomach with air and failure to perform the Sellick maneuver (i.e., firm cricoid pressure to occlude the esophagus during airway management procedures) are more likely to cause vomiting than are muscle fasciculations alone. It is also important to note that oropharyngeal manipulation of the nonparalyzed patient is far more likely to cause vomiting than are succinylcholine-induced fasciculations.

Other reported but distinctly uncommon side effects of muscle fasciculations include elevations of intraocular pressure and skeletal fractures or dislocations. The clinical significance of a transient rise in intraocular pressure in a patient with a penetrating eye injury is unknown. There has never been a reported case of vitreous expulsion occurring during rapid-sequence intubation with succinylcholine, despite its widespread use in open eye surgery. [61] However, paralysis will prevent spontaneous patient motor activity such as coughing or gagging, both of which are associated with a greater risk of vitreous expulsion. Although it may be prudent to use a nondepolarizing agent in patients with penetrating eye injuries, a necessary intubation using succinylcholine should never be delayed or avoided because of this theoretical concern. [62]

The precise mechanism by which succinylcholine causes hyperkalemia is unknown, but it is thought to occur secondary to the asynchronous depolarization of muscle cells and resultant cellular injury. The elevation is typically less than 0.5 mEq/L. [63] In certain pathologic states the hyperkalemic response may be as much has 5.0 mEq/L. These conditions include late severe burns, [64] major muscle trauma, [65] and upper motor neuron disease. These large elevations occur only in patients who have had significant tissue injury or muscle denervation for several days or weeks before succinylcholine use. Succinylcholine is not contraindicated in the acute initial management of these patients.

Malignant hyperthermia is a rare complication with an autosomal dominant inheritance pattern. It occurs in approximately 1 in 15,000 children and 1 in 50,000 adults. [68] The clinical syndrome consists of high fever, tachypnea, tachycardia, cardiac arrhythmias, hypoxia, acidosis, myoglobinuria, and impaired coagulation. Muscle spasm rather than

relaxation is frequently seen. [69] Treatment includes aggressive cooling measures (see Chapter 72) , volume replacement, and correction of hypoxia and acid-base and electrolyte abnormalities. Dantrolene sodium, a direct-acting skeletal muscle relaxant, has been shown to be effective in reducing the muscle hypermetabolism that causes the fever. [70]

An associated abnormal response to succinylcholine is isolated masseter spasm. [71] Barlow and Isaacs reported two cases in which masseter spasm was the first abnormality noted in fatal episodes of malignant hyperthermia. [72] Masseter spasm may be more common in patients with neuromuscular disorders such as myotonia congenita.

Prolonged apnea may occur because of decreased pseudocholinesterase levels (e.g., in hepatic disease, anemia, renal failure, pregnancy, advanced age, bronchogenic carcinoma, or connective tissue disorders) or, more commonly, because of the inheritance of an atypical pseudocholinesterase present in about 0.03% of the population. This atypical enzyme has both a decreased affinity for the succinylcholine molecule and a decreased ability to hydrolyze it. The period of apnea is therefore increased from 3 to 5 minutes to up to several hours. Patients with markedly decreased levels of normal cholinesterase experience only a two- to three-fold increase in the duration of apnea. [73] Patients with cocaine intoxication may experience prolonged muscle relaxation if given succinylcholine, because cocaine is competitively metabolized by the cholinesterases.

The magnitude and significance of the increase in ICP that occurs with succinylcholine use remain controversial. [74] [75] [76] [77] [78] Increases in the range of 5 to 10 mm Hg have been reported by several investigators, but other researchers have shown no increase, nor is there evidence of neurologic deterioration associated with these transient elevations in ICP. Mechanisms that have been proposed to explain the elevated ICP include (1) a direct effect of fasciculations, (2) an increase in cortical electrical activity with a resultant increase in cerebral blood flow and blood volume, and (3) sympathetic postganglionic stimulation. Minton and colleagues have demonstrated that pretreatment with vecuronium (0.14 mg/kg) reduces the rise in ICP following succinylcholine

administration from mean values of 5 mm Hg to 1 mm Hg. [79] It has been postulated that nondepolarizing blockade prevents muscle spindle firing and the increase in cortical activity that may lead to increased ICP. Pretreatment with a nondepolarizing agent may not be practical when intubation must be performed rapidly; furthermore, the dose that has been shown to be effective is itself a paralyzing dose and would obviate the need for succinylcholine.

At present, questions concerning the safety of succinylcholine in the setting of acute intracranial pathology do not have clear answers. The drug has been used widely and successfully in this setting, and its continued use is supported. The eminent risk of airway compromise without the use of a depolarizing agent must always be weighed against the rare harmful effects mentioned.

## Nondepolarizing Agents

Nondepolarizing agents act in a competitive manner to block the effects of acetylcholine at the neuromuscular junction. Drugs in this class include pancuronium, atracurium, vecuronium, mivacurium, and *d*-tubocurarine. These drugs, particularly the intermediate-acting agents vecuronium and atracurium, have fewer side effects than succinylcholine and have the potential for reversal. However, they generally have longer onset and duration of action than succinylcholine, making them less attractive choices for rapid intubation. *In most instances, succinylcholine remains the agent of choice to facilitate emergency intubation,* and nondepolarizing agents are indicated to maintain paralysis after intubation. Of note, a new nondepolarizing agent, rocuronium, has an onset of action within 1 to 1.5 minutes, which may permit its use for rapid-sequence intubation when succinylcholine is contraindicated.

Because nondepolarizing agents act competitively, their effects may be reversed by increasing the concentration of acetylcholine. Cholinesterase inhibitors such as neostigmine or edrophonium may be used, but not until some spontaneous signs of reversal are seen. Thus, the concept of reversal is of limited clinical importance. When reversal is required, neostigmine 0.02 to 0.04 mg/kg is given by slow IV push. Additional doses of 0.01 to 0.02 mg/kg may be given in 5 minutes if reversal is incomplete, but the total dose should not exceed 5 mg in the adult. Atropine 0.01 mg/kg (with a *minimum* dose of 0.1 mg for adults) should be given concurrently with neostigmine to block its cholinergic effects. [80] [81] [82]

Of the nondepolarizing agents, pancuronium has been used the most widely, but its comparatively slow onset of action, long half-life, and greater incidence of side effects make it less than ideal for ED use. The recently introduced agents vecuronium and atracurium are preferable in most instances.

### Pancuronium

Pancuronium is an aminosteroid derivative that is primarily excreted in the urine within 1 hour of IV administration. [82] Classified as a long-acting agent, its onset and duration of

action are dose related.

Following the recommended 0.1 mg/kg IV dose, paralysis occurs within 2 to 5 minutes and lasts approximately 60 minutes. Paralysis may be maintained safely by repeated bolus or drip infusion. Because the effects of the drug are cumulative, repeating the original dose significantly lengthens the duration of paralysis.

**Adverse Effects.**

Relatively few adverse effects are associated with the use of pancuronium. Many patients experience an increase in heart rate, blood pressure, and cardiac output because of the vagolytic effect of the drug. Ventricular tachycardia and severe hypertension have been reported but are quite rare. Pancuronium may cause histamine release that results in bronchospasm or anaphylactic reactions. [85] Prolonged paralysis may also occur, primarily in patients with myasthenia gravis or with significant impairment of renal function.

**Vecuronium, Atracurium, Mivacurium, and Rocuronium**

Vecuronium and atracurium are intermediate-acting agents with an onset of action of about 3 minutes and a duration of action of 30 minutes. Mivacurium has an onset of action of 2 to 3 minutes and a duration of action of 15 to 20 minutes. These drugs represent significant advances over pancuronium in that they have minimal cardiovascular effects, cause little histamine release, and lack cumulative effects. [86]

**Dose.**

The recommended doses of vecuronium, atracurium,

mivacurium, and rocuronium are listed in Table 3-3 . Use of a larger than recommended dose will hasten the onset of action and greatly prolong the period of paralysis. For example, vecuronium at a dose of 0.25 mg/kg IV will cause paralysis in as little as 1 minute, but the period of paralysis will last 1 to 3 hours.  Because a rapid onset of action comparable to succinylcholine is achieved at high doses of intermediate-acting agents, they may be used as the sole agents to facilitate intubation, particularly if a long period of paralysis is desired after intubation. Similarly, rocuronium produces paralysis sufficiently rapidly to be used for rapid-sequence intubation. The duration of paralysis seems related to the dosing: a mean of 45 minutes is achieved with 0.6 mg/kg, and a mean of 85 minutes with 1.2 mg/kg. [89] However, the use of succinylcholine prior to intubation and an intermediate-acting agent at a normal dose after intubation provides rapid intubating conditions and excellent control over the duration of paralysis.

Paralysis induced by vecuronium or atracurium may be maintained by repeat bolus or drip infusion. Unlike both pancuronium and succinylcholine, there are no side effects specifically related to repeated dosing in the emergency department. A repeated dose of

0.01 to 0.02 mg/kg of vecuronium will extend the period of paralysis 12 to 15 minutes.

# ANALGESIA AND SEDATION

## Overview

An alternative to the induction of anesthesia in patients requiring intubation is the use of anesthetic and sedative agents in the conscious patient. Laryngoscopy in the awake patient has been likened to the "mouth being held open with a wrench." [90] The upper airway is richly innervated by sensory branches of the fifth, seventh, ninth, and tenth cranial nerves. In addition to pain fibers, there are stretch receptors that stimulate coughing and gagging reflexes with even minor airway manipulation. It is therefore essential that adequate analgesia be provided before intubation in all but the most critical circumstances. Treatment options include topical application of anesthetic agents to the pharyngeal and tracheal mucosa and IV infusion of analgesic or sedative agents.

## Local Anesthesia

Local or topical anesthesia techniques may be used in patients who are awake, either in place of or as a supplement to IV analgesia or sedation. They are particularly useful as adjuncts to nasotracheal intubation but do not generally provide the degree of analgesia or relaxation desirable for orotracheal intubation. In addition, it is time consuming to achieve good topical anesthesia, which may limit the usefulness of these techniques in emergency situations.

Topical anesthesia may be achieved by direct application using laryngoscopy, by cricothyroid membrane puncture, or by inhalation of a nebulized anesthetic.

### Direct Application Using Laryngoscopy

Achieving anesthesia of the oral and pharyngeal mucosa is a relatively simple procedure using commonly available agents such as 4% lidocaine or benzocaine-tetracaine-butamben-benzalkonium (Cetacaine). Achieving anesthesia of the hypopharynx is more difficult, because optimal results require application of the anesthetic to the epiglottis and vocal cords under direct vision.

This procedure is begun by spraying the tongue and pharynx with a topical agent. After allowing at least 2 to 3 minutes to permit numbing of the tongue and pharynx, the epiglottis and vocal cords are visualized using a laryngoscope and are sprayed directly with the anesthetic agent. Even if laryngoscopy is not used to facilitate this procedure, it is at best an unpleasant experience. It is also time consuming because of the inherent delay associated with mucosal absorption of an anesthetic agent. An alternative is percutaneous injection of an anesthetic agent into the trachea at the level of the cricothyroid membrane. [91] [92]

**Cricothyroid Membrane Puncture**

The cricothyroid membrane is identified in the trapezoidal space between the cricoid and thyroid cartilages. After appropriate skin preparation with an alcohol or povidone-iodine (Betadine) swab, the overlying tissue and membrane are punctured with a 22-ga needle in the midline and just above the superior border of the cricoid cartilage. Care should be taken to maintain the needle in the midline at all times to avoid injury to the recurrent laryngeal nerves. The needle should be advanced until air can be aspirated, indicating placement of the tip in the trachea. A volume of 2 mL of 4% lidocaine is then injected rapidly. Alternatively, 3 to 4 mL of 1% or 2% lidocaine, as used for local anesthesia, may be injected if the 4% concentration is not available. Typically this will precipitate a cough, which adequately distributes the anesthetic over the upper trachea, vocal cords, and epiglottis (see Chapters 4 and 5) .

**Nebulized Anesthesia**

This simple and painless technique can be used to facilitate awake intubation when the patient's condition is stable enough to permit a several-minute delay. [93] The anesthetic is delivered using a standard nebulizer and face mask connected to an oxygen source that delivers 4 to 8 L/min. A volume of 4 mL of a 4% solution is nebulized over about 5 minutes. Bourke and colleagues reported achieving consistently good topical anesthesia using this technique, although their patients were often premedicated with opioids, sedatives, or both. [94]

## Intravenous Analgesia and Sedation

The IV infusion of analgesic or sedative agents either alone or in combination is an excellent means of facilitating relatively painless intubation. In recent years a number of drugs have been released that are characterized by high potency, rapid onset of action, short half-life, and minimal potential for cardiorespiratory depression. Depending on the clinical situation encountered, the doses of these drugs may be titrated to produce alterations of consciousness ranging from very light sedation to complete loss of consciousness. To facilitate orotracheal intubation, these drugs are frequently used in conjunction with succinylcholine, thus producing a state of complete muscle relaxation, good analgesia, reduced anxiety, and loss of recall for the event.

The most commonly used classes of drugs for sedation prior to intubation are opioids and benzodiazepines. Anesthetic

agents previously discussed, in particular the barbiturates, may also be used as sedatives, although in lower doses than required for RSI.

**Opioids (Fentanyl)**

Although any of several opioids administered IV could be used to induce conscious sedation, only fentanyl will be discussed because it possesses significant advantages

over other opioid agents.

**Pharmacology.**

Fentanyl is a synthetic opioid related to the phenylpiperidine family. Since its introduction in 1968, fentanyl has been used widely in a variety of settings, often replacing meperidine as the agent of choice for rapid short-term analgesia.  Its favorable pharmacologic properties include a highly lipophilic nature, rapid serum clearance, high potency, and minimal histamine release.  Fentanyl crosses the blood-brain barrier rapidly, producing analgesia in as little as 1½ minutes. Serum levels decline rapidly from peak concentrations because of extensive tissue uptake. [ Unlike with morphine, the brain concentration of the drug falls in conjunction with the serum level. The duration of analgesic action is 30 to 40 minutes, although at high doses a second peak of activity may be seen several hours later because of the release of the bound drug from tissue stores. Fentanyl is about 50 to 100 times as potent as morphine sulfate. [106] This unique combination of potency and short half-life permits the administration of numerous small doses that can be titrated to the desired clinical effect. Similarly to other opioids, fentanyl is competitively reversed with naloxone or nalmefene.

**Dose.**

The relative safety of fentanyl permits considerable latitude in dosing. When used as a primary anesthetic agent for major surgical procedures, doses ranging from 50 to 100 mug/kg produce minimal side effects. [107] Comparatively tiny doses produce sedation, and 3 to 5 mug/kg, given at a rate of 1 to 2 mug/kg/min, is generally an effective analgesic dose. More rapid administration will cause greater depression of the level of consciousness. Mostert and coworkers reported successful awake intubation in 99 of 103 patients who were administered an average dose of 3.7 mug/kg. [100] Most of these patients were able to follow commands, and many recalled the events surrounding the intubation. A small percentage could not be intubated even after receiving 500 mug of fentanyl.

Larger doses, perhaps up to 25 mug/kg, may be needed to produce ideal intubating conditions, although if given rapidly, 10 mug/kg is usually adequate. However, even this lower dose is more likely to produce anesthesia than sedation and it may cause a longer period of unresponsiveness than is desirable. It is preferable to use a low dose of fentanyl (2 to 3 mug/kg) for analgesia combined with a paralytic agent (e.g., succinylcholine) to produce adequate muscle relaxation and a sedative (e.g., midazolam) to reduce anxiety and produce amnesia for the event.

**Adverse Effects.**

Unlike other opioids, fentanyl causes little or no histamine release, and its use is seldom associated with emesis or hypotension. It is probably the safest opioid to use in the hypovolemic patient. Fentanyl also has significantly less emetic effects than other opioids. Adverse effects that have been reported with fentanyl are few and primarily follow the rapid IV infusion of very large doses. Like other opioids, fentanyl may cause

rigidity of the skeletal musculature, including the chest wall and diaphragm. Typically this occurs with doses in excess of 15 mug/kg, but it has also been reported with doses as low as 10 mug/kg. [100] [108] The muscular rigidity may be prevented or treated with standard doses of succinylcholine or naloxone. [109] Grand mal seizures have also been reported but are very uncommon. Chudnofsky and colleagues [7] reported a complication rate of less than 1% in 841 emergency department patients treated with fentanyl. The most common complication was respiratory depression, and it generally occurred when fentanyl was given in combination with other CNS depressants.

**Benzodiazepines (Midazolam)**

**Pharmacology.**

The benzodiazepines are a widely used class of drugs characterized by anxiolytic, hypnotic, sedative, anticonvulsant, muscle relaxant, and amnestic effects. Several of these properties make the benzodiazepines ideal adjuvant agents for intubation, particularly when used in combination with opioids. It is important to remember that benzodiazepines do not have analgesic effects. Although they may produce excellent sedation and impair the patient's memory of an unpleasant experience, they will not prevent the pain associated with intubation. Diazepam has been used widely to facilitate intubation, but its use has been supplanted in recent years. Diazepam has a variable onset of action and a long elimination half-life, and it causes significant infusion site pain and frequently phlebitis when given intravenously.

Midazolam has, to a great extent, replaced diazepam as a preoperative sedative agent. Midazolam is also used widely as an anesthesia induction agent, even in high-risk elderly and cardiac patients. ] Compared with diazepam, the primary advantages of midazolam include a two-fold increase in potency, a shorter half-life, and a lessened potential for cardiorespiratory depression. Midazolam possesses a unique imidazole ring that is stable and water soluble in an acid medium but highly lipophilic at physiologic pH.

Because it does not require suspension in propylene glycol, midazolam is not a tissue irritant. It causes minimal pain on injection, is rarely associated with phlebitis, and can be given intramuscularly when a very rapid onset of action is not required. The highly lipophilic character of the drug permits rapid accumulation in the CNS with onset of sedation in as little as 1 to 2 minutes. Rapid penetration into fatty tissue coupled with extensive binding to plasma proteins causes a prompt fall in serum levels after IV administration. This may account for the paucity of side effects outside the CNS. The half-life of elimination is 1 to 4 hours and is dependent on release of the drug from adipose tissue and protein-binding sites. The period of sedation following a single IV dose is considerably shorter. Emergence from a 0.15 mg/kg dose occurs in 15 to 20 minutes. [118]

Clinical experience using midazolam for conscious sedation before the performance of surgical and dental procedures or as an adjunctive agent for the induction of anesthesia is considerable. Baker and Gordon reported the use of midazolam to achieve conscious sedation in 400 ambulatory surgery patients. [114] Used in combination with either

fentanyl or ketamine, midazolam was considered to be both effective and safe.

**Dose.**

The recommended dose for conscious sedation with midazolam is 0.05 to 0.1 mg/kg given in 1-mg boluses and not exceeding 2.5 mg over 2 minutes. Doses up to 0.1 mg/kg are often needed to produce good conditions for intubation. All patients receiving midazolam should be monitored

closely, and skilled personnel prepared to manage the airway should be present.

**Adverse Effects.**

Although midazolam was initially touted to be free of cardiorespiratory side effects, recent experience suggests that the potential adverse effects of midazolam are quite similar to those of other benzodiazepines. A small increase in heart rate is seen frequently, as is a small decrease in systolic blood pressure. [119] Changes in blood pressure may be exaggerated in the presence of hypovolemia. [120] Cardiac index and coronary artery blood flow are generally not affected. Respiratory depression may occur even at standard doses but most often follows rapid administration of an excessive dose. Respiratory depression is also more likely to occur in debilitated or elderly patients and in those simultaneously receiving opioids. Reports of fatalities soon after midazolam's introduction in the United States prompted changes in the recommendations for its use. These included lowering of the dosage schedule, reduction of the speed of administration, and careful patient monitoring during administration. [121] The effects of midazolam are rapidly reversed by the administration of the benzodiazepine antagonist flumazenil.

Limited information is available on the use of midazolam to facilitate ED intubation. Wright and colleagues reported its use in 289 ED patients, 20 of whom were undergoing intubation. [122] The overall complication rate was 1.4%, including 2 cases of hypotension and 2 cases of respiratory depression. In every case, patients had received other drugs in combination with midazolam that may have been responsible for the observed adverse effects. Most patients in their study (71%) also received fentanyl. The midazolam-fentanyl combination has been reported to provide an excellent level of conscious sedation that has rapid onset and is of short duration.

# PREVENTING THE COMPLICATIONS OF INTUBATION

## The Pressor Response

The pressor response to stimulation of the pharynx, larynx, and trachea was first described by King and coworkers in 1951. [125] This reflex, mediated by the sympathetic nervous system, consists of a transient increase in blood pressure and pulse rate. The

stretching of the hypopharynx that occurs with laryngoscopy is the most common precipitant of the pressor response, but any manipulation of the upper airway, including nasotracheal intubation or suction, may elicit a potent response. An increase in plasma catecholamines, including adrenaline and noradrenaline, is found in association with the pressor response.

Considerable variation exists in the magnitude and duration of the pressor response. Studies of young healthy normotensive subjects have shown that an average increase of 20 to 25 mm Hg in mean arterial pressure occurs with laryngoscopy and intubation. The magnitude of the response increases as the duration of the stimulus increases, reaching a peak at 45 seconds. Data from controls in several studies of patients with a broad spectrum of medical problems reveal blood pressure increases ranging from 14 to 48 mm Hg, with an average of about 30 mm Hg. Similarly, the increase in heart rate ranges from 8 to 45 beats/min, with an average of approximately 30 beats/min. Typically these elevations last less than 5 minutes. The magnitude of the pressor response may be increased in hypertensive patients and those with cardiovascular disease, even if the underlying hypertension is adequately controlled before intubation.

In addition to sinus tachycardia, a number of dysrhythmias have been reported following intubation. These are primarily ventricular in origin and include ectopic beats, bigeminy, and, occasionally, short runs of ventricular tachycardia. Bradyarrhythmias have been reported uncommonly. Electrocardiographic changes suggestive of ischemia have been reported, particularly in patients with dramatic increases in blood pressure. [136] [137] In 1977, Fox and colleagues reported two patients, [138] both of whom deteriorated after induction of anesthesia and orotracheal intubation. This report has been widely quoted as evidence that the pressor response should be prevented. However, no studies have reported comparative data and none have established a direct relationship between the response and subsequent clinical deterioration in a large patient population. It is also unclear that attenuation of the pressor response will prevent dysrhythmias or electrocardiographic evidence of ischemia, although it is prudent to avoid sudden increases in blood pressure in unstable patients with acute cardiac or atherosclerotic vascular disease.

Multiple studies have evaluated procedures to pharmacologically block the pressor response. Lidocaine has been the most extensively evaluated, but the results of these studies are inconclusive.  Although it appears that lidocaine given at 1.5 to 2.0 mg/kg may blunt the response, it is not clear that the reductions reported (10 to 15 mm Hg and 20 beats/min) are of any clinical significance. Other drugs, including thiopentone, sodium nitroprusside, labetalol, nitroglycerin, verapamil, nifedipine, clonidine, fentanyl, sufentanil, etomidate, and magnesium have shown variable responses.  Of these drugs fentanyl may be the most effective. It completely suppresses the pressor response at anesthetic doses of 50 mug/kg,  but considerably smaller doses may also be effective. Two studies  have shown marked suppression of the response at doses of 5 to 6 mug/kg, although in both studies patients also received 5 mg/kg of thiopental. Fentanyl has also been shown to blunt the pressor response when administered in conjunction with etomidate.

It is important to note that even those studies demonstrating blunting of the pressor

response failed to demonstrate that this provided any real benefit to the patients. It is likely that the pressor response is innocuous in the vast majority of patients, but it may be exaggerated and potentially harmful in the presence of preexisting hypertension and cardiovascular disease. [138] In addition, the pressor response may contribute to the rise in ICP that follows laryngoscopy, and therefore it is potentially harmful in patients with intracranial pathology. Administration of lidocaine or fentanyl to blunt the pressor response is appropriate in these subsets of patients.

**Intracranial Hypertension**

Physical stimulation of the respiratory tract by maneuvers such as laryngoscopy, tracheal intubation, and endotracheal suctioning is commonly associated with a brief rise in ICP. The exact mechanism responsible for this rise in ICP is

unknown. One potential mechanism is the coughing and gagging that frequently follows manipulation of the upper airway and subsequent transmission of intrathoracic pressure to the cerebral circulation. An alternative explanation is the catecholamine release that accompanies laryngoscopy, causing a rise in mean arterial pressure and cerebral perfusion pressure. A small rise in ICP has also been reported following administration of succinylcholine.

Although the exact significance of a transient rise in ICP is unknown, it is logical to assume that it may be detrimental in patients with head trauma or intracranial hypertension. A number of drugs, including lidocaine, succinylcholine, and the majority of the anesthesia induction agents, have been studied to determine whether their use prevents this response. Many of the existing clinical data are not particularly relevant to the emergency department setting because they are derived from patients in various stages of general anesthesia, often incorporating a wide variety of drug combinations and doses.

Lidocaine may be an effective agent either because it prevents coughing associated with airway manipulation or because it blunts the pressor response and the resultant rise in cerebral perfusion pressure. At a dose of 1.5 mg/kg IV, lidocaine suppresses coughing induced by citric acid inhalation [156] and at 2.0 mg/kg IV, it suppresses coughing associated with intubation. [157] However, studies have demonstrated conflicting results regarding lidocaine's ability to suppress the rise in ICP that follows airway manipulation. Optimal results have been demonstrated in paralyzed patients, suggesting that paralysis may be the best method to suppress coughing and resultant ICP elevations during intubation. [160] Thus, the use of lidocaine to prevent coughing may have a role only when paralysis is not an option. In the paralyzed patient, lidocaine's effectiveness is uncertain, but it may be beneficial in blunting the pressor response and, secondarily, ICP elevations.

There is good evidence that deep general anesthesia prevents the rise in ICP associated with intubation, although depending on the drug used, anesthesia may compromise cardiovascular performance and critically reduce cerebral blood flow. Consequently, the ideal anesthetic agents to facilitate intubation of patients with acute intracranial pathology may be those that have minimal effects on cardiovascular

performance such as etomidate or fentanyl. Etomidate has been demonstrated to prevent changes in both cerebral perfusion pressure and ICP following tracheal intubation of patients with space-occupying intracranial lesions. [20]

At the present time, the clinical consequences of intubation-induced physiologic changes are not thoroughly understood, nor is the role of drugs in preventing these changes clear. Despite this lack of data, it may be prudent to attempt to protect patients at theoretic risk. The approach outlined in Table 3-4 is recommended.

## CONCLUSION

This chapter has described a number of pharmacologic adjuncts that permit a much more sophisticated approach to intubation of the critically ill patient. As the expertise of physicians practicing in the acute care setting increases, it is appropriate that they incorporate these adjuncts into their airway management protocols. The agents discussed in this

| **TABLE 3-4** -- Sample Protocol for Intubation of a Head-Injured Patient |
| --- |
| 1. Preoxygenate with 100% O$_2$ for 2-3 minutes. |
| 2. Administer 1.5-2 mg/kg lidocaine. |
| 3. Administer 0.01 mg/kg vecuronium (OPTIONAL). |
| 4. Sedate with 3-5 mug/kg fentanyl. |
| 5. Induce anesthesia with 0.3 mg/kg etomidate. |
| 6. Paralyze with 1.5 mg/kg of succinylcholine. |
| 7. Apply cricoid pressure and perform intubation. |

| |
|---|
| 8. Maintain postintubation analgesia and sedation. |
| 9. Maintain paralysis if indicated (vecuronium 0.1 mg/kg). |

chapter are representative of drugs currently used to facilitate intubation in U.S. EDs.

# Chapter 4 - Cricothyrotomy and Translaryngeal Jet Ventilation

**Sharon E. Mace**

Although intubation is the usual method of definitive airway control, clinical situations occur in which intubation is difficult, impossible, or contraindicated. Under these circumstances, cricothyrotomy may be the quickest, easiest, safest, and most effective way to obtain an airway.

Cricothyrotomy is the procedure by which an opening is made in the cricothyroid membrane (Fig. 4-1) to establish an airway. Cricothyrotomy is also known as a laryngostomy, laryngotomy, cricothyrostomy, or coniotomy. *Surgical* cricothyrotomy is the use of a blade to create the opening in the cricothyroid membrane. Once access to the airway is created, oxygen is administered through the opening in the cricothyroid membrane. A needle or small cannula also can be passed percutaneously (i.e., *needle* cricothyrotomy) through the cricothyroid membrane to permit *translaryngeal jet ventilation*. Cricothyrotomy procedures differ from *tracheostomy,* in which entry into the airway is at a lower point (i.e., between the tracheal rings).

An emergency cricothyrotomy allows oxygenation and prevents hypoxemia and its complications, including anoxic encephalopathy and death. Management of the airway is of paramount importance, because the physician has only 3 to 5 minutes in which to obtain an airway and achieve effective ventilation to prevent the complications of hypoxemia. [2]

## BACKGROUND

The importance of securing an airway has been recognized since ancient times. Egyptian tablets dating from 3600 B.C. depict a surgical tracheostomy. [3] Historical records give credit for the first tracheostomy to Asclepiades of Prusa in 124 B.C.[4] In the 2nd century A.D., Galen suggested tracheostomy as the treatment for emergency management of airway obstruction [5] and Antyllus performed a tracheostomy with an incision between the third and fourth tracheal rings. [6]

In the United States, tracheostomies were a known procedure even in colonial times. In 1796, tracheostomies were advocated as a possible treatment for drowning victims. [7] Perhaps the first recorded "successful" case of a surgical cricothyrotomy was in 1852. The cricothyrotomy was successful, but the patient died later from airway stenosis, a complication of the procedure. [8] In 1886, the tracheostomy mortality rate was about 50%, with a high complication rate from airway stenosis, which was usually fatal.

Although tracheostomies and cricothyrotomies were successful and life saving, they were generally done only on severely hypoxic, terminally ill patients, thus contributing to the high morbidity and mortality of the surgical procedure. The fear of complications with the surgical procedure led to a reluctance of the physician to actively intervene until the

patient was near death. "Certainly the delay due to this great dread of tracheostomy was itself largely accountable for the fatality attending its performance."

In 1909, Jackson described the surgical technique for tracheostomy and the factors that are critical for successful tracheostomy or cricothyrotomy. [11] These factors, which are still important today, include (1) obtaining the best airway control possible before surgery; (2) using local anesthesia initially rather than sedation or general anesthesia, which can lead to respiratory arrest in patients with an already compromised airway (although sedation and general anesthesia may be acceptable if the patient is already intubated); (3) emphasizing careful, precise surgical technique with good exposure; (4) using an inert, appropriately sized and shaped tracheostomy tube; and (5) ensuring meticulous postoperative care, which helps avoid contamination or infection and other complications. He noted a surgical mortality of approximately 3%, which is similar to that reported in current series.

Jackson achieved worldwide recognition and patient referrals regarding cricothyrotomy complications. In 1921, Jackson published an investigation of 200 patients referred to him with tracheal stenosis. [12] The study was biased in that patients who did well and had no surgical complications (e.g., no laryngeal stenosis) were not referred to Jackson. Many of the patients had stenosis because of their underlying medical condition and not because of the procedure. Factors relating to surgical technique and postoperative care were not controlled, including aseptic technique, location of the opening in the larynx, and size and type of the tracheostomy tube. Jackson condemned cricothyrotomy because of the complication of subglottic stenosis.

Years later, Brantigan and Grow reported the results of cricothyrotomy on 655 patients. [13] Only 8 patients (0.01%) developed airway stenosis, and none of the patients developed chronic subglottic stenosis. Major vessel hemorrhage and operative misadventures were absent, and the complication rate, which included minor problems, was only 6.1%. The literature supports their conclusions that cricothyrotomy is a safe and effective procedure for airway management with relatively few complications. [When examined at bronchoscopy, no signs of laryngeal stenosis or damage have been noted in patients with cricothyrotomy. [17] Animal studies have documented a normal larynx after cricothyrotomy. [18]

## ANATOMY

Knowledge of the anatomy of the neck and the upper airway is essential when performing a cricothyrotomy. The anatomic landmarks include the hyoid bone, the thyroid cartilage, the cricoid cartilage, and the tracheal rings (see Fig. 4-1) .

The hyoid bone is located midway between the mental protuberance of the mandible and the third cervical vertebra. The midpoint of the body of the hyoid is also the transverse midpoint of the neck and can function as an anchor for stabilizing the airway in an edematous neck. The thyroid cartilage and the cricoid cartilage, both part of the larynx, and the tracheal rings are the major supporting structures of the airway. The trachea is the downward continuation of the larynx. The trachea is supported by cartilaginous rings that are deficient posteriorly. Posteriorly, the trachea lies against the

esophagus.

The thyroid cartilage consists of two approximately quadrilateral-shaped laminae of hyaline cartilage that fuse anteriorly to form the laryngeal prominence. Above the laryngeal

prominence is the superior thyroid notch, where the two laminae are separated. The anterior superior edge of the thyroid cartilage, the laryngeal prominence, is known as the Adam's apple and is usually easily seen in men. Except in the infant, the markedly obese patient, or the patient with massive neck edema, the laryngeal prominence of the thyroid cartilage is usually easily recognized and is palpable. It is probably the most important landmark in the neck when performing a cricothyrotomy.

The cricoid cartilage is the only circumferential ring in the larynx. It is shaped like a signet ring with the shield located posteriorly. The cricoid cartilage forms the inferior border of the cricothyroid membrane. The thyroid cartilage forms the superior border of the cricothyroid membrane.

The highly vascular thyroid gland lies over the trachea at the level of the second and third tracheal rings. If the tracheal rings or the thyroid gland is encountered when performing a cricothyrotomy, then the clinician is too low in the neck and must redirect the incision superiorly toward the cricoid and thyroid cartilages. Avoid the thyroid gland, because marked bleeding may occur if it is injured.

The cricothyroid membrane is a dense fibroelastic membrane located between the thyroid cartilage superiorly and the cricoid cartilage inferiorly; it is bounded laterally by the cricothyroideus muscles. The cricothyroid membrane covers an area that is trapezoidal in shape. The average size of the cricothyroid membrane in the adult is approximately 22 to 30 mm wide and 9 to 10 mm high. The cricothyroid membrane can be identified by palpating a notch, a slight indentation or dip in the skin, inferior to the laryngeal prominence. The cricothyroid membrane is located approximately 2 to 3 cm below the laryngeal prominence in an adult.

Several anatomic and physiologic features make the cricothyroid membrane an excellent choice for gaining access to the airway. The membrane is immediately subcutaneous in location. It does not calcify with age. It has no overlying muscles or fascial layers and no major arteries, veins, or nerves are in the region. Although the right and left cricothyroid arteries, branches of the right and left superior thyroid arteries, respectively, transverse the superior part of the cricothyroid membrane (i.e., nearer the thyroid than the cricoid cartilage) and anastomose in the midline, these vessels generally have not been found to be of clinical significance when performing a cricothyrotomy.

**Anatomic Variations**

The larynx has a few anatomic features that vary according to age and sex. In children, the larynx is much higher than it is in the adult. The larynx descends from approximately

the level of the second cervical vertebra at birth to the level of the fifth or sixth cervical vertebra in the adult.

The laryngeal prominence, the angle at which these two laminae of the thyroid cartilage meet anteriorly, varies from 90 to 120°. This angle is smaller in the male, which makes the laryngeal prominence meet at a more acute angle, or a sharper, more prominent point, than in the female.

In infants, the prominent structures in the anterior neck are the hyoid bone and the cricoid cartilage. The laryngeal prominence does not develop until adolescence and young adulthood.

A few vascular anomalies in the neck may result in a major artery crossing the midline of the neck. This is usually not a problem, because these vascular anomalies are almost always located lower in the neck.

# SURGICAL CRICOTHYROTOMY

### Indications

There are several indications for cricothyrotomy (Table 4-1) . The most common indication is inability to perform endotracheal intubation because intubation is either contraindicated or cannot be done easily and quickly. This decision is always best made by the clinician at the bedside using clinical judgment to weigh the individual circumstances.

Clinical conditions in which endotracheal intubation is difficult at best and is often impossible may include significant bleeding of any of the structures in the upper airway (e.g., massive oral, nasal, or pharyngeal hemorrhage), massive emesis or regurgitation, masseter spasm, clenched teeth, spasm of the larynx or pharynx (or both), laryngeal stenosis, and structural deformities of the upper airway (e.g., congenital or acquired abnormalities or deformities of the oropharynx). Whenever several unsuccessful attempts at endotracheal intubation cause an inordinate delay in airway control and oxygenation, cricothyrotomy is indicated to prevent cerebral anoxic damage. [19]

Obstruction of the upper airway is another indication for cricothyrotomy. [20] It has been stated that patients who are completely obstructed or in extremis are best managed by

**TABLE 4-1** -- Indications for Surgical Cricothyrotomy

Failure of oral or nasal endotracheal intubation
   Massive oral, nasal, or pharyngeal hemorrhage
   Massive regurgitation or emesis
   Masseter spasm
   Clenched teeth
   Structural deformities of oropharynx, congenital or acquired Stenosis of upper airway (pharynx or larynx)
   Laryngospasm
   Mass effect (cancer, tumor, polyp, web, or other mass)
Airway obstruction (partial or complete)
   Nontraumatic
      Oropharyngeal edema
      Laryngospasm
      Mass effect (cancer, tumor, polyp, web, or other mass)
   Traumatic
      Oropharyngeal edema
      Foreign body obstruction
      Laryngospasm
      Obstruction secondary to a mass effect or displacement
   Stenosis
Traumatic injuries making oral or nasal endotracheal intubation
   difficult or potentially hazardous (relative)
   Maxillofacial injuries
   Cervical spine instability
Need for prolonged intubation
Need for definitive airway during procedures on face, neck, or upper airway
   Laryngeal surgery
   Oral surgery
   Maxillofacial surgery
   Laser surgery
   Bronchoscopy

establishing an airway via the cricothyroid membrane. [21] When simple airway procedures are unsuccessful, cricothyrotomy should be undertaken without delay.

When maxillofacial, cervical spine, head, or soft tissue neck injuries are present, several factors may prevent intubation. These factors include (1) gross distortion of structures, (2) airway obstruction, (3) disruption of upper airway structures, (4) massive emesis, (5) significant hemorrhage, (6) patient discomfort, and (7) the possibility of aggravating existing or suspected injuries that would result in additional damage. In such patients, cricothyrotomy is an excellent alternative for obtaining definitive control of the airway. [22]

Cricothyrotomy should be considered (1) for patients with a known cervical spine injury or a high probability of cervical spine injury in whom definitive airway control is needed

before the cervical spine can be assessed and (2) for patients with certain head injuries, especially basilar skull fractures or cribriform plate fractures. In trauma patients with known or anticipated cervical spine injury, movement of the neck while positioning the patient for intubation is to be minimized because of the possibility of causing additional cervical spine injury. [23] This recommendation has more recently been questioned. [24] *In-line stabilization of the neck with minimal movement during direct laryngoscopy appears to be a relatively safe alternative during emergent intubation of the trauma patient.* [25] [26]

Cricothyrotomy has several advantages over tracheostomy when prolonged intubation is needed (Table 4-2) . [27] [28] Cricothyrotomy has been used for definitive airway control before elective surgery of the face, head, and neck. [29] Translaryngeal jet ventilation has become a routine method of ventilation during procedures (both endoscopy and surgery) on the upper airway. [30] This ventilation technique allows better access to and visualization of the upper airway for the surgeon or endoscopist than is possible with intubation from above the larynx, which often obscures and limits the surgical field. It also decreases the chance of flames or a "mini-explosion,"

---

**TABLE 4-2** -- Advantages of Surgical Cricothyrotomy Over Tracheostomy

Advantages due to anatomic considerations
   Immediate subcutaneous location (vs deep dissection)
   Absence of critical structures overlying cricothyroid membrane
   Easily seen landmarks, recognizable from the surface anatomy
   Less chance of esophageal injury (circumferential cricoid cartilage vs deficient tracheal cartilage posteriorly)
   Further away from mediastinum, dome of pleura (less encroachment on thoracic structures)
Overall advantages
   Easier to do, faster, safer
   Does not need to be done in an operating room
   Less need for hyperextension of the neck
   Better cosmetic appearance of scar (shorter, less adherent)
   Decreased incidence of late complications (e.g., swallowing problems, voice disturbances, fistulas, erosion of innominate artery)
   Decreased incidence of early complications (e.g., pneumothorax, pneumomediastinum, mediastinal perforation, esophageal injury) because of less encroachment on the mediastinum and other critical structures
   Can be done quickly by non-surgeons
   Requires a minimum of instruments

| TABLE 4-3 -- Contraindications to Surgical Cricothyrotomy |
| --- |
| Absolute contraindications<br>   Endotracheal intubation can be accomplished easily and quickly, and no contraindications to endotracheal intubation are present<br>   Transection of trachea with retraction of distal end into the mediastinum<br>   Fractured larynx or significant damage to the cricoid cartilage or larynx<br>Relative contraindications *<br>   For infants and toddlers (<5 yr) transtracheal ventilation may be preferred over surgical cricothyrotomy<br>   Bleeding diathesis<br>   Patients with massive neck edema (may use modified technique for these patients)<br>   Acute laryngeal disease |

Relative contraindications may be overlooked in the true emergency situation because it is more important to obtain an airway and to avoid hypoxemia.

which may occur during laser surgery or during other procedures that require flammable anesthetics or other gases. [31]

## Cricothyrotomy vs Tracheostomy

The decreased incidence of complications with cricothyrotomy compared with that of tracheostomy is due at least partly to anatomy. [32] Anatomic considerations make tracheostomy a relatively complicated and difficult procedure. Many delicate complex structures in the neck lie in close proximity to the trachea. Less encroachment on the mediastinum occurs with a cricothyrotomy than with a tracheostomy, because the cricothyroid membrane is further away from the mediastinum and other critical structures. Early complications, including pneumothorax, pneumomediastinum, and mediastinal perforation, occur less often with cricothyrotomy. Because the tracheal cartilage, unlike the cricoid and thyroid cartilage, is deficient posteriorly, the chance of damage to the posterior structures (e.g., esophagus) lying immediately behind the airway is greater with a tracheostomy. The incidence of late complications, including fistulas, erosion of the innominate vessels, swallowing problems, and voice disturbances, is less with cricothyrotomy.

Cricothyrotomy is preferred over tracheostomy for definitive emergency management of the airway when intubation is impossible or contraindicated. The few exceptions are in children 5 years old (although some recommend needle cricothyrotomy with translaryngeal jet ventilation rather than tracheostomy) and for transection of the trachea with retraction of the distal trachea into the mediastinum.

## Contraindications

Cricothyrotomy has relatively few contraindications (Table 4-3) . Cricothyrotomy should not be done in patients who can be quickly, easily, and safely intubated. Transection of the trachea with retraction of the distal end is a contraindication to cricothyrotomy. A fractured larynx or other significant damage to the larynx or cricoid cartilage is another contraindication. [33]

| **TABLE 4-4** -- Equipment for Surgical Cricothyrotomy Tray |
| --- |
| Scalpel with No. 15 blade and scalpel with No. 11 blade<br>Tracheal dilator (Trousseau dilator) or spreader<br>Two hemostats<br>Scissors<br>Tracheal hook<br>Needle holder<br>Tracheostomy tube (appropriate-size Portex or Shiley tube--No. 5-6 in an adult)<br>25-ga needle and syringe containing lidocaine with epinephrine (for local anesthesia)<br>Preparation solution<br>Sterile gauze pads<br>Sterile tracheal suction catheter<br>Suture or circumferential tie (to secure tracheostomy tube in place) |

Factors shown to be associated with subglottic stenosis include prolonged intubation, underlying laryngeal disease, and younger age (e.g., infants and small children). [34] Some believe that these conditions are relative contraindications to cricothyrotomy and that a cricothyrotomy should be converted immediately to a surgical tracheostomy. [35] Current studies indicate that subglottic stenosis is not a common complication of cricothyrotomy, even in the presence of laryngeal pathology, and that patients can have a cricothyrotomy for months without having subglottic stenosis. These considerations (i.e., prolonged intubation and underlying laryngeal disease) are no longer absolute contraindications to cricothyrotomy.

A bleeding diathesis is not an absolute contraindication to emergency cricothyrotomy, because it is easier to obtain hemostasis with a cricothyrotomy than with a tracheostomy. Loss of cervical landmarks with massive neck edema makes the procedure more difficult, although one approach using a measured estimation of the cricothyroid membrane location in this setting is promising. [36] These relative

contraindications may be overlooked in a true emergency, i.e., when obtaining an airway is imperative.

## Equipment

In the out-of-hospital setting, the only essential items are a sharp blade and a hollow tube to maintain the airway. In the emergency department, the optimal instruments needed for surgical cricothyrotomy should be easily accessible. A sterile tray containing all the necessary instruments in an organized fashion should be part of the standard equipment stocked in the major resuscitation area (Table 4-4) . Prepackaged percutaneous kits are also available.

An appropriate-sized tracheostomy tube should be selected (Table 4-5) . A small tube becomes obstructed more easily by secretions, whereas a large tube may damage the surrounding cartilage and lead to a fractured larynx. In an adult, a tracheostomy tube with an 8-mm internal diameter (ID), such as a No. 5 Portex or Shiley tube, is usually an appropriate size (see Table 4-5) .

If an appropriately sized tracheostomy tube is not available immediately, an endotracheal tube can be used as is or modified for use as a tracheostomy tube (Fig 4-2) . The uncuffed (proximal) end of the endotracheal tube is cut to an appropriate length (see Fig. 4-2 ) *A*). The adapter is then attached to the cut end (see Fig. 4-2 *B*), and the modified endotracheal tube is inserted like any tracheostomy tube. After insertion, the cuff of the endotracheal tube should be inflated, if possible.

The essential supplemental equipment for airway management, specifically supplemental oxygen, suction, and a bag-valve device, should be readily available.

## Procedure

### Preparation and Positioning

In a truly emergent situation, rapid airway access is critical, and there may not be enough time even for local anesthesia. Because of the dangers of respiratory depression in a patient who may have a compromised airway already, sedation and general anesthesia may be contraindicated.

The patient is positioned to expose the neck and its landmarks. If there are no contraindications, such as known

---

**TABLE 4-5** -- Tracheostomy Tube Sizes

| Age | Internal Diameter of Tracheostomy Tube (mm) | Holinger or Magill Tube Size | Internal Diameter of Endotracheal Tube (mm) |
|---|---|---|---|
| Premature (<1.8 kg; <4 lb) | | 000 | 2.5 |
| Premature (1.8 kg; 4 lb) | | 00 | 2.5 |
| Newborn | 2.5 | 00 or 0 | 3.0 |
| 0-6 mo | 3.5 | 0 | 3.5 |
| 6-12 mo | 4.0-4.5 | 1 | 4.0 |
| 1 yr | 4.5-5.0 | 1 or 2 | 4.5 |
| 2 yr | 5.0 | 2 | 5.0 |
| 4 yr | 5.5 | 3 | 5.5 |
| 6 yr | 6.0 | 4 | 6.0 |
| 8 yr | 6.5 | 4 | 6.5 |

| | | | |
|---|---|---|---|
| 10 yr | 7.0 | 4 | 7.0 |
| 12 yr | 7.5 | 5 | 7.5 |
| 14 yr | 8.0 | 5 | 7.5 |
| Adult female | 8.0-8.5 | 5 | 7.5-8.5 |
| Adult male | 8.5-9.5 | 6 | 8.0-9.0 |

**Figure 4-2** Modification of an endotracheal tube for use as a tracheostomy tube. *A,* An appropriate-sized endotracheal tube is cut to an appropriate length. *B,* An adapter is attached to the cut end before the modified endotracheal tube is inserted into the larynx via the opening in the cricothyroid membrane.

or suspected cervical spine injury, the patient's head should be hyperextended.

The cricothyroid membrane is located by identifying the dip or notch in the neck below the laryngeal prominence. The cricothyroid membrane is bounded by the thyroid cartilage superiorly and the cricoid cartilage inferiorly. The landmarks are most easily found by placing the index finger on the prominence of the thyroid cartilage and slowly palpating downward until the finger "drops off" the thyroid cartilage and onto the cricoid membrane (Fig. 4-3 *A*). When the tip of the finger is in the proper position, the hard cricoid cartilage may be palpated with the fat pad of the index finger. If time permits, local anesthesia and skin preparation with povidone-iodine may be used. The skin and subcutaneous tissue immediately above the cricothyroid membrane may be infiltrated using lidocaine with epinephrine.

**Incision and Tube Placement**

A skin incision is made over the cricoid membrane. A midline longitudinal (vertical) skin incision about 3 to 4 cm long is made for an emergency cricothyrotomy. For an elective cricothyrotomy, a more cosmetic 2-cm transverse (horizontal) skin incision can be made. A longitudinal skin incision is preferred during an emergency, because if the skin incision is too high or too low, a longitudinal skin incision merely needs to be extended, thus saving time and avoiding a second incision. As an option, one can insert a 20- to 25-ga needle through the cricothyroid membrane and confirm intra-airway positioning by aspirating air. The needle can be left in place to serve as a guide for the surgical

procedure and then removed before insertion of the tracheostomy tube.

The larynx is stabilized by holding it between the nondominant thumb and middle finger or by using a tracheal hook. After the skin incision, a short horizontal (transverse) stabbing incision about 1 cm long is made in the lower part of the cricothyroid membrane (i.e., nearer the cricoid cartilage than the thyroid cartilage to avoid the cricothyroid arteries). The stabbing incision is made in such a way that only the tip of the scalpel blade enters the trachea (see Fig. 4-3) .

Curved Mayo scissors are inserted beside the scalpel blade and then spread horizontally to widen the space. An alternative method is to widen the opening by using a scalpel handle and turning the scalpel 90° ("longitudinally" with respect to the patient) instead of using curved Mayo scissors. The scalpel handle then is removed and a tracheal (Trousseau) dilator or curved hemostat is inserted into the incision site in the cricothyroid membrane. The dilator is then opened to enlarge the opening in the cricothyroid membrane.

When attempting to insert the tracheostomy tube, the larynx may be displaced posteriorly. Posterior displacement of the larynx may make it difficult or impossible to insert the tracheostomy tube. Stabilization of the larynx is important to lift and hold the larynx anteriorly in proper anatomic position so that this potential problem can be avoided. The tracheostomy tube is inserted between the tracheal dilator or curved hemostat blades (see Fig. 4-3 *E*). The dilator or hemostat is then removed. The cuff of the tracheostomy tube is inflated.

In an adult, a No. 5 or 6 Shiley tracheostomy tube is usually an appropriate size (No. 5 for an adult female and No. 6 for an adult male). If edema is present, a smaller (No. 4) Shiley tube may be used. Other tube size guidelines are provided in Table 4-5 .

If a tracheostomy tube is not available immediately, then an endotracheal tube can be used as a temporary airway device. The endotracheal tube can be replaced later when an appropriate-sized tracheostomy tube is available.

The lungs are ventilated, and proper tube placement should be ensured. The tracheostomy tube is secured in place via a circumferential tie around the neck or by suturing. After confirming proper tube placement, the tracheostomy tube may be attached to a ventilator or a bag-valve device. Flexible connector tubing is recommended to avoid excessive forces on the tracheal wall during mechanical ventilation.

**Precautions**

The skin incision and the incision in the cricothyroid membrane should not be made too far laterally (see Figs. 4-1 and 4-3 *A*). A central incision will decrease the chance of hemorrhage due to laceration of vessels. The skin incision should not be made too far caudally in the neck, thus avoiding hemorrhage from and damage to the thyroid gland.

The stabbing incision should be made in the lower half of the cricothyroid membrane (i.e., nearer the cricoid cartilage than the thyroid cartilage) in an attempt to avoid bleeding from the cricothyroid arteries, which course superiorly across the cricothyroid membrane near the lower edge of the thyroid cartilage.

Transection of the cricoid cartilage or tracheal rings should be avoided. These structures help maintain the stability of the laryngeal lumen. Injury to these structures may predispose to late complications, including subglottic stenosis.

The scalpel should be directed in a caudal direction, so that injury to the vocal cords is avoided. The vocal cords are located above the cricothyroid membrane and are protected by the thyroid cartilage on three sides. When a cricothyrotomy is being done, the needle or scalpel should be directed posteriorly at a 90° angle to the cricothyroid membrane (see Fig. 4-3) .

The physician should avoid making a blind stab with the scalpel in the region. Controlled, anatomically placed incisions will minimize hemorrhage and injury to any of the adjacent structures.

The handle of the scalpel should be held in such a way that only the tip of the scalpel blade can enter the trachea during the stab incision through the cricothyroid membrane. For control, the physician should place the thumb and index finger low on the handle of the scalpel (just above the scalpel blade). A controlled stab incision should help avoid injury to the posterior wall of the trachea, esophagus, and other posterior structures by limiting the depth of scalpel insertion.

Placement of too large a tracheostomy tube through the cricothyroid membrane should be avoided. Placement of an oversized tracheostomy tube can lead to a fractured larynx. [25] In the adult, the average size of the cricothyroid membrane is about 2 to 3 cm wide and 1 cm high. Thus, the appropriate tracheostomy tube in an adult is one with an 8- to 9-mm internal diameter (a No. 5 in an adult female and a No. 6 in an adult male) unless edema is present, in which case a smaller size (No. 4) is used.

**The Patient with Massive Neck Swelling**

One method of surgical cricothyrotomy uses the hyoid bone as a landmark and an anchor to stabilize the mobile airway in a patient with massive neck edema. [37] [38] The horizontal midpoint of the body of the hyoid is also the midpoint of the neck. The key measurement in this technique is distance from the angle of the mandible to the mental protuberance of the chin (line *A* on Fig. 4-4) . Distances can be measured using a piece of suture or a string. This distance from the angle of the mandible to the mentum is divided in half.

A point is identified on the anterior midline of the neck below the chin (point *C*), which equals line *B* or one half the length of the line *A* (see Fig. 4-4) . A needle is placed  at a point *C* and directed toward the midline so that it will reach an imaginary line connecting

both angles of the mandible. The tip of the needle should hit the hyoid bone. A large-bore (e.g., 18-ga spinal needle) should be used, as the needle must be long enough to pass through massive neck swelling and hit the hyoid bone. If the hyoid bone is not found, the angle of the needle should be adjusted.

When the hyoid bone is found, the needle is left in position, and a No. 11 blade is inserted along the needle tract until the hyoid bone is reached. A skin hook is placed alongside the scalpel's tract and under the hyoid bone to retract the hyoid superiorly and anteriorly. A longitudinal skin incision is made inferiorly from the skin hook with care taken to stay in the midline. The hyoid bone acts as a fulcrum, holding and stabilizing the larynx in position, so that the incision will stay in the midline despite massive swelling of the neck. The incision is extended caudad over the thyroid cartilage until the cricothyroid membrane is exposed. The remainder of the procedure follows the routine steps for cricothyrotomy. The only additional supplies needed for this modification of cricothyrotomy in patients with massive neck edema are the suture (or string) to measure the distance from the angle of the mandible to the mentum of the chin and an 18-ga spinal needle to find the hyoid bone.

### Infant and Children

Cricothyrotomy, as with tracheostomy, is technically more difficult in infants and children than in adults and may have a higher rate of problems or complications. Because the laryngeal prominence is not well developed until adolescence and young adulthood, the most prominent structures in the anterior neck of the infant or child are the hyoid bone and the cricoid cartilage. In children <5 years old, the cricoid is the narrowest part of the airway, and the cricothyroid membrane is quite small. Furthermore, the larynx is higher, relatively less accessible, and smaller in the child than in the adult.

Some advocate tracheostomy rather than cricothyrotomy in infants and children (when endotracheal intubation is contraindicated or impossible), because of the difficulty in palpating and identifying the important landmarks of the neck, and because of the small diameter of the cricoid cartilage. Others note that emergency surgical cricothyrotomy has fewer complications than emergency tracheostomy in infants and children. It has been suggested that a cricothyrotomy be converted to a tracheostomy when the infant or child is stabilized, to reduce the possibility of subglottic stenosis.

### Complications

The incidence of complications for an elective surgical cricothyrotomy is approximately 6 to 8%. The complication rate for an emergency cricothyrotomy ranges from 9 to 31%. [39] [40] The complication rate for emergency cricothyrotomy compares favorably with that for tracheostomy, which has an average complication rate of approximately 45%. [41] The mortality rate for cricothyrotomy of 0.15% is less than that for tracheostomy, which is 1.6 to 5.0% and as high as 16% in recent series. [42] [43] [44] In general, there has been a higher incidence of complications with cricothyrotomy in infants and children than in adults. This is also true for tracheostomy. Tumor, inflammation, other masses, certain congenital anomalies, and significant trauma may distort the anatomic features of the neck and airway, creating additional difficulty in performing a cricothyrotomy and

increasing the complication rate for this procedure.

Minor complications are more common than serious complications (Table 4-6) . Early complications are more frequent than late complications. In one series of patients who had emergency cricothyrotomies, the overall incidence of complications was 23% with no long-term complications. [45]

The most common complications are bleeding, incorrect site of tube placement, unsuccessful tube placement, and prolonged procedure time (see Table 4-6) . The overall procedure time for cricothyrotomy should be less than 3 minutes. It is not unusual for surgical cricothyrotomy to be completed in 30 to 60 seconds.

---

**TABLE 4-6** -- Complications of Surgical Cricothyrotomy

Immediate or early complications
   Common
      Bleeding, hematoma
      Incorrect tube placement
      Unsuccessful tube placement
      Prolonged procedure time
      Subcutaneous emphysema
      Obstruction
   Infrequent
      Esophageal perforation
      Mediastinal perforation
      Pneumothorax, pneumomediastinum
      Vocal cord injury
      Laryngeal fracture or disruption of laryngeal cartilage
      Aspiration
Late complications
   Most common
      Obstructive problems
      Voice changes or dysphonia
      Infections
      Late bleeding
      Persistent stoma
      Subjective feeling of lump in the throat
   Infrequent complications
      Subglottic or glottic stenosis
      Tracheoesophageal fistula
      Tracheomalacia

Bleeding from the procedure is usually minor and occurs early. Such bleeding can usually be controlled by packing the cricothyrotomy site with gauze. It is unusual for bleeding vessels to require ligation. Persistent hemorrhage is usually attributable to vessels located at the edges of a transverse skin incision. This complication is minimized if a longitudinal skin incision is made. [46] Potential bleeding may be minimized by making the stab incision in the lower part of the cricothyroid membrane near the cricoid cartilage to avoid the cricothyroid arteries. Major bleeding generally has not been a problem. [47] [48] One cadaver study noted a high number of small vessels in the region of the cricothyroid membrane that are at risk during a cricothyrotomy. Hence, hemorrhage could still be a problem even if the technique of cricothyrotomy is done correctly and all the guidelines for the procedure are followed. [49] Even a small amount of bleeding could be life-threatening if it occurs endotracheally, with resultant pneumonitis or suffocation. [50] The mortality from acute bleeding during a tracheostomy has been attributed to airway obstruction from the blood and not from volume loss. [51] In the management of postcricothyrotomy or post-tracheostomy bleeding, the airway is protected if the tracheostomy tube is in place and the cuff is inflated. If the airway is protected, then hemorrhage control (i.e., local pressure on the bleeding vessels and, if necessary, ligature) can be done in a controlled fashion.

An incorrectly placed transverse skin incision may lead to bleeding and incorrect or unsuccessful tube placement. Too low a skin incision may lead to tube placement between the tracheal rings. In an emergency, a longitudinal skin incision is preferred, because a misplaced longitudinal skin incision, whether too high or too low, can be easily extended. [52]

Other less common complications include infection and airway obstruction. [53] An indwelling tube will become colonized by bacteria even if meticulous care is taken, and infection can result. Infection can occur in the structures or tissues of the neck and in the tracheobronchial tree. Potential infections include cellulitis, perichondritis, abscess in the incision site, and laryngotracheitis.

Acute airway obstruction can occur with tracheostomy or cricothyrotomy. The most common cause of acute airway obstruction is a mucus plug. A mucus plug that causes obstruction should be cleared from the airway by careful suctioning, using sterile technique (see Chapter 5) . If this cannot be accomplished, then the entire tracheostomy tube may need to be changed. Less frequently, acute airway obstruction can be caused by overinflation of the tracheostomy balloon with herniation of the balloon over the tip of the tracheostomy tube or by retrograde intubation of the pharynx. [54] The tracheostomy tube can be passed into a subcutaneous fascial or tissue plane instead of into the trachea. This complication can occur during insertion of the tracheostomy tube or, more commonly, during a tube change, especially during the immediate postoperative period.

Long-term complications following cricothyrotomy are unusual. [55] The major long-term complication is a change in voice or dysphonia. [56] Laryngeal damage may occur

secondary to a laryngeal fracture caused by using a tracheostomy tube that is too large for the size of the cricothyroid membrane. Dysphonia can also be caused by direct injury to the vocal cords.

Other late and rare complications include a suture sinus, a subjective feeling of a lump in the throat, a persistent stoma, chondritis of the thyroid or cricoid cartilage, and subglottic or glottic stenosis. Swallowing problems have been reported in tracheostomy patients [57] but not in cricothyrotomy patients.

Airway stenosis is a potential complication whenever a tube is placed in the airway. Airway stenosis can occur with intubation, cricothyrotomy, or tracheostomy. The most common cause of subglottic stenosis is endotracheal intubation and not cricothyrotomy or tracheostomy. [58] The pathophysiology of subglottic stenosis involves mucosal ulceration and damage secondary to a tube eroding the mucosal surface and laryngeal disease along with bacterial colonization of the region. Excessive cuff pressures, frequent tube motion, and rigid tubes that are anatomically incorrect, contribute to mucosal damage. The use of flexible, anatomically correct tubes with low-pressure cuffs and flexible tubing between the tube and the ventilator will minimize mucosal damage.

Prolonged endotracheal intubation followed by tracheostomy or cricothyrotomy is associated with a higher incidence of subglottic stenosis than with either endotracheal intubation or the surgical procedure (tracheostomy or cricothyrotomy) alone. [59] The airway stenosis produced by an indwelling tube is generally located at the cuff site of the tube. If airway stenosis occurs with a standard tracheostomy, the stenosis occurs quite distal in the trachea, thus making it difficult to reach during surgical repair. Early aggressive surgical management of granulation associated with mucosal injury has been successful in limiting chronic airway stenosis.  In recent studies of cricothyrotomy, subglottic or glottic stenosis is rarely encountered if at all.

There are other theoretical complications of cricothyrotomy. These include pneumothorax, pneumomediastinum, major vessel hemorrhage, tracheoesophageal fistula, and esophageal perforation. These complications are seen with tracheostomy but have not yet been reported as complications of cricothyrotomy.

The complications of asphyxia, including dysrhythmias and cardiac arrest, during the performance of tracheostomy or cricothyrotomy are generally due to the lack of adequate oxygenation and not to a vagal reaction. Because cricothyrotomy is one of the easiest, quickest, and safest ways to achieve definitive airway control and oxygenation, the complications of hypoxemia should occur less commonly than with tracheostomy when performed in critically ill patients.

## PERCUTANEOUS TRANSLARYNGEAL JET VENTILATION (NEEDLE CRICOTHYROTOMY)

### Overview

Needle cricothyrotomy is a percutaneous technique in which a needle is placed through

the skin, the subcutaneous tissue, and the cricothyroid membrane without using a formal incision. This airway access procedure is *ideally* followed by intermittent high pressure administration of oxygen through the cannula in the cricothyroid membrane ( *translaryngeal jet ventilation*). Only when a sufficiently large catheter (generally 3 mm ID) is inserted, can a low-pressure ventilation source (e.g., bag-valve device) be used to partially support ventilation in the adult. However, a low-pressure ventilation source can be effective for ventilation through a 12- to 14-ga IV catheter in very small infants (5 kg) and may partially support ventilation in larger infants without complete airway obstruction.

The technique of puncture of the cricothyroid membrane is relatively simple and has many uses, ranging from obtaining sputum samples, to giving local anesthesia before intubation, to ventilating the patient.  Whether the lungs can be ventilated adequately via a cannula in the cricothyroid membrane has been debated. We now know that percutaneous translaryngeal ventilation can be an effective and relatively safe procedure for obtaining and maintaining an airway for extended periods of time provided that appropriate equipment and techniques of ventilation are used. [66]

Historically, oxygen was simply passed through the needle or cannula in the cricothyroid membrane ( *apneic oxygenation*). In this way, hypoxia was treated, and tissue oxygenation was generally adequate. Clinical studies and animal experiments noted that although the partial pressure of $O_2$ ($pO_2$ ) was adequate, $CO_2$ was retained, and the elevated partial pressure of $CO_2$ ($pCO_2$ ) led to respiratory acidosis. The oxygenation could be maintained for only about 30 minutes because of the severe hypercapnia and resultant respiratory acidosis, even when the hypoxia was treated successfully. [67] This procedure, whereby oxygen is run continuously through a needle or cannula in the cricothyroid membrane, is referred to as *translaryngeal oxygenation*.

The solution to avoiding a rise in $pCO_2$ and respiratory acidosis is to ventilate the patient, allowing time for both inhalation and exhalation, thus simulating normal respiration. [68] Intermittent bursts of oxygen provide sufficient "tidal volume" and time for exhalation thus avoiding hypercapnia and respiratory acidosis. *Translaryngeal jet ventilation*, is the

ventilation of the lungs by intermittent bursts of oxygen through a needle or cannula in the cricothyroid membrane. The technique can supply adequate ventilation up to high degrees of airway obstruction. Percutaneous *transtracheal jet ventilation* is the same procedure except that the cannula is placed through the larynx into the trachea.

## Physiology

The idea of gas insufflation via an opening in the trachea was described in the early 17th century, [69] although the current usage of the technique has only become popular in the last 25 years. A lack of understanding of the differences between transtracheal oxygenation and ventilation and a failure to standardize the technique itself (with regard to such factors as pressure, volume, intermittent vs continuous administration of air vs oxygen, size of opening in the membrane, and location of opening in the airway) has led

to conflicting reports in the literature and to many misconceptions. [70]

Studies as early as 1909 showed that animals could survive for approximately 30 minutes when oxygen was given via a needle placed in the cricothyroid membrane. [71] Reed and coworkers, in 1954, showed that animals could be kept alive for a longer period of time with a normal pH if they were ventilated with intermittent bursts of compressed air instead of a continuous flow of oxygen. [72] The key to avoiding respiratory acidosis and maintaining a normal pH was the use of a method allowing for adequate ventilation, not just a continuous inflow of oxygen or air (as with apneic oxygenation).

A popular misconception is that percutaneous transtracheal ventilation is a temporizing procedure, good for only 30 minutes at best. Animal experiments and clinical studies have demonstrated that transtracheal ventilation is an effective, quick, fairly simple, and safe way to obtain and maintain an airway for a prolonged period of time. [73] Percutaneous translaryngeal ventilation with an adequate-sized cannula, appropriate ventilation pressures, sufficient tidal volumes, and adequate exhalation times can provide adequate ventilation for extended periods. [74]

Transtracheal jet ventilation has been used extensively as a means of ventilation during surgery and procedures of the upper airway.  Translaryngeal jet ventilation may be used even with partial airway obstruction.] Percutaneous translaryngeal jet ventilation is a rapid procedure for obtaining airway control in both elective and emergency situations in patients of all ages and in many clinical situations.

The advantages of needle cricothyrotomy with translaryngeal jet ventilation over surgical cricothyrotomy include (Table 4-7) faster performance (may take only 10 seconds), easier performance, less equipment and smaller surgical setup required, no need for an assistant, smaller scar, less bleeding, expulsion of oropharyngeal secretions and other particles or small objects from the proximal trachea, less tracheal erosion, and less frequent subglottic or glottic stenosis (see Table 4-1) . Some of the advantages are a result of the smaller size of the opening and the percutaneous technique.

The disadvantages of percutaneous transtracheal jet ventilation are (1) incomplete control of the airway, with a greater potential for aspiration than with a cuffed tracheal

---

**TABLE 4-7** -- Advantages of Percutaneous Translaryngeal Ventilation vs Surgical Cricothyrotomy

Faster (may take as little as 10 sec vs 30 sec to 3 min for surgical cricothyrotomy)
Less bleeding (due to smaller opening, smaller incision)
Simpler technique, easier to perform
Does not need an assistant, can be done by nonsurgeons
Requires fewer instruments
Lesser need for extensive surgical setup (lighting, equipment, and preparation)
Smaller scar (stoma is only as large as the size of the tube)
Less tracheal erosion
Less subglottic or glottic stenosis
Forces oropharyngeal secretions out of the proximal trachea
May force foreign body out of proximal trachea with a partial airway obstruction

tube; (2) likelihood of barotrauma (subcutaneous emphysema or pneumothorax) if exhalation is inadequate and airway pressure is elevated; and (3) inability to perform adequate suctioning through a percutaneous catheter setup. Airway protection during transtracheal ventilation is attained by positioning the patient to allow drainage of secretions away from the larynx during expiration, so upward gas flow through the larynx causes secretions and blood, for example, to be blown away from the larynx. [82]

**Indications**

Indications for needle cricothyrotomy with translaryngeal jet ventilation are similar to those for surgical cricothyrotomy. Cricothyrotomy is indicated in any situation in which intubation is contraindicated or cannot be performed (see Table 4-1) . Failure to achieve endotracheal intubation in a timely fashion, and a subsequent inordinate delay in definitive airway control and oxygenation, is an indication for needle or surgical cricothyrotomy to prevent hypoxemia.

Percutaneous translaryngeal ventilation can be used in a patient with airway obstruction, but it requires an adequate-sized catheter or catheters. [83] [84] Translaryngeal ventilation has been recommended as the procedure of choice for airway control and oxygenation during surgery and procedures on the upper airway. [85] Percutaneous translaryngeal ventilation has been used as an elective procedure and in patients of all ages in an emergency (even premature infants), for extended periods of time. [86]

**Contraindications**

The absolute contraindications to surgical or needle cricothyrotomy are (1) the ability to accomplish endotracheal intubation easily and rapidly; (2) retraction of the distal end of the trachea into the mediastinum after tracheal transection; and (3) occurrence of known significant direct damage to the cricoid cartilage or larynx (see Table 4-3) . [87]

Known complete airway obstruction requires an adequate-sized catheter or catheters to

avoid barotrauma and hypercarbia. Barotrauma with high pressures occurring in the trachea can lead to complications such as a pneumothorax and/or pneumomediastinum during percutaneous transtracheal ventilation. [88] If intratracheal pressures 20 cm $H_2O$ are maintained, the complication rate of percutaneous transtracheal ventilation is low. If complete upper airway obstruction is present, surgical cricothyrotomy is preferred over percutaneous translaryngeal ventilation, although percutaneous translaryngeal ventilation could be used with partial or complete airway obstruction provided appropriate-sized catheters are used. [89]

Relative contraindications to percutaneous translaryngeal ventilation are different from those for surgical cricothyrotomy. Age <5 years has been mentioned as a relative contraindication to surgical cricothyrotomy. Age is not a factor in percutaneous translaryngeal ventilation. Percutaneous translaryngeal ventilation has been recommended by some as the procedure of choice in infants and young children as a means of obtaining an airway, especially in an emergency, rather than tracheostomy or surgical cricothyrotomy. [90]

## Equipment

Translaryngeal jet ventilation requires three components: an oxygen supply at 50 psi, an in-line one-way "valve" to allow for the intermittent administration of oxygen, and a needle or cannula with a bore 13 or 14 ga in the cricothyroid membrane (Table 4-8) .

### Oxygen Supply

The oxygen supply must be at a pressure of 50 psi to deliver enough volume to ventilate the lungs rapidly between periods of exhalation through the glottic opening. An oxygen source of 50 psi can be obtained by attaching a connector to the piped oxygen wall line found in any emergency department, attaching the tubing to the flush valve of an anesthetic machine, or attaching the oxygen tubing directly into an oxygen tank line (regulator) but not to the flow valve of the oxygen cylinder. The oxygen tanks in ambulances or other patient transport vehicles for prehospital care should have two components: (1) an outlet that leads directly into the cylinder line, and (2) a simple, easy-to-connect device that allows the oxygen hose to be quickly attached to the outlet.

Stewart recommends unscrewing the demand valve connection from the oxygen regulator of the tank and rapidly

---

**TABLE 4-8** -- Equipment for Needle Cricothyrotomy and Translaryngeal Jet Ventilation

Oxygen source
   High-pressure oxygen source at 50 psi
   Deliver 100% oxygen at 20 bursts/minute with I:E = 1:4
Manual jet ventilator device
   High-pressure tubing (to be attached to cannula or catheter and oxygen at other end with the manual jet ventilator device in the middle)
   Manual jet ventilator device (Y-connector or push-button device) to allow for ventilation with inhalation and exhalation
Cannula
   Large bore (gauge less than or equal to 13- or 14-ga needle) with a Teflon or plastic cannula or, alternatively, one of the commercial cricothyrotomy devices, or 9 Fr catheter

I , inhalation; E, exhalation.

connecting the oxygen hose that has a nipple in place for fitting into the outlet. [69] He suggests leaving a small wrench fastened to the oxygen hose to allow a rapid transfer.

Two key points should be remembered: (1) high-pressure tubing should be used throughout the system, and (2) all connections and attachments should be well secured to avoid any disconnection under high pressure. Ligatures or other fasteners may be helpful in securing the attachments.

**"Valve" Apparatus**

It is essential to have the proper attachment for delivery of oxygen to the cannula or needle. Whatever the device or valve apparatus selected, it is critical to have all of the equipment ready, all of the connections available, and everything easily accessible. The apparatus can be gathered together and placed in one bag or kit and should be stored with the other airway management equipment.

First, high-pressure tubing goes from the oxygen source at 50 psi to a manually controlled device, which is then connected via high-pressure tubing to the cannula or needle in the cricothyroid membrane (Fig. 4-5) . Stewart [70] recommends the manual valve by Instrumentation Industries (Bethel Park, Pa), although others such as the Manujet by VBM (Germany) are also available. [69] A list of other "valve" apparatus options with varying costs ($5 to $200) has been published elsewhere. [91]

Note that standard commercial demand valves deliver only 60 cm $H_2$ O (approximately 0.9 psi). [87] Systems using makeshift bag-valve assemblies (Fig. 4-6) do slightly better, but may deliver as little as 12 to 15 L/min of oxygen using 14- and 13-ga catheters,

respectively.

## Cannula or Needle

The third piece of equipment required is the cannula. A 13- or 14-ga needle catheter with a length of about 2 cm (or 1 inch) or a 3-mm ID cannula is preferred for percutaneous translaryngeal ventilation. The size of the needle or cannula is critical. [92] Too small a cannula will not allow for sufficient ventilation, causing hypercapnia and even hypoxemia to develop.

Modifications for the cannula have been suggested: side holes, a slight curve, and a plate or flange. The advantage of side holes is that they will help keep the end or point of the cannula away from just one area of the tracheal wall. Thus, the full force of the 50 psi oxygen source is not directed at just one area of the laryngeal wall but is distributed in the larynx. The shape of the cannula may facilitate its initial placement or enhance complications. A fixation plate, flange, or side handle helps secure the cannula into place. A snug fit of the cannula will help prevent subcutaneous emphysema and ventilation of overlying soft tissue.

The use of a large-bore (9 Fr ID with 13 Fr outside diameter [OD] and length of 6 cm) has been recommended instead of a 14-ga IV catheter. The large-bore cricothyrotomy catheter has several advantages: less chance of kinking or plugging up, improved mechanical structure, and the ability to be used with jet ventilation or bag-valve device ventilation.

## Commercial Kits

Cannulas designed for cricothyrotomy or percutaneous translaryngeal ventilation are commercially available (Table 4-9) . Several studies suggest that the "commercial" kits may have a higher complication rate due to a higher puncture/insertion force and/or a curved shape with more frequent posterior perforation.  Placement of a commercial device may take longer than the traditional surgical cricothyrotomy.  The use of one commercial cricothyrotomy device (Pertrach) is shown in Figure 4-7 (Figure Not Available) .

## Ventilation

Ventilatory parameters may be critical in preventing barotrauma and significant hypotension and in eliminating hypoxemia and hypercarbia. Animal studies suggest that the minute volume as reflected by a short inspiratory and a prolonged expiratory phase is an important parameter.

*Bag-valve devices should not be used for ventilation* of adults or children unless a large catheter (3 mm ID) has been passed. The clinician should verify that adequate tidal volumes are being delivered with this technique by monitoring for hypoxemia.

**Procedure**

The actual technique of needle cricothyrotomy with translaryngeal jet ventilation is fairly simple (Fig. 4-8) . The anatomy, landmarks, and several of the steps in the technique are identical to that for surgical cricothyrotomy (see Figs. 4-1 and 4-3) .

---

**TABLE 4-9** -- Commercial Devices

**Commercial Cricothyroidotomy Devices**

There are many commercial cricothyrotomy devices. A few of them are listed below. Mention is not intended to imply endorsement of these devices.

1. NU-TRAKE and PEDIA-TRAKE are distributed by International Medical Devices, Inc., PO Box 408 Canoga Park, 19355 Business Center Drive, Suite 8, Northridge, CA 91324, or PO Box 408 Canoga Park, CA 91305; phone (818) 701-5433 or (800) 522-LIFE (outside California)

2. Pertrach: Pertrach, Inc., 900 Davisson Run Rd, Suite 301, Clarksburg, WV 26301, phone (304) 624-7122 or (800) 736-3194

3. Abelson cricothyrotomy cannula: Gilbert Surgical Instruments, 115 Harding Ave, PO Box 458, Bellmawr, NJ 08031, phone (609) 933-2770

4. Quick Trach emergency cricothyrotomy device for adults and infants: VBM Medizintechnik GMBH, D-7247 Sulz am Neckar, Germany, phone (07454)6211; telefax (07454)4953

5. Portex Mini Trach Kit

| |
|---|
| 6. Melker emergency transcricothyrotomy catheter set: Cook Critical Care, PO Box 489, Bloomington, IN 47402; phone (800) 457-4500 |
| **Commercial Transtracheal Puncture Devices** |
| Also designed for transtracheal puncture, the Trans-Cricothyrotomy device is distributed by VBM Medizintechnik (same address and phone as above) |
| **Commercial Manual Ventilator Devices** |
| 1. Manual valve that allows for the flow of oxygen when the control button is pushed. Manual Valve, by Instrumentation Industries, Bethel Park, PA |
| 2. Manujet: a manual jetting device by VBM Medizintechnik GmbH, Bruhlstrasse 10, 0-727 Sulz am Neckar, Germany |

A small 3- to 5-mL syringe containing 1 to 2 mL of sterile normal saline or water is attached to a large-bore needle (13 or 14 ga). If lidocaine will be injected, then 1 to 2 mL of 1% lidocaine without epinephrine should be in the syringe. If the patient is responsive, 1 to 2 mL of lidocaine may be injected into the larynx to prevent reflex coughing when the needle or cannula enters the larynx.

While the dominant hand (usually the right) holds the syringe, with the needle directed caudally at less than 45° to the skin, the other hand (nondominant) holds and stabilizes the larynx. The thumb and middle fingers of the nondominant hand stabilize the cricoid cartilage, and the index finger palpates the cricothyroid membrane.

The needle is inserted through soft tissues, the skin, and the cricothyroid membrane (see Fig. 4-8) . While negative pressure is exerted on the barrel of the syringe, the needle is inserted through the cricothyroid membrane into the larynx. Air bubbles in the fluid-filled syringe signify entry into the larynx. After entering the larynx, the cannula is advanced into the larynx, and then the needle is removed. The cricothyroid membrane should be punctured in the inferior aspect (i.e., nearer the cricoid cartilage than the thyroid cartilage) to avoid the cricothyroid arteries (see Fig. 4-3 *A*).

If there is much resistance to the needle's or catheter's passage through the skin,

subcutaneous tissue, or cricothyroid membrane, kinking or bending of the catheter may occur unless a stiffer catheter is used. A small nick in the skin may be needed to facilitate passage through the dermis into the subcutaneous tissue. A percutaneous dilatational or Seldinger technique may result in fewer complications. [103] [104]

The cannula is secured by suturing it to the skin or by placing a circumferential tie around the neck. The oxygen source is connected to the cannula. It is critical that the proximal end of the cannula be snug or tightly fitting and securely held around the puncture wound opening. If it is not securely held in place, subcutaneous emphysema will result and/or the cannula may be dislodged from the larynx. A trial of several bursts of oxygen flow is recommended to make certain that the cannula is correctly placed and the setup is working and ventilating properly.

The hypoxic patient should receive 100% oxygen in intermittent bursts <50 psi at a rate of 20 bursts per minute. For children, 30 psi has been recommended. The percentage of inspired oxygen concentration can then be adjusted, depending on blood gas results. The inspiratory phase or insufflation with the burst of oxygen should last approximately 1 second, and the expiratory phase long enough to allow for adequate exhalation (2 to 9 seconds has been suggested).

**Precautions**

The location of the needle puncture (or incision) in the skin and cricothyroid membrane is critical. The preferred location is near the midline in the inferior aspect of the cricothyroid membrane, just above the cricoid cartilage. The needle (or scalpel) is directed at an approximately 45° angle to the skin in a caudal direction. The key landmarks in the neck should be identified before inserting the needle-cannula combination into the cricothyroid membrane.

Too small an opening in the cricothyroid membrane leads to hypoxemia, hypercapnia, and respiratory acidosis. In an adult, a 13- or 14-ga needle or larger bore with a 3-mm ID is needed to provide an adequate-sized opening.

Use of a bag-valve device or a demand valve resuscitator apparatus to ventilate a patient through a translaryngeal cannula is generally inadequate. Using a 50 psi oxygen source, up to 89 L/min can be delivered through a 13-ga cannula, and up to 72 L/min can be delivered through a 14-ga cannula. [87] Additional side holes or ports in the cannula to allow for the additional egress of oxygen into the trachea and to limit the obstruction of the cannula by mucus plugs are desirable.

During the insufflation phase of percutaneous translaryngeal ventilation, secretions in the upper airway are blown out of the mouth and nose. It is recommended that personnel stand clear of the patient's face to avoid being sprayed with secretions when oxygen exits through the patient's glottis and pushes oropharyngeal secretions out the glottis and then out the nose and mouth. This may be a useful side effect if partial upper airway obstruction is present, as with a foreign body (e.g., a bolus of meat or a peanut), because some of the delivered volume of air exiting through the patient's glottis might dislodge such a foreign body. The risk of aspiration is decreased or eliminated by

lowering the head. The proximal end of the cannula should be snug against the puncture wound to minimize localized subcutaneous emphysema and should be secured in place to prevent the cannula from being dislodged.

## Complications

The exact incidence of complications with percutaneous translaryngeal ventilation is not known, but it is thought to be low, considering that the complication rate of translaryngeal puncture alone is in the range of 0.03 to 0.8% (Table 4-10) . [105]

Bleeding can occur at the site of the needle puncture, but this is usually not a major problem. Hemoptysis may occur but is infrequent. Fatal hemorrhage secondary to transtracheal aspiration for sputum cultures has been reported, [50] although no bleeding fatalities secondary to percutaneous transtracheal ventilation have been reported.

Minor bleeding is the most common complication of a surgical cricothyrotomy, whereas subcutaneous emphysema is the most frequent complication of percutaneous translaryngeal ventilation. With percutaneous translaryngeal ventilation, subcutaneous emphysema will occur if egress of gas is prevented by inadequate exhalation (e.g., with occlusion of the mouth and nose or with inadequate cannula size). The development of subcutaneous emphysema with percutaneous transtracheal ventilation is often immediate and dramatic. If this complication does occur, the subcutaneous emphysema may be gently squeezed away from the midline and the trachea can often be recannulated and successfully ventilated. Subcutaneous emphysema can be decreased or avoided by making sure that the flange or hub of the cannula fits securely against the skin.

Barotrauma secondary to high airway pressures may occur if percutaneous translaryngeal ventilation is used when complete airway obstruction is present. Such barotrauma could lead to serious complications, including pneumothorax, pneumomediastinum, and subcutaneous emphysema.

Another side effect that can occur in the conscious patient is coughing with each burst of oxygen. A few milliliters

---

**TABLE 4-10** -- Complications of Percutaneous Translaryngeal Jet Ventilation

Common
   Subcutaneous emphysema--most common (less occurrence if there is a "secure" fit at the skin)
   Kinking of the catheter
   Blockage or obstruction of the catheter
   Coughing (in a conscious patient)
Infrequent
   Bleeding (minor), hematoma
   Infections
   Aspiration
   Incorrect or unsuccessful catheter placement
   Prolonged procedure time
   Persistent stoma
   Subjective feeling of a "lump in the throat"
   Pneumatocele
Serious, rare complications
   Barotrauma (secondary to high airway pressures, more common with complete airway obstruction)
   Pneumothorax
   Pneumomediastinum (less occurrence if high airway pressures are avoided, and not performed with complete airway obstruction)
   Mediastinal perforation
   Esophageal perforation
   Dysphonia or voice changes (secondary to vocal cord injury, laryngeal fracture, or disruption of laryngeal cartilage)
Potential or theoretical complications (not yet commonly associated with percutaneous translaryngeal jet ventilation, although reported with tracheostomy and other airway procedures)
   Subglottic/glottic stenosis
   Tracheoesophageal fistula
   Damage to laryngotracheal mucosa (such as tracheobronchitis)
   Swallowing problems

of lidocaine may be injected into the larynx to help prevent coughing when the cannula enters the larynx. The initial instillation of lidocaine solution will precipitate a brief coughing episode, but further coughing should be minimized. However, because the patient undergoing cricothyrotomy in the emergency department is almost always critically ill and is rarely conscious, coughing is usually not a problem.

Percutaneous translaryngeal ventilation for an extended period of time can dry the membranes of the airway unless appropriate humidification is obtained. Techniques or devices for humidification are available. [106]

One complication that may occur with needle cricothyrotomy is kinking of the catheter as it enters the neck. This problem can be overcome if a stiffer catheter is used. A 14-ga

(1¼ inch) catheter or any device approximately 2 cm long from the hub or flange of the cannula has been recommended as the best length, because it puts the tip of the catheter in the middle or midstream of the airway.

Infection, ranging from cellulitis, perichondritis, or laryngotracheitis, is a possible complication of percutaneous translaryngeal ventilation, but it has not been reported in most series. Careful technique and follow-up care should help prevent these complications of infection and obstruction by blockage of the tube.

Damage to the laryngotracheal mucosa, including necrotizing tracheobronchitis, from percutaneous translaryngeal ventilation is a theoretical complication that has not been reported. Most studies have concluded that percutaneous translaryngeal ventilation or high-frequency jet ventilation may be performed "without undue risk of tracheobronchial injury." [107]

Blockage or obstruction of the tube from bleeding or from a mucus plug occurs infrequently, although such blockage can cause acute airway obstruction. Additional small holes near the tip of the catheter may help to prevent this problem.

A pneumatocele caused by incorrect needle placement is a rare and benign complication that can be treated by aspiration with a needle. Misplacement of the cannula can lead to tracheal, mediastinal, or esophageal perforation if the needle is advanced too far. Other complications that occur with surgical cricothyrotomy could occur with needle cricothyrotomy, such as damage to the laryngeal cartilage, which may cause dysphonia or voice changes.

## CONCLUSION

Cricothyrotomy, is a simple, reliable, rapid, and effective means of achieving airway control and ventilation. It has relatively few complications. The value of cricothyrotomy as a life-saving procedure in emergency situations has been clearly demonstrated, and its usefulness has been well established in emergency care. Both surgical cricothyrotomy and needle cricothyrotomy with translaryngeal jet ventilation can be used for an extended period of time if appropriate ventilation factors are maintained. Cricothyrotomy has also gained acceptance as an elective procedure for surgical access to the airway. In many instances, cricothyrotomy is the airway procedure of choice.

# Chapter 5 - Tracheostomy Care and Tracheal Suctioning

**Steven M. Joyce**

## TRACHEOSTOMY CARE

The key to optimal routine tracheostomy care is cleanliness, which keeps the tracheostomy tube free of obstruction. Tube changes are done routinely but also may be necessary in an emergency. The emergency physician should be alert for late postoperative complications such as tube obstruction or displacement, false passage, hemorrhage, infection, tracheal stenosis, and tracheoesophageal fistula. In this chapter, several procedures are discussed that may be useful to the emergency physician when a complication is recognized.

Tracheostomy, one of the oldest operations described in the medical literature, is used when long-term mechanical ventilation or control of airway secretions is indicated. It is also performed occasionally for relief of upper airway obstruction, especially in children. [1] The procedure is usually performed in the operating room and occasionally at the bedside. Complications requiring emergency management, although infrequent, can occur in both the early and late postoperative periods. The emergency physician is most likely to encounter complications occurring during the latter period and should be familiar with the equipment and techniques for handling tracheostomy-related emergencies.

Tracheostomy tubes are made of many materials, including metal, Silastic, Teflon, polyethylene, and rubber. They may be cuffed or uncuffed and may have a single lumen or a removable inner cannula (Figs. 5-1 (Figure Not Available) and 5-2) . A removable blunt-end obturator may be used to aid passage. The inner cannula is a removable tube that fits snugly inside the tracheostomy tube itself. Normally the patient breathes through the inner cannula; the cannula can be removed daily for cleaning (which is usually performed with hydrogen peroxide and water), thereby lessening the buildup of mucus in the tracheostomy tube. Tubes may be curved, angled, or flexible. [2] Although it is important that the emergency physician recognize the type of device in place, the basic and emergency care of the tracheostomy itself is relatively independent of the type of tube used.

### Routine Tracheostomy Care and Changing a Tracheostomy Tube

The most important factor in keeping a tracheostomy tube functioning is the control of mucus to maintain the patency of the tube or inner cannula. If humidified air is inhaled, mucus remains liquid and generally is expelled freely by coughing. The use of intermittent humidified air at home may minimize inspissation of mucus in the trachea and cannula. When mucus is excessive, suctioning is performed as often as needed to clear secretions and is usually done by the patient or family. The technique for suctioning through a tracheostomy tube is described later in this chapter. Clearing of

inspissated secretions may be aided by instillation of 5 to 10 mL of sterile saline.

Once the tracheostomy wound has matured, generally at about 5 days after surgery, routine changing of the tracheostomy tubes can be carried out safely and is necessary to clean the tubes satisfactorily. This is usually done by the patient or family on an as-needed basis, generally about every 2 weeks. Emergencies may also occasionally require changing of the tracheostomy tube.

## Equipment and Setup

Before changing the tracheostomy tube, the new tube should be carefully checked. It should be the same size, and preferably the same type, as the tube being removed. Size is usually marked on the tube or neck plate and usually reflects the inside diameter in millimeters. All component parts should fit together easily, and when a cuffed tube is placed, the cuff should be tested under pressure. Once this has been accomplished, the patient is positioned *with the neck hyperextended*, when possible. Adults may sit or lie down. Children should be lying down with a rolled towel under the shoulders, and held firmly. Some patients may require soft hand restraints during the changing procedure. Because breath holding or coughing may occur during the procedure with resultant hypoxia, it is optimal to provide supplemental oxygen through the existing tracheostomy tube prior to the procedure. However, patients with near total obstruction require immediate relief of the obstruction, as discussed later in this chapter.

## Technique

The patient's tracheostomy tube should be removed in a single sweeping motion, and the new tube, which has been prepared with the obturator in position and lubricated, should be gently inserted during inspiration into the stoma with the same sweeping circular motion. No force should be exerted, because creating false passages is deceptively easy and may prove disastrous. The tube may also be changed by using a removable obturator or over a catheter using a modified Seldinger method, which greatly reduces the chance of creating a false passage (Fig. 5-3) .

Once the tracheostomy tube is in position, the obturator is removed, and the inner cannula (if needed) is placed. Proper position is confirmed by good airflow and breath sounds with inspiration and expiration. The tracheostomy tube should be tied snugly around the patient's neck with tracheostomy tape while the head is held in flexion. The tape should be touching the skin around the neck with nothing intervening. Slight tension should be felt as a finger is passed under the tape. If an inner cannula is being used, the patient should be instructed on how it can be removed for cleaning. Furthermore, the patient should be encouraged to remove it for cleaning as often as necessary to keep it mucus free.

## Complications of Tracheostomy

### Obstructed Tracheostomy Tube

A tracheostomy patient who presents with respiratory distress should be *assumed to have a partially or completely obstructed tracheostomy tube*. Such patients should immediately be placed on high-flow humidified oxygen, with the flow directed either at the tracheostomy tube or over the mouth, whichever has the best airflow. The usual cause of obstruction is inspissated mucus. Obstruction usually occurs in the inner cannula or tracheostomy tube itself, although more distal tracheal plugging can also occur.

If an inner cannula is present, it should be removed so it can be cleaned and obstructing plugs removed. If a cannula is not present, immediate suctioning is appropriate in an attempt to clear the tracheostomy tube. If suctioning is not successful, 5 to 10 mL of sterile saline should be injected into the trachea and suctioning repeated. This loosens mucus and stimulates coughing, often enough to dislodge a large obstructing plug. If suctioning and removal of the inner cannula do not immediately clear the airway obstruction, the entire tracheostomy tube should be removed and replaced with a new tube. [4] In an emergency, one should not hesitate to remove a tracheostomy tube, because most patients are able to breathe easier through a stoma than through a partially blocked tube. The tube may then be replaced as described above, using a catheter or flexible laryngoscope as a guide. [3]

Persistent respiratory distress despite a successful tube replacement should lead to a search for other etiologies (e.g., low tracheal stenosis, pneumonia, aspirated material). Oral endotracheal intubation should be considered if the physician is unable to replace the tube in a ventilator-dependent patient.

**Displaced Tube and Stomal Closure**

A tracheostomy tube may come out during either cleaning or coughing and cannot be replaced because the stoma is closing. Even if the tube has been out for only a few hours, compromise of the stoma may have already begun. Accept the patient's judgment that the old tube will not fit. Forceful attempts to insert the same size tube or to dilate the stoma with hemostats may create false passages, bleeding, and traumatic edema, making reintubation almost impossible. The patient's tube should be inspected to identify its size, which can usually be found on the neck plate. A new tracheostomy tube one or two sizes smaller can generally be inserted with relative ease. Care should be taken to avoid false passages. Once the smaller tube has been inserted to maintain the tracheal stoma, the stoma may gradually be enlarged on a daily basis by insertion of larger tubes until a maintenance tube of appropriate size can again be worn. [5]

**False Passage**

Occasionally, when a tracheostomy tube has been removed, the proper passage for easy insertion of a new tube cannot be found. Often, simple extension of the neck will line up tissue planes and facilitate passage. False passages are easily created and can be recognized by failure of positive-pressure ventilation to inflate the lungs and rapid development of subcutaneous emphysema. Use of a catheter as a guide prior to removal of the old tube should prevent this complication. A catheter or laryngoscope

also can be inserted into the stoma and the new tube advanced over it (see Fig. 5-3) . [5]

**Hemorrhage**

After the immediate postoperative period, bleeding from a tracheostomy should be considered a life-threatening emergency until proven otherwise. A tracheoinnominate fistula has been reported in up to 4.5% of tracheostomies and is most common between 1 and 3 weeks postoperatively. [6] The fistula is caused by erosion from an improperly fitted or placed tube, a low tracheotomy site, or an overinflated cuff. This life-threatening complication may be preceded by coughing of more than 10 mL of blood from the tube or noticeable pulsating tube movement. Death is by exsanguination or asphyxiation from aspirated blood and occurs in more than 86% of cases with this complication. [7]

If premonitory bleeding and hemoptysis occurs, the patient should undergo prompt bronchoscopy with the tube removed to inspect the tracheal mucosa and identify the bleeding site. When massive hemorrhage occurs and a tracheoinnominate fistula is suspected, the tube cuff should be overinflated to apply pressure from inside the trachea. If significant hemorrhage continues, digital pressure may be applied simultaneously over the skin of the suprasternal notch, or a gloved finger can be inserted through the skin incision and into the pretracheal soft tissues to compress the artery. [7] [8] Digital pressure is maintained until emergency sternotomy can be performed and the vessel ligated.

Minor bleeding (i.e., <10 mL) at the stoma may be due to minor trauma during tube insertion. If visual inspection clearly identifies a superficial bleeding site, it may be packed or cauterized.

**Infection**

Bacterial contamination of the granulating stoma invariably occurs, but infection is uncommon. Early peristomal cellulitis is treated with oral antibiotics and close follow-up. Paratracheal abscess requires prompt drainage and intravenous antibiotics to prevent mediastinitis. Necrotizing infections around the stoma are rare and are treated by prompt removal of the tracheostomy tube, placement of an oral or nasal endotracheal tube, and aggressive debridement and antibiotic therapy. [6]

**Tracheal Stenosis and Malacia**

Overinflation of the tracheostomy tube cuff may result in pressure necrosis of tracheal tissues, which may subsequently result in tracheal stenosis or malacia. Symptoms usually develop several weeks or longer after decannulation but may become apparent while the tube is still in place. Initially the patient may complain of dyspnea on exertion, chronic cough, or inability to clear secretions. As obstruction progresses, dyspnea at rest and stridor may develop.

Initial treatment for partial obstruction with the tube in place, as described above, should be attempted but is unlikely to afford improvement. If the patient has been

decannulated, the stoma will be closed or nearly so, and endotracheal intubation will not be likely to bypass the narrowed segment, which is several centimeters below the stomal site.

The patient's head should be elevated and humidified supplemental oxygen provided. Nebulized racemic epinephrine and parenteral steroids have been suggested. [7] Plain radiographs may not be helpful. Diagnosis is made by bronchoscopy, and dilation or stenting may be attempted during this inpatient procedure. Definitive treatment is surgical resection of the stenosed or weakened section and tracheal reconstruction.

**Tracheoesophageal Fistula**

Development of a fistula between the trachea and esophagus in the postoperative period is rare and is usually due to pressure on the membranous trachea by an overinflated cuff or poorly positioned tube. Increased secretions and coughing after oral feeding are the common presenting symptoms. Aspiration of food or gastric contents may cause pneumonia or pneumonitis. Gastric distention may be noted in ventilated patients. Mediastinitis may develop.

Treatment is surgical repair, as these fistulae do not close spontaneously. Repair may be deferred until positive-pressure ventilation is no longer needed, although a feeding gastrostomy is necessary in these cases. [7]

**Conclusion**

Knowledge of the basic types and routine care of tracheostomy tubes, as well as the scope of postoperative problems, should allow the emergency physician to deal confidently with emergent complications of tracheostomy.

# TRACHEAL SUCTIONING

Tracheal suctioning removes secretions, aspirated material, or both from the airways of patients whose cough reflex has failed or is bypassed by an artificial airway. Removal of secretions prevents atelectasis, improves gas exchange, and helps eliminate infected material. Secretions obtained by suctioning also may be useful in diagnosis of pulmonary infections. With minimal training, the procedure may be

performed by respiratory therapists, nurses, or physicians. Although tracheal suctioning may be performed easily at the bedside, there is a potential for serious complications, so proper precautions must always be taken.

Tracheal suctioning has been practiced since the introduction of positive-pressure ventilation in the 1950s as a method of removing tracheobronchial secretions. The technique remains vitally important for the maintenance of proper gas exchange in

endotracheally intubated patients and in selected patients whose cough reflex is otherwise impaired. The development of new catheter materials has resulted in catheters with various diameters and tip designs that minimize mucosal injury and allow selective bronchial catheterization. Other features, such as concomitant oxygen delivery with in-line systems, permit secretion removal while minimizing hypoxemia.

**Indications and Contraindications**

The following discussion refers principally to tracheal suctioning through an endotracheal or tracheostomy tube. Nasotracheal suctioning is discussed separately.

The basic indications for tracheal suctioning are the need to perform bronchopulmonary toilet in intubated or obtunded patients and to sample lower respiratory tract secretions for diagnostic tests (when coughed specimens are inadequate or cannot be obtained). In an emergency setting, tracheal suctioning is usually performed immediately after endotracheal intubation to clear the airways of aspirated materials or accumulated secretions.

Bronchopulmonary toilet is indicated when secretions are visible at the endotracheal tube orifice; when auscultation of the lungs reveals coarse rales, rhonchi, or tubular breath sounds; or when the sudden onset of dyspnea or arterial oxygen desaturation occurs in patients at risk for lower airway occlusion. Those at risk include all patients with artificial airways (which make coughing ineffective), especially those patients with bronchopulmonary infections and excess secretions.

Most authors agree that "routine" suctioning is unwarranted and potentially dangerous; the procedure should be performed only on an as-needed basis when the previously mentioned indications exist. [9] [10] [11] The only relative contraindication to tracheal suctioning is cardiovascular instability, which is exacerbated by the procedure, as in the patient with cardiogenic pulmonary edema and hypoxemia. However, even in such cases, when treatment of the underlying disorder is not sufficient to maintain adequate gas exchange, tracheal suctioning should be undertaken using techniques to minimize the likelihood of hypoxemia and arrhythmia.

**Equipment**

Appropriate vacuum settings for optimal secretion removal and minimal mucosal trauma have been proposed. [10] Settings of 60 to 80 mm Hg for infants, 80 to 120 mm Hg for children, and 120 to 150 mm Hg for adults are recommended. Higher settings cause turbulent flow through the catheter, which can reduce the efficacy of secretion removal and may increase tracheal mucosal injury.

Catheters made of polyvinyl chloride are most commonly used. They are flexible; require no lubrication; and are translucent, allowing visualization of secretions. [9]

The external diameter of the suction catheter should be approximately one half the internal diameter of the endotracheal tube. [11] Larger catheters may prevent adequate

airflow into the airways during suctioning, and the resultant evacuation of gases may result in atelectasis and hypoxemia. However, in the setting of life-threatening airway obstruction, the largest catheter that will fit the airway should be used. Smaller diameter catheters may not remove secretions effectively.

Catheter tip designs vary depending on the intended use. Conventional catheters with an end hole and several side holes are the most commonly available types in an emergency department setting. Tips designed for specialized uses exist but offer no great advantages for uncomplicated tracheal suctioning. Catheters designed for selective bronchial suctioning are seldom used in an emergency situation. A closed in-line system that does not require interruption of the ventilator circuit has been developed for use in ventilated patients who have unstable cardiovascular conditions or are dependent on positive end-expiratory pressure (PEEP). [11] Use of such a closed system has been shown to reduce the incidence of both hypoxemia and arrhythmia during suctioning. [12]

A cardiac monitor and, if available, a pulse oximeter should be attached prior to the procedure. Standard resuscitation drugs and equipment should be immediately available.

**Procedure**

The clinician should assemble all equipment and ensure that it is functioning properly.

Aseptic technique should be used as much as possible. Hands are washed with antimicrobial soap, and a sterile glove is used to manipulate the suction catheter. In emergencies, asepsis may not be practical, but one should try to minimize contamination of the airways.

The clinician should preoxygenate the patient by delivering 6 to 7 breaths of 100% oxygen either by handheld bag or ventilator. However, many ventilators will not deliver an $Fi_{O2}$ of 1.0 for up to 2 minutes after the setting is changed. [10]

Immediately after preoxygenation, introduce the sterile catheter into the endotracheal tube with the gloved hand, taking care *not* to occlude the vacuum vent during insertion. Pass the catheter tip past the distal end of the artificial airway until resistance is met (usually at the carina). Pull the catheter back slightly before applying suction. Then occlude the vent to apply suction and rotate the catheter in a circular motion around the tube lumen while slowly withdrawing it from the airway.

This entire procedure should interrupt the patient's ventilation for only approximately 20 seconds, with the catheter actually in the airway for <15 seconds, to minimize hypoxemia and atelectasis. [11]

Immediately after the procedure, the patient should be reoxygenated using a combination of hyperoxygenation, hyperventilation, and hyperinflation. Hyperoxygenation is achieved by delivering an $Fi_{O2}$ higher than the patient's

maintenance value (generally 100% oxygen is used). Hyperinflation is achieved by delivering up to 1.5 times the usual tidal volume, again as estimated either by bag ventilation or by using the ventilator sigh function. A clinical study by Baun and an exhaustive review by Barnes and Kirchoff support

the combination of these techniques to minimize postsuctioning hypoxemia and its attendant complications. [13] [14]

Thick secretions may be difficult to remove. In these cases, 5 to 10 mL of sterile normal saline should be injected into the artificial airway, after which several deep breaths should be delivered by bag or ventilator just before suctioning. After the procedure, $Fi_{O2}$ and other ventilator settings should be returned to presuctioning values. Clotted blood or other thick secretions not removed by suctioning may be retrievable by extraction with a No. 6 Fogarty catheter. [15] [16] The catheter is passed beyond the occluding material and then inflated. The material is pulled ahead of the catheter during its withdrawal.

Secretions for microbiologic studies may be obtained using a sterile mucus trap. The specimen is transported in the sealed trap for prompt processing.

**Complications**

As with most invasive procedures, tracheal suctioning is not without the potential for serious complications. Fortunately, most can be prevented or minimized.

Difficulty in passing a suction catheter may be due to the patient biting on the endotracheal tube, airway kinking, inspissated secretions, herniation or rupture of the cuff balloon, tube malalignment, or placement of the tube outside the trachea. Solutions to these problems include placement of a bite block; removal of redundant endotracheal tube length to prevent kinking; injection of saline down the tube to loosen secretions; and prompt replacement of blocked, malfunctioning, or malpositioned tubes.

Hypoxemia, although transient, is perhaps the most serious complication of tracheal suctioning, as it can lead to life-threatening cardiac dysrhythmias. Inadequate oxygenation before or after suctioning, prolonged suctioning (i.e., more than 15 seconds), and excessive catheter size are the most common causes. Hypoxemia may be presumed when dysrhythmias develop during or just after the procedure. Dynamic oxygen saturation measurement with a pulse oximeter may prove helpful. Fortunately, hypoxemia may be prevented or minimized by proper oxygenation techniques, as described above. If available, some suction catheters allow alternating or simultaneous oxygen delivery through the catheter itself during the procedure. [17] [18] If the patient is ventilator dependent, a closed in-line suction system has been shown to reduce hypoxemia during this procedure. [12]

Dysrhythmias may be caused by hypoxemia, as discussed previously, as well as by tracheal irritation. When the mucosa is irritated by the suction catheter, both vagal and sympathetic afferents may be stimulated. Vagal stimulation may produce bradycardia with resultant hypotension, whereas sympathetic stimulation can precipitate

tachydysrhythmias or even ventricular fibrillation, especially when hypoxemia is present. The incidence of dangerous dysrhythmias during suctioning is unknown, but they are probably not rare. Shim and coworkers described significant dysrhythmias (some persisting for 2 minutes) that occurred in 6 of 17 patients undergoing suctioning while breathing air; all dysrhythmias were abolished when the patients breathed 100% oxygen for 5 minutes before undergoing the procedure. [19] In patients subject to bradycardia during suctioning, both nebulized and parenteral atropine were effective in preventing this arrhythmia. [20] Thus, attention to cardiac monitoring and oxygen saturation during and after suctioning is very important. Suctioning should be terminated immediately when dysrhythmias (other than preexisting dysrhythmias or sinus tachycardia) are observed, and oxygenation should be provided as previously described.

Intracranial pressure (ICP) may increase transiently during tracheal suctioning, which leads to theoretic concerns in patients whose cerebral perfusion pressure is already marginal. Rudy and colleagues reviewed a number of studies examining this transient ICP elevation; none were found to demonstrate a clinically significant deleterious effect in the patients studied. [21] They also reviewed studies that examined the efficacy of pretreatment with various drugs to prevent increases in ICP with tracheal suctioning. No significant effect of lidocaine, fentanyl, thiopental, or succinylcholine in abating the ICP increase with suctioning was demonstrated, with the exception of intravenous lidocaine (1.5 mg/kg) in patients receiving concurrent pentobarbital therapy. Other studies of patients with head injuries who had baseline elevations in ICP suggest that the cough reflex is the predominant cause of ICP rise with suctioning in the unanesthetized and nonparalyzed patient. Both intravenous lidocaine (1.5 mg/kg) and intratracheal lidocaine (2 mL of a 4% solution) administered before suctioning or temporary paralyzation attenuated ICP increases during suctioning in patients with severe head injuries. [22] [23] However, for patients without signs of elevated ICP, routine pretreatment with drugs to prevent transient ICP increases is probably unnecessary.

Tracheal mucosal injury is a frequent complication of tracheal suctioning. Some degree of irritation accompanies even properly performed suctioning, but it can be minimized. Catheters of soft polyvinylchloride should be chosen, and appropriate suction settings should be used. Suctioning should be performed no more frequently than indicated, because mucosal damage occurs more often in the absence of secretions. Tracheitis may occur as the result of frequent or improperly performed suctioning; it can be recognized by a persistent hacking cough and blood-streaked secretions. Tracheitis may be treated by instilling 1 mL of 1% lidocaine every 2 to 4 hours as needed and by reducing the frequency of endotracheal suctioning. [11] Using appropriate technique and vacuum settings should further minimize tracheal irritation during suctioning.

Atelectasis has been reported as a complication of suctioning and is thought to be related to the diameter of the suctioning catheter, the suction setting, and the duration of suctioning. Atelectasis can best be avoided by proper selection and use of materials for suctioning and can be treated by postprocedural hyperinflation, as described. Prevention and prompt treatment of atelectasis will reduce the chances of suction-induced hypoxemia.

Bronchoconstriction may result from mechanical airway irritation. Cessation of the

procedure, institution of positive-pressure ventilation, and use of bronchodilators as needed should bring about resolution of this complication.

Sudden death has been reported during tracheal suctioning and is probably due to one or more of the complications discussed earlier. For this reason, cardiopulmonary resuscitation equipment and resuscitative drugs should always be close at hand before the procedure is begun.

## Interpretation

Successful tracheal suctioning is evidenced by clearing of rhonchi or rales, relief of dyspnea, improvement of oxygenation

(when monitored), and removal of visible secretions. The procedure should be repeated only if indications remain.

## Nasotracheal Suctioning

Nasotracheal suctioning is an infrequently used procedure that is technically more difficult to perform than suctioning through an endotracheal tube and potentially more hazardous. [9]

### Indications and Contraindications

Nasotracheal suctioning is reserved for patients who require assistance in clearing secretions because of partial airway obstruction, impaired cough reflex, or excess secretions. Many would argue correctly that these are sufficient indications for endotracheal intubation. However, rare instances may occur in which nonintubated patients require tracheal suctioning. An example is the patient with chronic emphysema who refuses intubation but whose cough is insufficient to clear the excess secretions produced by an intercurrent bacterial bronchitis.

### Procedure

The materials used for this approach are the same as those used for tracheal suctioning, with the addition of a water-soluble lubricant for easier passage of the catheter through the nose. The patient is best positioned in the "sniffing position" as described for nasotracheal intubation.

As the catheter is advanced through the nasopharynx, airflow sounds are monitored through the unattached proximal catheter opening. The sounds increase as the distal tip approaches the glottis. Gentle, rapid advancement of the catheter during inspiration should allow the tip to pass into the trachea. Successful tracheal placement is signified by a vigorous cough and the inability of the patient to phonate much above a whisper. If the catheter instead enters the esophagus or valleculae, airflow sounds are lost, no

cough is produced, and the patient may gag or swallow.

After successful tracheal placement, the clinician should reoxygenate the patient with the catheter in place by allowing several deep breaths of 100% oxygen before suctioning. Then the catheter should be attached to the suction source at the appropriate setting, the tip advanced to about the level of the carina, and the suction port occluded and the trachea suctioned as discussed earlier, with care taken to ensure reoxygenation following the procedure.

### Complications

All of the complications discussed for endotracheal tube suctioning can occur during nasotracheal suctioning. In addition, nosebleeds, gagging, aspiration, and laryngospasm may occur, or the operator may simply be unable to direct the catheter tip into the trachea. Because of these drawbacks, nasotracheal suctioning is seldom the procedure of choice for clearance of airway secretions, nor is it an ideal method for obtaining specimens for microbiologic study, because contamination by nasopharyngeal flora is inevitable.

## Conclusion

Tracheal suctioning is an important adjunct to bronchopulmonary toilet in patients whose cough reflex is impaired, whether by an endotracheal tube or otherwise. Although easily taught, the procedure is not without the potential for serious morbidity or even mortality. Tracheal suctioning should be undertaken only when indications exist and should be performed by trained individuals with a full appreciation of its potential complications.

## TRANSTRACHEAL NEEDLE ASPIRATION

Patients with signs and symptoms of acute bronchopulmonary infection require expeditious diagnosis and treatment. Examination and culture of lower respiratory tract secretions are desirable in these patients and are mandatory in those who are critically ill. Although expectorated sputum is always contaminated somewhat with oropharyngeal flora, properly screened expectorated sputum is a sufficient specimen in most cases. Occasionally, results of sputum examination and culture are equivocal, or an adequate specimen simply cannot be obtained. In these selected cases the emergency physician may wish to use the technique of transtracheal aspiration to obtain a specimen of tracheobronchial secretions before initiation of antibiotic treatment.

Poor correlation between cultures of expectorated sputum and those of lower respiratory tract secretions has been demonstrated. [24] Recognizing the need for uncontaminated culture specimens, Pecora introduced transtracheal needle aspiration, a modification of the technique commonly used for translaryngeal anesthesia. [25] Cultures of lower respiratory tract secretions obtained by transtracheal needle aspiration are more predictive of pulmonary infection than are those obtained from expectorated washed sputa or older bronchoscopic aspiration techniques. [26] [27] Newer

bronchoscopic methods using a protected specimen brush have improved the diagnostic accuracy of the bronchoscopic technique, although it may not be available in all hospitals or in a timely manner. [28] Transtracheal aspiration specimens have been shown to be especially useful in the diagnosis of unusual pulmonary infections, including those caused by anaerobic bacteria, tuberculosis bacilli, *Aspergillus, Pneumocystis carinii,* and hospital-acquired and partially treated pneumonias. [29]

**Indications and Contraindications**

Failure to obtain adequate expectorated sputum is the primary reason for performing transtracheal needle aspiration. Many patients with pneumonia are obtunded and are unable to produce a coughed sputum specimen. Transtracheal aspiration also may be preferred in patients in whom unusual infections are suspected.

Transtracheal aspiration should not be used when a sputum specimen that is suitable for proper diagnosis can be obtained by expectoration or when bronchoscopy is indicated. Diagnostic specimens may be more safely obtained in such cases by bronchoscopy using a protected specimen brush. [28] Absolute contraindications involve patient safety. Uncooperative patients are most likely to suffer tracheal damage and bleeding. Patients with bleeding diatheses should not be considered for the procedure. Hypoxia may predispose patients to potentially dangerous cardiac dysrhythmias. In addition, the inability to identify the proper anatomic landmarks for cricothyroid puncture should preclude use of this technique.

A relative contraindication to the use of transtracheal needle aspiration is the presence of chronic bronchitis, which may yield false-positive culture results. A severe paroxysmal cough increases the likelihood of subsequent subcutaneous emphysema. Bronchoscopy may be best in that situation.

**Equipment**

Commercially available intravenous catheter sets consisting of an introducer needle, J-wire guide, and a 15- to 20-cm length, 16- to 18-ga catheter are commonly used. A large (10- to 50-mL) syringe should be used to collect the sample. Two to 5 mL of *nonbacteriostatic* normal saline in a syringe should be available. Cardiac monitoring is desirable, and resuscitative drugs should be available.

**Procedure**

The patient, family, or both should be counseled regarding the procedure, and informed consent should be obtained. The patient's ability to suppress a cough on command should be assessed. Supplemental oxygen should be administered, and an arterial $P_{O2}$ >70 mm Hg or oxygen saturation >90% should be documented before beginning the procedure. [30]

The patient is positioned as shown in Figure 5-4 (Figure Not Available) *A*, with a pillow or rolled towel placed between the shoulder blades to allow full extension of the neck.

The neck is sterilized and draped. The operator then palpates the cricothyroid membrane, and a small intradermal weal is raised with local anesthetic directly over the membrane (Fig. 5-4 (Figure Not Available) *B*).

The catheter is introduced using a guide wire technique. The introducer needle is inserted as described in Chapter 4 (Needle Cricothyrotomy) and as shown in Figure 5-4 (Figure Not Available) *C*. Air is aspirated to confirm entry into the trachea. The J wire is advanced into the trachea, the needle removed, and the catheter advanced over the wire.

As soon as the catheter is in place, a 10- to 50-mL syringe is attached to the catheter hub, and suction is applied. A small aspirate amount is sufficient for analysis. If a specimen is not forthcoming, 2 to 5 mL of sterile, nonbacteriostatic saline may be injected and suction reapplied immediately. As soon as a suitable amount of sample is obtained, the catheter is withdrawn, and direct pressure is applied to the puncture site. The patient is instructed to avoid coughing as much as possible in the next 24 hours and should not receive therapies that stimulate coughing during this period.

## Complications

The complication rate for transtracheal needle aspiration is very low. Subcutaneous emphysema is usually confined to the anterior neck; is self-limited; and may be minimized by instructing the patient to avoid strenuous coughing and by avoiding positive-pressure ventilation, when possible, for 24 hours. Mediastinal emphysema is rare and is likewise self-limited.

Sustained hemoptysis is rare (1 to 2% of cases) and is minimized by attention to needle placement and prescreening for coagulopathies, when indicated. Digital pressure over the puncture site for 5 minutes will prevent prolonged hemorrhage in most instances. Persistent intratracheal hemorrhage has caused death from asphyxia in at least one case and is treated by placement of a cuffed endotracheal tube and correction of any coagulopathies.

Cardiac arrhythmias are probably secondary to vagal stimulation and are discussed under Tracheal Suctioning. Treatment of infections at the puncture site is discussed under Tracheostomy Care, and other complications are discussed in Chapter 4 (Needle Cricothyrotomy).

## Interpretation

If properly obtained, screened, and processed, cultures of transtracheal aspirates are highly predictive of the pathogen responsible for pulmonary infection.

## Conclusion

In the infrequent instances in which tracheobronchial secretions are not obtainable from expectorated sputum and when accurate bacteriologic diagnosis is essential to care of

the patient with suspected pulmonary infection, transtracheal needle aspiration is a relatively safe, practical method for obtaining such a sample. When one adheres to the precautions and guidelines presented herein, the procedure has low morbidity, high yield, and high diagnostic accuracy.

# Chapter 6 - Noninvasive Assessment and Support of Oxygenation and Ventilation

**William E. Baker ,Richard Lanoix , Dan L. Field , Jerris R. Hedges**

Ensuring delivery of oxygen ($O_2$ ) to the cell is the primary critical action in emergency medicine. Without $O_2$ to fuel cellular energy production, the cells ultimately falter and the organism dies. Except during cardiopulmonary bypass, tissue perfusion is dependent on an adequate inspired $O_2$ content, ventilatory effort, alveolar gas exchange, blood $O_2$ carrying capacity, and cardiac output. This chapter covers emergency department (ED) assessment of spontaneous ventilation and $O_2$ delivery, noninvasive means of improving inspired $O_2$ concentration and spontaneous ventilation, and delivery of inhalant medications for the treatment of reactive airway disease.

## BACKGROUND

Respiratory illness has been poorly understood until recent times. The Talmud, the ancient law book of the Israelites, blames the etiology of asthma-like illness on a malignant spirit. Later, Celsus (A.D. 25), an encyclopedist of the late Roman period, noted a favorable prognosis for a respiratory illness if the "expectoration is white as if mucus from the nose, but unfavorable if sputum is purulent, and accompanied by fever," descriptions that are consistent with chronic bronchitis and pneumonia. Celsus recommended bleeding, purgatives, emetics, and diuretics; this therapy was less preferable perhaps to his prescription for phthisis (tuberculosis), for which he recommended a leisurely sojourn down the Nile and drinking tea and honey. [1]

The Greek word alphasigmathetamualpha signified panting and was applied generally to difficult breathing and respiratory illness; the term eventually gave rise to the word *asthma*. The earliest comprehensive distinction between asthma and other respiratory diseases came from Aretaeus of Cappadocia, who first recognized and recorded the chronic recurrent nature of the disease. [2]

In 1698, Sir John Floyer wrote the first book devoted entirely to asthma and recorded the first description of pulsus paradoxus. Atropine therapy began in England in 1802, and in 1830 John Eberle deduced that "it is highly probable, therefore, that asthma consists essentially in a peculiar irritation of the pneumogastric nerves (vagus), in consequence of which the smaller bronchial tubes and air-cells are thrown into a state of spasmodic constriction." [3]

The American Thoracic Society statement on asthma in 1962 is an often-quoted definition of the disease: "asthma is a disease characterized by an increased responsiveness of the trachea and bronchi to various stimuli and manifested by a widespread narrowing of the airways that changes in severity either spontaneously or as a result of therapy." The term *asthma* is not appropriate for bronchial narrowing, which

results solely from widespread bronchial infection; from destructive diseases of the lung, as in pulmonary emphysema; or from cardiovascular disorders.

Asthma is the most common chronic disease of childhood and among the most frequent complaint of adults, resulting in 2 million outpatient visits per year; it is also the most common cause of absence from school and work. [4] Asthma, depending on one's definition, affects between 7 and 20 million people in the U.S. and is especially prevalent among those living below the poverty level. [5] The National Center for Health Statistics estimates that asthma affects 9.7 million people in the U.S. population and chronic obstructive pulmonary diseases (COPDs) afflict up to 14 million adults, with similar economic consequences. [6] Although many investigators and studies distinguish between asthma (pure reactive airway disease) and COPDs, which include chronic bronchitis (airway inflammation with increased mucus secretion) and emphysema (airway destruction and loss of airway elasticity), clinically the distinction is blurred by the similarities in ED management. In fact, the current literature cites studies that can document little to distinguish the response of either entity to bronchodilators. [7] These diseases must, however, be separated from other causes of dyspnea and respiratory distress (Table 6-1) , many of which are associated with wheezing.

## PULMONARY FUNCTION TESTING

Airway maintenance and breathing are given primacy in the ABCs of emergency medicine. Clinical assessment always

| TABLE 6-1 -- Causes of Respiratory Failure [*] | |
|---|---|
| **Neurologic** | **Lower Airway** |
| Drug overdose | Tracheobronchitis |
| Stroke | Tracheal stenosis |
| Central hypoventilation | Bronchospasm |

| | |
|---|---|
| Guillain-Barre syndrome | |
| Head trauma | **Lung Parenchyma** |
| Poliomyelitis | Adult respiratory distress syndrome |
| Botulism | Emphysema |
| | Pneumonia |
| **Muscles and Chest Wall** | Interstitial pneumonitis |
| Myopathy | |
| Myasthenia gravis | **Heart** |
| Kyphoscoliosis | Pulmonary edema |
| Flail chest | Mitral stenosis |

| | |
|---|---|
| **Oropharynx** | |
| Foreign body | |
| Laryngospasm | |
| Tonsillar hypertrophy | |

* Disease at any level of the respiratory system, central or peripheral nervous system, bellows mechanism, or heart may cause respiratoryfailure.begins with the patient's ventilatory function. The physician notes quickly the patient's mental status, level of distress, skin color, character of effort, use of accessory muscles, presence of diaphoresis, lung sounds, and vital signs. In conjunction with this clinical overview, a brief clinical history provides the physician with sufficient information on which to initiate therapy.

Unfortunately, the physician's initial clinical impression of the patient's ventilatory status is based on imprecise subjective findings that may not detect serious illness in all patients. The ability of experienced physicians to detect compromised pulmonary function when compared with pulmonary function testing seems only moderately better than chance alone. [8] In the study by Godfrey and coworkers, the sensitivity of clinical impression did not improve when the clinicians underwent training on the common and more subtle signs of respiratory distress. [8] Patients also appear superior to physicians in predicting their own pulmonary function [9] and in assessing day-to-day variation in disease, using pulmonary function testing as the standard. [10]

Regardless of the initial clinical presentation, results of treatment in a subjectively asymptomatic patient with reactive airway disease will reach, at best, only 40 to 50% of predicted normal pulmonary function and 60 to 70% when all abnormal physical signs have resolved. [11] This potentially undetected degree of dysfunction may contribute to recrudescence. Objective measures of pulmonary dysfunction serve both to quantify results of therapy and as possible predictors of admission.

Spirometry has been used for decades by pulmonary specialists to assess airway limitation. The spirometric measurements were originally validated based on comparisons with clinical and body plethysmographic data. The terminology of pulmonary function testing was derived from the various measured subsegments of spirometry (Fig. 6-1) . More recently, inexpensive handheld electronic meters have replaced formal spirometry in many clinical settings. These devices accurately measure or calculate the peak expiratory flow rate (PEFR), forced expiratory volume in 1 second

(FEV1 ), forced vital capacity (FVC), and percent FEV1 /FVC. [12] The Wright peak flow meter (Fig. 6-2) was originally designed by Wright and McKerrow for use in their pneumoconiosis unit in 1959. Subsequently, development of compact, less complex and less expensive calibrated spring-mechanism peak flow meters have allowed for widespread use in acute care and home settings (Fig. 6-3) . These peak flow meters also have been successfully used as an adjunct in the assessment of pediatric patients. In the acute clinical situation, PEFR meter readings correlate well with formal spirometry, offer simplicity, and reduce the need for patient cooperation. [13]

**Indications and Contraindications**

In the ED, acute respiratory diseases such as asthma and chronic lung diseases make up the bulk of the situations requiring the objective assessment of ventilatory status. Emergency department pulmonary function testing with either a peak flow meter or spirometer provides objective data on pulmonary status and patient response to therapy. These tests may assist the clinician in determining patient disposition [14] and may facilitate patient transfer at the time of admission by providing an objective and reproducible measure of the patient's failure to improve. Studies have found excellent correlation between PEFR and FEV1 , [13] [15] as well as between Wright and mini-Wright meters. [16] One investigation has evaluated PEFR for aiding differentiation of congestive heart failure from chronic lung disease in the patient with moderate to severe dyspnea. [17] Although opportunities will be infrequent, physicians evaluating neuromuscular diseases affecting ventilatory function, such as the Guillain-Barre syndrome, may find these techniques useful in both initial and ongoing assessment. Near respiratory arrest may be the only true contraindication to obtaining these measurements because of limited patient cooperation and delay of immediate therapy.

**Equipment**

A *spirometer* is a tube connected to a bellows-type device having communication with a recording device. The subject breathes in and out through an orifice, causing expansion and contraction of the bellows, which in turn activates the recorder that traces a curve corresponding to the lung volume. This traditional volume method is complex and cumbersome. Handheld electronic spirometers now available use sensing devices either to translate the pressure of exhalation (e.g., Respiradyne, Fig. 6-4) or to detect the number of rotations of a small turbine (e.g., Pocket Spirometer, Micro Medical Instruments, Rochester, NY) by an optical system. Both systems are self-calibrating, take little practice to use, and can calculate PEFR, FEV1 , and FVC. Some systems, such as the transducer-based Respiradyne, also give additional calculated information such as percent FEV1 /FVC and forced mid-expiratory flow rate (FEF25-75% ). Results are displayed digitally and maintained in memory until cleared.

*Peak flow meters* are simple mechanical devices that use the force of exhalation to rotate or push a membrane-coupled measuring arm or spring-loaded piston to statically record a position of maximum flow. Several brands of peak flow meters are commercially available including the Pulmo-Graph (De Vilbiss Healthcare Inc., Somerset, Pa), Assess (HealthScan Products Inc., Cedar Grove, NJ), Wright Pocket (Ferraris Medical International, Holland, NY), and the Mini-Wright (Clement Clark

International, Columbus, Ohio). Low-flow units designed for children are available.

## Procedure

The operation of peak flow meters and electronic spirometers is similar in many ways. Disposable mouthpieces are inserted or attached. If the mouthpiece is to be reused by patients sharing a common meter (during serial evaluations), the "mouth" end should be identified with pen or tape to limit the possibility of spreading infection between patients. The electronic devices must be switched on for a 30-second self-calibration period. Both the handheld spirometers and the peak flow meters can be operated with the same respiratory maneuver: a maximal inhalation followed by a maximum forced expiration into the mouthpiece. Three attempts are standard, and the highest value is recorded, provided the best two of three readings are within 10% of each other. [18] Children as young as 3 years have consistently achieved the level of cooperation necessary to perform PEFR testing. [19]

## Interpretation

Although intraindividual peak flow variability is low, [20] variability between different brands of calibrated spring mechanism peak flow meters may be significant. Flow measurements of four brands studied at low to medium flows differed by as much as 100 L/min, [21] and accuracy waned with age of the device. [22] This information should be considered when interpreting PEFR values both clinically and in the literature. Likewise, measurement of serial PEFRs should be done on the same meter. Altitude minimally affects PEFR interpretation; readings at 1400 m underestimate PEFR by 5.3 to 6.9%. [20] As with $FEV_1$ measurement, patient size affects PEFR and is most important when interpreting readings in children. Most charts of normal PEFR in pediatric patients are based on height. As age and height are also related, some investigators have developed age-PEFR charts. [14] [23] Having a measuring tape secured to the wall alongside a chart of normal values in an accessible area enables the clinician to quickly interpret the PEFR of a child. Table 6-2 lists approximate peak flow and spirometric values for various degrees of obstruction in adults and Figure 6-5 (Figure Not Available) *A* and *B* demonstrate percentile charts of PEFR vs height in boys and girls. These charts were constructed from data obtained by Carson and coworkers in 2752 healthy children in Dublin. [23] Normal PEFRs

| TABLE 6-2 -- Approximate Values for Spirometry and Peak Flow for Various Degrees of Obstruction in Adults | | |
|---|---|---|
| | $FEV_1$ (L) | $FEV_1$ /FVC (%) | Peak Flow (L/min) |

| Normal | 4.0-6.0 | 80-90 | 550-650 (males)<br>400-500 (females) |
|--------|---------|-------|-------------------------------|
| Mild | 3.0 | 70 | 300-400 |
| Moderate | 1.6 | 50 | 200-300 |
| Severe | 0.6 | 40 | 100 |

Peak flow and spirometry values vary with height, sex, and distance above sea level; men and taller persons in general have larger flow rates. The reduced barometric pressures found at higher elevations also increase airflow.

do vary in children based on a number of variables including race, geographic location, and local environment. [23] [24] [25]

The use of PEFR measurements to predict the need for admission early during emergency department treatment found that in severely compromised adults (i.e., with initial PEFRs <16% of the predicted value), an improvement in PEFR <60 L/min after the first epinephrine treatment indicated the need for admission. [26] Similarly, an initial FEV1 of 0.6 L and a post-treatment FEV1 of 1.6 L are predictive of the need for inpatient therapy. [27] PEFR values of <100 L/min initially and <300 L/min after treatment are predictive of the need for inpatient therapy in a follow-up study. [15]

These studies have been challenged due to lack of blinding. A subsequent study found that neither the initial PEFR reading nor the response to the first bronchodilator treatment was helpful in predicting admission. [28] In fact, in this double-blind study, with strict admission criteria and with the additional use of nebulized bronchodilators, <50% of those requiring admission were detected by initial measurements. However, measurements performed later in therapy, just before release, were more accurate in predicting relapse. Similarly, in a series of 83 COPD patients, a post-treatment FEV1 <40% of predicted value identified patients requiring admission (i.e., at high risk for relapse [>30%] if sent home). [29]

As with adults, initial spirometric values are weakly associated with pediatric asthma admission decisions. [30] [31] However, children with PEFRs >60% of expected *after* treatment with nebulizer therapy can be considered in the category "admission probably

unnecessary," and those with PEFRs <40% can be considered in the category "admission probably necessary." [14]

These studies indicate that when initial flow rates are extremely poor, admission is likely, because a brief course of therapy in the ED is unlikely to provide sufficient improvement. However in patients with less severe flow rates, the decision to admit should be based on the patient's response to therapy (which is dependent in part on the form of therapy and the duration of the therapy chosen). Prediction of the need for admission based on PEFR and FEV1 values obtained shortly after presentation is largely unsuccessful because of patient response variability and the option of more prolonged and aggressive ED therapy.

Hence, in adults, initially low flow rates (PEFR <100 L/min) and spirometry values (FEV1 <1.0 L) identify sick patients with the potential need for admission or close follow-up. Patients with PEFR or FEV1 values of <40% of predicted normal values after bronchodilator therapy probably require admission. Also, one can reserve blood gas analysis for patients with poor initial flow measurements or lack of improvement in pulmonary function (or hypoxia based on oximetry) and hence reduce the expense and morbidity of evaluation for reactive airway disease. [32] [33]


# PULSE OXIMETRY

Pulse oximetry has made a significant contribution to noninvasive monitoring of oxygenation in a wide variety of clinical situations. Pulse oximetry requires no special training, is noninvasive, is inexpensive, gives continuous real-time estimates of arterial saturation in the range of 80 to 100%, and provides an early warning of diminished perfusion while avoiding the discomfort and risks of arterial puncture. As a result, it has become standard of care during procedures requiring general anesthesia, [34] and it is gaining wide acceptance in out-of-hospital care, [35] neonatal and pediatric critical care, [36] adult ED care, [37] and other clinical areas. [38] [39]

**Background**

Oximetry was first developed in Germany in 1932 by Nicolai, Kramer, and Matthes. The principle of oximetry is based on the Beer-Lambert law, which states that the concentration of an unknown solute dissolved in a solvent can be determined by light absorption. Early oximeters relied exclusively on this spectrophotometric principle, using a finger or an ear as a curette containing hemoglobin, compressing these tissues to obtain a bloodless baseline absorption, and warming them to obtain a signal from maximally arterialized tissue.

Pulse oximetry, combining the principles of optical plethysmography and spectrophotometry, was invented by Aoyagi and coworkers in 1974. [40] However, it was not until 1980 that the pulse oximeter reached modern form, consisting of a probe and an onboard computer. [41] The probe, set into a reusable clip or a disposable patch, is

made up of two photodiodes producing light at 660 nm (red) and at 900 to 940 nm (infrared), and a photodetector, which is placed across a pulsatile vascular bed such as the finger or ear (Fig. 6-6) . These particular wavelengths are used because the absorption characteristics of oxyhemoglobin and reduced hemoglobin are quite different at the two wavelengths. The majority of light is absorbed by connective tissue, skin, bone, and venous blood. The amount of light absorbed by these substances is constant with time and does not vary during the cardiac cycle. A small increase in arterial blood occurs with each heartbeat, which results in an increase in light absorption (Fig. 6-7) (Figure Not Available) . By comparing the ratio of pulsatile and baseline absorption at these two wavelengths, the ratio of oxyhemoglobin to reduced hemoglobin is calculated.

Because the pulse oximeter uses only two wavelengths of light, it can only distinguish two substances. As a result, pulse oximeters measure "functional saturation," which is the percentage of oxyhemoglobin compared to the sum of the oxyhemoglobin and reduced hemoglobin. The disadvantage of functional saturation is that the denominator does not include other hemoglobin species that may be present, such as carboxyhemoglobin and methemoglobin. The advantage is that the use of only two wavelengths in the oximeter reduces device cost, size, and weight. The CO-oximeter, one example of a commercially available in vitro oximeter and the standard by which pulse oximetry is calibrated, uses four or more wavelengths, measures the "fractional saturation," and is able to measure additional hemoglobin species.

The arterial $O_2$ saturation ($Sa_{O_2}$ ) measures the large reservoir of $O_2$ carried by hemoglobin, 20 mL of $O_2$ /100 mL of blood, compared with the arterial $O_2$ partial pressure ($Pa_{O_2}$ ), which only measures the relatively small amount of $O_2$ , approximately 0.3 mL of $O_2$ /100 mL of blood, dissolved in the plasma. The $Sa_{P_2}$ correlates well with the $Pa_{O_2}$ , but the relationship is nonlinear and is described by the oxyhemoglobin dissociation curve (Fig. 6-8) . For the hypoxic patient, small changes in $Sa_{O_2}$ represent large changes in the $Pa_{O_2}$, because these $Sa_{O_2}$ values fall on the steep portion of the curve. Conversely, measurements of $Sa_{O_2}$ are relatively insensitive at detecting significant changes in $Pa_{O_2}$ at high levels of oxygenation because these $Sa_{O_2}$ values fall on the plateau portion of the curve.

Currently available pulse oximeters are accurate and precise when saturations range from 80 to 100%. [42] [43] This range is satisfactory, because for most patients an $O_2$ saturation of 80% is as much an urgent warning as one of 67%.

**Clinical Utility**

Pulse oximetry has been suggested as a fifth vital sign. [39] By providing the physician with either continuous or frequent intermittent $Sa_{O_2}$ measurements, pulse oximetry offers a more physiologic means of assessing the adequacy of oxygenation than the arterial blood gas. The latter must be drawn from an arterial source and quickly transported under specific conditions to a machine that is often distant from the patient's bedside. With pulse oximetry, the test results are available at the bedside without an invasive arterial procedure.

All recommended uses of pulse oximetry fall into one of two broad categories. First, as a

real-time indicator of hypoxemia, continuous monitoring of pulse oximetry can be used as a warning system, as many (but not all) adverse patient events are associated with arterial desaturation. [44] The second broad use of pulse oximetry is as an end point for titration of therapeutic interventions.

Although limited data address the clinical efficacy of routine monitoring of pulse oximetry in intensive care or emergency department settings, there are studies addressing operating suite and recovery room use that allow extrapolation. [45] [46] [47] These studies reported a decreased incidence and duration of desaturation episodes, fewer adverse impact events in the recovery room, and a shortened time to discovery of hypoxia. Therefore, routine monitoring of pulse oximetry in the critically ill patient population should result in fewer episodes of severe arterial desaturation and a more rapid recognition of the adverse physiologic events that produce arterial desaturation. Patient outcomes should be improved with therapeutic interventions resulting from immediate knowledge of an unfavorable $Sa_{O2}$ . [48]

## Indications

Clinical conditions where continuous monitoring with pulse oximetry is indicated are shown in Table 6-3 . Of note regarding assessment of respiratory illness severity in children, clinically unrecognized hypoxia ($Sa_{O2}$ <92%) has been documented by pulse oximetry in preschool children with respiratory symptoms. [49] Also, an initial $Sa_{O2}$ <91% in children with bronchospasm, regardless of subsequent improvement with ED management, has been associated with increased morbidity and need for admission. [50] As a "fifth vital sign," $Sa_{O2}$ monitoring affords the opportunity to assess both oxygenation and perfusion. This screen is helpful in those patients with suspected sepsis.

## Procedure

The location for the probe is determined by the clinical situation and the probes that are available. A reusable clip-on

| **TABLE 6-3** -- Clinical Applications of Pulse Oximetry |
| --- |
| During emergency airway management |
| Assess need for further airway management |

| |
|---|
| Assess adequacy of preoxygenation before endotracheal intubation |
| Monitor ventilator and Fi $O_2$ changes |
| Provide early indicator of ventilator dysfunction |
| Assist routine weaning of $O_2$ therapy, but not mechanical ventilation |
| Oxygenation monitor |
| Monitor patients with lung dysfunction for unexpected hypoxic events |
| Monitor during procedures including during systemic sedation |
| Monitor oxygenation during interhospital and intrahospital transfer |
| Assess acute childhood asthma |
| Fifth "vital sign" |

probe makes the digits easily accessible. Little is needed in the way of preparation, and as mentioned subsequently, even nail polish may generally be left on. Other sites include the earlobe, the nasal bridge or septum, the temporal artery, and the foot or palm of an infant. Tape and splints can be used to secure the digit probe and to minimize motion.

After placement of the probe, the machine is turned on. The computer analyzes the incoming data to identify the arteriolar pulsation and displays this in beats per minute;

newer devices also display a pulse plethysmograph. Simultaneously, $O_2$ saturation is displayed on a beat-to-beat basis. Some machines have hard copy capability and can provide paper documentation of the patient's status. If the oximeter fails to detect pulsatile flow, either the reading will not be displayed or the Sa $_{O2}$ value is given with a poor signal quality warning, depending on the machine. It is valuable to evaluate serial measurements and to verify that measurements correlate with other clinical markers.

## Interpretation

Patients with good gas exchange have $O_2$ saturations between 97 and 100%. When the $Sa_{O2}$ falls below 95%, hypoxia is present, although patients with COPD may live in this range. Oxygen saturations below 90% represent relatively severe hypoxia. Children with $Sa_{O2}$ values <92% often require admission for $O_2$ and additional therapy. [49] [50] In adults and older children, as with PEFR and spirometry, a low isolated early measurement of Sa $_{O2}$ does not mandate admission because of the wide variability of response to therapy. [51] [52] However, low $Sa_{O2}$ readings should be heeded as important clinical warning signs.

Although pulse oximetry represents a significant advance in noninvasive monitoring, clinicians must recognize and understand its limitations. Pulse oximetry measures $O_2$ saturation. In comparison to arterial blood gas determination or end-tidal $CO_2$ (carbon dioxide) monitoring, which is discussed below, pulse oximetry provides no information on pH or $Pa_{CO2}$ levels, and only indirectly assesses the adequacy of ventilation. During both conscious sedation and general anesthesia, monitoring of ventilation is a more desirable goal for prevention of hypoxia. Hypoventilation and resultant hypercapnia may precede a decrease in hemoglobin $O_2$ saturation by many minutes. Furthermore, supplemental $O_2$ may mask hypoventilation and $CO_2$ retention by masking the resultant $O_2$ desaturation for which pulse oximetry monitoring is designed. Several other pulse oximeter limitations have been noted and are summarized in Table 6-4 .

### Effects of Dyshemoglobinemias

Carboxyhemoglobin results in falsely high $Sa_{O2}$ estimates of hemoglobin $O_2$ saturation. Low quantities of methemoglobin (MetHb) will reduce a pulse oximeter reading by about half the actual MetHb percentage. [53] However, even large quantities of MetHb (>10%) can result in a stable pulse oximeter reading of 85% regardless of the actual $Sa_{O2}$ . In summary, in patients with methemoglobinemia or with elevated carboxyhemoglobin levels, the reading on the pulse oximeter does not accurately depict quantitative hemoglobin $O_2$ saturation changes. [54] [55] The pulse oximeter will variably *underestimate* the percentage of abnormal hemoglobin; a CO-oximeter is required for confirmation of these conditions and quantitative analysis.

---

**TABLE 6-4** -- Factors Affecting Pulse Oximetry Readings

| |
|---|
| *Severe anemia:* Satisfactory readings obtained down to hemoglobin level of 5 mg/dL |
| *Motion-artifact:* See text regarding probe sites |
| *Dyes:* Transient effect unless methemoglobinemia results |
| *Light-artifact:* Minimize by covering probe with opaque material |
| *Hypoperfusion:* Inadequate pulse signal will be displayed |
| *Electrocautery:* Minimize by increasing the distance of the sensor from the surgical site |
| *Deep pigmentation:* Use fifth finger, earlobe, or other area of lighter pigmentation |
| *Dark nail polish:* Remove with acetone or place sensor sideways on digit |
| *Dyshemoglobinemias* (e.g., carboxyhemoglobin and methemoglobin): Falsely elevate saturation reading |
| *Elevated bilirubin:* Accurate up to bilirubin level of 20 mg/dL in adults; no problem reported for jaundiced children |
| *High saturation:* Pulse oximetry not useful for monitoring hyperoxemia in neonates |
| *Fetal hemoglobin:* No effect on pulse oximetry; falsely reduced CO-oximetry readings |

| |
|---|
| *Venous pulsations:* Artificially lower O$_2$ saturation; choose probe site above the heart |
| *Dialysis graft (A-V fistula)*: No difference from contralateral extremity unless fistula produces distal ischemia |

**Fetal Hemoglobin**

Newborn full-term infants can have up to 75% of total hemoglobin in the form of fetal hemoglobin. [56] Pulse oximeters remain accurate in the presence of fetal hemoglobin. [57] However, a CO-oximeter will erroneously interpret the carboxyhemoglobin level to be elevated, and the oxyhemoglobin level to be artificially reduced. [58] Therefore, when fetal hemoglobin levels are high, CO-oximetry readings should not be used to confirm pulse oximetric readings. It should be noted that newborns normally have up to 5% carboxyhemoglobin.

**Low Perfusion**

To function properly, pulse oximeters require a pulsating vascular bed. Hypotension with vasoconstriction, hypothermia, or the administration of vasoconstricting drugs may reduce the pulsatile component to <0.2% of the total signal. At this level, the true signal cannot be distinguished from background noise. Under these conditions, many pulse oximeters display a message indicating an inadequate pulse signal. A change in the location of the sensor to an area with higher perfusion, such as an earlobe, may improve the pulse signal. [59]

**Intravenous Dyes**

A number of dyes and pigments interfere with the accuracy of pulse oximetry. [60] [61] Methylene blue, the treatment for methemoglobinemia, absorbs light at 660 nm, similar to the absorption of reduced hemoglobin, and can artificially lower pulse oximeter saturation readings to as low as 1%. Low readings also can be seen with other intravenous dyes such as indigo carmine, indocyanine green, and fluorescein, although the rapid clearance of these agents minimizes the phenomenon. [62] [63]

**Bilirubin**

Hyperbilirubinemia does not affect the accuracy of pulse oximetry measurements. However, hyperbilirubinemia may have an effect on absorption at the lower wavelengths used by CO-oximeters, resulting in a discrepancy between pulse oximeter and

CO-oximeter readings. [64]

**Skin Pigmentation**

Pulse oximeter accuracy is somewhat reduced by deeply pigmented skin. This effect is likely due to a shift in the light-emitting diode's output spectrum as the light output is increased. This effect is small and results in only a slight decrease in accuracy over a large number of samples. [64] Placing the probe on an area of lighter pigmentation, such as the fifth finger or an earlobe, has been suggested as a means to minimize this effect.

**Nail Polish**

Conflicting data exist about the effect of nail polish on the accuracy of pulse oximeter determinations. Mounting the probe side-to-side on the finger was found to be as accurate as readings on uncovered nails. [65] This technique also circumvents the problem of only partial placement of the probe because of very long fingernails, which may cause a low $O_2$ saturation reading. An alternative solution to the problem of nail polish is to remove it with acetone.

**High Saturation**

Because the $O_2$ dissociation curve plateaus at saturation levels >90%, a large increase in $Pa_{O2}$ results in a small increase in saturation. Therefore, an error of a few percentage points could represent a large error in $Pa_{O2}$ . This is inconsequential for most adult patients but is of extreme importance for neonates at risk of retinopathy caused by hyperoxemia.

**Venous Pulsations**

Increased venous pulsations resulting from right heart failure, tricuspid regurgitation, or placement of a tourniquet or blood pressure cuff above the probe can interfere with accurate readings and lead to artificially lower $O_2$ saturations, because the pulse oximeter interprets any pulsatile measurement as arterial. [66] Placing the probe on a site above the heart may improve accuracy. Some pulse oximeters have the capability to synchronize pulsations at the probe site to electrocardiographic (ECG) signals, thus enhancing the signal-to-noise ratio.

**Anemia**

Because pulse oximeter measurements depend on light absorption by hemoglobin, they become less accurate and reliable in conditions of severe anemia. However, accuracy is not diminished until the hemoglobin content is <5 mg/dL. [67] [68]

**Ambient Light**

As the pulse oximeter's photodetector is nonspecific, high-intensity ambient light can

produce interference. Surgical, fluorescent, and heating lamps are the common sources of this interference. This problem can be corrected by wrapping the probe with a light barrier, such as a dark cloth or other opaque material. [59]

### Motion

Motion of the probe can produce considerable artifact and inaccurate pulse oximetry readings. However, this phenomenon is transient and seldom influences assessment. Correlating

a pulse oximeter signal with an ECG waveform [64] or using alternate probe sites, such as the ear or toe, also may reduce motion artifact.

### Probe Site

The finger is the most common probe site used for adult pulse oximetry. If the finger is inaccessible or unsuitable, other probe sites, such as the earlobe, nose, and forehead (using reflectance instead of transmittance), may be used. It should be noted, however, that the forehead and nasal bridge probes may be less accurate than the finger and ear probes. [64] In infants and small children, an adhesive sensor unit is preferred. Common sites of attachment include the great toe, the heel, and the lateral aspect of the foot.

### Electrocautery

Electrical interference from devices such as electrocautery also can impair the accuracy of pulse oximetry. This interference can be reduced by increasing the distance between the surgical site and the probe.

## Conclusions

Pulse oximetry is a widely available and relatively inexpensive technology that provides an easy, noninvasive, and generally reliable method to monitor oxygenation. As measurements are continuous, pulse oximetry allows for earlier detection of clinically unsuspected hypoxic episodes than does intermittent arterial blood gas analysis. Frequent measurements should lead to earlier corrective measures with the prevention of adverse consequences.

# CARBON DIOXIDE MONITORING

Capnometry is a noninvasive method used to assess the adequacy of ventilation by measuring the $CO_2$ concentration of exhaled air. With an intact pulmonary circulation, $CO_2$ is present in tracheal but not esophageal gas efflux. The absence of $CO_2$ in an endotracheal tube indicates esophageal intubation, technical malfunction, circulatory arrest, circuit disconnection, or intraluminal/extraluminal tube obstruction. The continued presence of $CO_2$ in the expired air confirms tracheal intubation. In addition, capnometry provides a measure of systemic metabolism and circulation which may be continuously

monitored. Although the measurement of $Pa_{CO_2}$ via arterial sampling is the most direct method of evaluating ventilation, it is invasive, costly, and provides only intermittent data.

Capnography, introduced into clinical practice by Smalhout and Kalenda in the mid-1970s, [69] displays the $CO_2$ measured by capnometry graphically with respect to time throughout the respiratory cycle. The capnogram is the displayed waveform of breath-to-breath variations in the expired $CO_2$ concentration, and characteristic variations in its shape are associated with specific abnormalities. [70]

## Indications

### Verification of Endotracheal Tube Placement

In an emergency situation, the clinician desires a quick and reliable method to verify correct placement of an endotracheal tube (ETT). Traditional clinical methods of evaluating ETT placement (breath sounds, chest movement, epigastric auscultation), although adequate in the majority of circumstances, are not fail-safe. [71] Although, direct cord visualization reliably assesses ETT passage into the trachea, such visualization is not always possible. Additionally, an ETT may become dislodged even after correct placement as a result of head movement or patient positioning. Numerous studies in animals and humans under controlled and emergency conditions support the use of $CO_2$ measurement in expired air as a means of ETT placement confirmation. [54] [71] [72] [73]

### Systemic ("Conscious") Sedation

Many procedures performed in the emergency department are facilitated by sedation. The use of hypnotic doses of benzodiazepines can result in respiratory depression, and opioid analgesics predictably reduce ventilatory drive and the normal response to both hypoxia and hypercarbia. Moreover, the combination of benzodiazepines and opioids substantially increases the incidence of hypoxemia and apnea as compared with the effects of either agent alone. [74]

Monitoring of ventilation, perfusion, and oxygenation increases the safety of sedation. As mentioned earlier, pulse oximetry indirectly assesses ventilation. Although hypoventilation, hypercarbia, and apnea ultimately lead to a decrease in $O_2$ saturation on pulse oximetry, hypoxia is preceded by a rise in end-tidal $CO_2$ partial pressure ($Pet_{CO_2}$). [75] [76] Hence, combining exhaled $CO_2$ monitoring with pulse oximetry should further enhance the safety of systemic sedation.

Recently, nasal cannula (side-stream) $CO_2$ analyzers have made continuous $Pet_{CO_2}$ monitoring possible during systemic sedation. Nasal cannula-derived $Pet_{CO_2}$ values correlate well with $Pa_{CO_2}$ in awake, spontaneously breathing patients and may be as accurate as those obtained from an ETT. [76] Two beneficial features of the $Pet_{CO_2}$ monitor are immediate evidence of changes in the respiratory pattern by observation of the capnographic waveform and an audible apnea alarm. This latter feature is especially valuable after completion of emergency procedures when personnel are less likely to be

at the bedside and patient stimulation is minimized.

**Cardiopulmonary Resuscitation**

The role of $Pet_{CO_2}$ for monitoring the effectiveness of cardiac compressions, estimating the potential for cardiac resuscitation, and detecting restoration of spontaneous circulation is discussed in more detail in Chapter 16 .

**Mechanical Ventilation**

Many EDs manage ventilated patients past the initial resuscitation phase while they are awaiting transfer to a critical care unit or during further diagnostic testing. Capnography can noninvasively and continuously monitor for ventilation failure (e.g., discontinuities in the ventilator circuit, $CO_2$ rebreathing) and assess changes in mechanical support, including weaning from the ventilator. [77]

## Contraindications

In the critically ill patient, capnography may not be an adequate substitute for arterial blood gas sampling during adjustments of or weaning from mechanical ventilation. In such patients, $Pet_{CO_2}$ , when used, should be correlated with $Pa_{CO_2}$ measurements via arterial blood gas analysis. That is,

$Pet_{CO_2}$ values should be used as a supplement to, rather than as a replacement for, $Pa_{CO_2}$ values.

In spontaneously breathing patients, side-stream analyzers with nasal cannula sampling techniques must be used with caution, because ambient air may be entrained, which dilutes $Pet_{CO_2}$ measurements. Moreover, the accuracy of monitoring $Pet_{CO_2}$ by this method is critically dependent on the relative placement of the sampling tube in the patient's naris or nasopharyngeal airway relative to the source of supplemental $O_2$ . The presence of mouth breathing also can dilute $Pet_{CO_2}$ values when nasal cannulas are used.

In children with fast ventilatory rates and low tidal volumes, a side-stream analyzer with a slow sampling flow rate underestimates the $Pet_{CO_2}$ . Sampling flow rates of 150 mL/min yield consistently accurate estimates of $Pa_{CO_2}$ and an acceptable capnogram in neonates, infants, and children. [78] Adult-size disposable end-tidal $CO_2$ detectors are not recommended in children who weigh less than 15 kg, because their small tidal volumes may be diluted in the large (38 mL) dead space of the device, resulting in failure to detect $CO_2$ appropriately. [72] [79]

## Equipment

Of the two quantitative methods of $CO_2$ gas analysis used in hospitals, mass spectrometry and infrared absorption spectrophotometry, only the latter is used

commonly in the intensive care unit and ED. Qualitative $CO_2$ sensitive colorimetric technology also is available. A product comparison of the many commercial capnography devices has been published elsewhere. [80]

The two types of infrared capnometers are as follows:

1. Mainstream (in-line) capnometers, which consist of a transducer that is housed in an airway connector and placed in-line with the patient's breathing circuit (Fig. 6-9) (Figure Not Available) . The transducer contains the infrared light source and photodetector, heated to approximately 40 °C to prevent condensation. Mainstream sampling allows the capnometer to directly assess the patient's respiratory circuit. Hence, gas analysis is nearly instantaneous (<500 msec). The major disadvantage is the weight associated with the airway connector attached to the proximal end of the ETT. Examples of these devices are the Nellcor Stat-CAP N60B and Ultra-CAP N6000B (Nellcor Inc., Pleasanton, Calif). The former device is a portable, battery-operated instrument that provides continuous qualitative readings. The latter device is a bedside monitor that displays both a continuous waveform and numeric information.
2. Side-stream, or diverting capnometers, actively withdraw gas from the patient's airway via a sampling tube, and the concentration of $CO_2$ is measured in the machine rather than in the ETT (Fig. 6-10) (Figure Not Available) . The major advantage of side-stream analyzers is their applicability to non-intubated patients. Their major disadvantage is a slower response time, because expired gas must be transported to the capnometer itself for analysis. Slower response time results in dispersal of the gas bolus due to convection and diffusion during transit through the sampling catheter. These factors result in an underestimation of $Pet_{CO_2}$ , particularly in children who have high ventilatory rates and small tidal volumes. Response times of side-stream analyzers are dependent on the aspiration flow rate, sampling tube length, and tubing diameter. To achieve predictable $Pet_{CO_2}$ values and $CO_2$ waveforms, the response time of the analyzer must be less than the respiratory cycle of the patient. [81] The optimal gas sampling flow rate is considered to be 50 to 200 mL/min, which ensures that the capnographs are reliable in both children and adults. [82] Despite a water trap, side-stream analyzers also are affected by secretions, saliva, and water condensation.

Qualitative, lightweight, disposable end-tidal $CO_2$ detection devices are based on colorimetric changes. A pH-sensitive chemical indicator is enclosed in a plastic housing and is connected to the gas stream between the ETT and the ventilator. The concentration of $CO_2$ in ambient air or the esophagus is <0.3%. These devices change color when the concentration of $CO_2$ is higher than 4% or 5.4%. The color varies as the $CO_2$ level increases and decreases between expiration and inspiration. The devices are designed to function for approximately 2 hours. The response time of the device is sufficiently fast to detect changes of $CO_2$ breath by breath. [83] In addition to their portability and single-patient use, these devices have the advantage of not requiring electrical power. Originally, the disposable $CO_2$ detectors were only available for attachment to ETTs (EASY-CAP and Pedi-CAP, Nellcor Inc., Pleasanton, Calif). Recently, chemical $CO_2$ detection devices, built into disposable bag-valve ventilation devices, are available for emergency department use (CAPNO-FLO, Kirk Specialty

Systems, Carrollton, Tex). Both systems have components for adult and pediatric patients.

## Procedure

For mainstream capnometers, simply place the airway connector in-line between the ETT and respiratory circuit. For side-stream or diverting capnometers, position the sampling tube in the naris via nasal cannula, nasopharyngeal tube, or other adapters available with particular machines. After sufficient warm-up time, a continuous recording of the capnogram or a digital display of a $Pet_{CO_2}$ value will be displayed depending on the type of machine used. The chemical $CO_2$ detection devices are ready to use after attachment to the ETT (EASY-CAP, Pedi-CAP), or they come as a component of the bag-valve device (CAPNO-FLO).

## Interpretation

Various factors result in either increased or decreased (or even absent) $Pet_{CO_2}$ (Table 6-5) . With regards to the measurement device, a continuous recording of the capnogram is more advantageous than simply a digital display of a $Pet_{CO_2}$ value.

A normal capnogram has four phases (Fig. 6-11) (Figure Not Available) . At the onset of exhalation (point B) respiratory gases are free of $CO_2$ , because they represent the anatomic dead space. The steep rising slope of segment B-C represents the emptying of rapidly exchanging alveoli. Segment C-D represents the emptying of uniformly ventilated alveoli, where the concentration of exhaled $CO_2$ is nearly constant. True end-tidal $CO_2$ ($Pet_{CO_2}$ ) is the highest concentration measured during the alveolar plateau (point D) and immediately precedes the onset of the next inspiratory cycle. Inspiration is represented by the rapidly descending limb of the capnogram (segment D-E) and, unless significant $CO_2$ rebreathing occurs, return of the $CO_2$ concentration to near zero (baseline).

At the end of exhalation, the composition of expired air closely reflects that of alveolar air. Under normal conditions, the alveolar $CO_2$ reflects the $CO_2$ present in mixed venous blood ($Pv_{CO_2}$ ), which equilibrates with the resultant value for arterial blood ($Pa_{CO_2}$ ). Under normal conditions, only a small gradient (<6 mm Hg) exists between $Pa_{CO_2}$ and $Pet_{CO_2}$ . [84] The $P(a-et)_{CO_2}$ gradient is increased with age, COPD, pulmonary embolism, decreasing cardiac output, hypovolemia, and anesthesia itself. [85] [86] [87]

The principal determinant of the size of the $Pa_{CO_2}$ -$Pet_{CO_2}$ gradient ($P[a-et]_{CO_2}$ ) is the matching of ventilation and perfusion (V/Q). The major cause of V/Q mismatch is dead space ventilation, in which lung units have more ventilation than perfusion. As a result, the end-tidal gas contains less $CO_2$ , and $Pet_{CO_2}$ values are lower, causing large differences in the $Pa_{CO_2}$ -$Pet_{CO_2}$ gradient. [87] [88] [89] In this situation, the alveolar plateau (see Fig. 6-11 (Figure Not Available) , segment C-D) will not be flat, but rather will have a slow, rising expiratory upstroke (Fig. 6-12) (Figure Not Available) . In a normal patient, the alveolar plateau is flat because the expired gas from all lung units have similar V/Q relationships. [86] If the patient has significant maldistribution of ventilation, exhaled gas will have varying concentrations of $CO_2$ , because the $CO_2$ concentration from some

airways will be low, representing dead space ventilation, whereas the $CO_2$ concentration from others will be high. Only when a distinct alveolar plateau is present can it be assumed that true alveolar gas is being sampled and analyzed. [77]

**Intubation Assessment**

False-positive capnographic tracings associated with esophageal intubation that closely mimic successful ETT intubation may occur in patients who have recently ingested carbonated beverages or antacids and those receiving mask ventilation with resultant gastric insufflation of $CO_2$ -containing gases. [90] However, after 3 to 6 esophageal ventilations, $Pet_{CO_2}$ concentration rapidly declines as gastric $CO_2$ is washed out and

| TABLE 6-5 -- Various Factors That Influence $Pet_{CO_2}$ | | | | |
|---|---|---|---|---|
| **Effect on Pet $_{CO_2}$** | **Factor** | | | |
| | **$CO_2$ production** | **Pulmonary perfusion** | **Alveolar ventilation** | **Technical/mechanical errors** |
| Increased | Fever | High cardiac output | Hypoventilation | Exhausted $CO_2$ absorber |
| | Thyrotoxicosis | Hypertension | Bronchial intubation | Inadequate fresh gas flow |
| | Malignant hyperthermia | | Partial airway obstruction | Leaks in breathing system |
| | Sodium bicarbonate | | Rebreathing | Faulty ventilator |
| | | | | Faulty valves |

| Decreased or absent | Hypothermia | Low cardiac output | Hyperventilation | Circuit disconnection |
|---|---|---|---|---|
| | | Hypotension | Apnea | Sampling tube leak |
| | | Hypovolemia | Total airway obstruction | Malfunction of ventilator |
| | | Pulmonary embolism | Partial airway obstruction | |
| | | Cardiac arrest | Tracheal extubation | |

diluted with fresh gas (Fig. 6-13) (Figure Not Available) . [73][91]Tracheal ventilation will of course continue to produce a normal waveform.

After initial successful ETT placement and confirmation, changes in the $CO_2$ waveform can provide immediate detection of tube-related adverse events. When the ETT is partially obstructed, the capnogram will have a slow upstroke without a significant change in the $Pet_{CO_2}$ (Fig. 6-14) (Figure Not Available) . [73] [77] Bronchospasm and COPD exacerbations produce obstruction to expiratory gas flow, thus increasing the slope of the expiratory upstroke. With inadvertent extubation, the capnogram shows a sudden drop in $Pet_{CO_2}$ (the same change that occurs when the tube becomes completely obstructed or malpositioned in the esophagus) (Figs. 6-15 (Figure Not Available) and 6-16) (Figure Not Available) . [73] ETT cuff leaks yield low $Pet_{CO_2}$ values because of the partial escape of the expired gas around the tube.

**Monitoring Ventilation**

Capnography is useful in diagnosing and monitoring maldistribution of ventilation during mechanical ventilation. In patients with a large V/Q mismatch, the capnogram will have a continued rise throughout the expiratory phase (see Fig. 6-12) (Figure Not Available) and will lose the normal alveolar plateau. When this waveform occurs, the measured end-tidal $CO_2$ may not represent a good estimate of $Pa_{CO_2}$ . However, by monitoring the slope of the expiratory phase on the capnogram, the efficacy of therapy can be evaluated. For example, in the case of asthma, as bronchospasm resolves, the slope of the expiratory phase will become less steep and the alveolar plateau better defined as a

result of improved distribution of ventilation. [77]

Furthermore, as the P(a-et)$_{CO_2}$ gradient is sensitive to increases in dead space, this gradient can be used to evaluate changes and trends in dead space ventilation in critically ill patients with significant intrapulmonary shunting. [92] [93] It also can be used as a guide to determine the best positive end-expiratory pressure (PEEP) level for optimal oxygenation. Because a low P(a-et)$_{CO_2}$ gradient implies the optimal V/Q relationship, the level of PEEP that produces the lowest P(a-et)$_{CO_2}$ gradient is the level at which optimal alveolar recruitment occurs. [94]

Excluding ventilator setting changes and circuit discontinuities, a sudden drop in end-tidal $CO_2$ may be an early indicator of shock or pulmonary embolism. [69] [95]

A dip in the plateau (curare cleft) (Fig. 6-17) (Figure Not Available) indicates a spontaneous respiratory effort during mechanical ventilation [96] [97] and may serve as an early clue to the waning of the effects of a paralytic agent. Capnometry may also be especially useful in assessing the adequacy of hyperventilation in the hemodynamically stable patient with head trauma. Other capnography patterns and their clinical significance are demonstrated in Figure 6-18 (Figure Not Available) .

## OXYGEN THERAPY

Earlier sections have dealt with ventilation and assessment of oxygenation. Delivery of $O_2$ is impaired in many clinical situations. Neurologic dysfunction is an early sign of hypoxemia. Otherwise-healthy individuals begin to experience short-term memory loss, euphoria, and impaired judgment when the Pa$_{O_2}$ level approaches 55 mm Hg. Progressive loss of cognitive and motor function, increasing tachycardia, and other physical signs occur when values are between 30 and 55 mm Hg. When levels are <30 mm Hg, the patient loses consciousness. Pa$_{O_2}$ is a function of the fraction of inspired $O_2$ tension (Fi$_{O_2}$ ), the alveolar ventilation, and the relative distribution of both ventilation and perfusion in the lung. Supplemental $O_2$ can increase the amount of $O_2$ dissolved in blood enough to deliver one third of the body's resting metabolic requirements. This section reviews the noninvasive delivery of supplemental $O_2$ .

### Indications

Oxygen therapy is generally indicated when hypoxia is present (Pa$_{O_2}$ <60 mm Hg or Sa$_{O_2}$ <90 to 95%). Clinical situationsthat are commonly associated with hypoxia and generally benefit from supplemental $O_2$ include pulmonary disease, cerebrovascular accidents, gastrointestinal bleeding, shock, and trauma. Supplemental $O_2$ also is indicated when hypoxia is anticipated from a procedure such as intubation or before a procedure in which intravenous analgesia may cause respiratory depression.

High altitude also results in hypoxia. At 7000 ft the barometric pressure is 564 mm Hg, the atmospheric partial pressure of $O_2$ is 116 torr, and the arterial partial pressure of $O_2$

(with healthy lungs) is approximately 63 torr. Commercial airlines maintain cabin pressures at levels between 5000 to 8000 ft at most cruising altitudes, and close to 10,000 ft temporarily under certain circumstances. Patients with chronic hypoxia may require supplemental $O_2$ , and they must contact the airline before flying to make appropriate arrangements (the reader is referred elsewhere for a review of air travel and $O_2$ therapy). [98] Oxygen is specifically indicated for carboxyhemoglobinemia; 100% $O_2$ reduces the half-life of carboxyhemoglobin from a mean of 5 hours to 1 hour; hyperbaric (more than 1.0 atm) $O_2$ can lessen this time by half again. However, discussion of hyperbaric $O_2$ therapy is beyond the scope of this chapter.

**Contraindications**

Paraquat turns $O_2$ into free radicals, thus making $O_2$ a substrate of the poison. Similar toxins in this group include bleomycin, cyclophosphamide, doxorubicin, ozone, and nitrous dioxide. [99] [100] Hence, $O_2$ therapy in the absence of documented hypoxia is contraindicated for this group of toxins.

Fears of ventilatory shutdown in the presence of $CO_2$ retention have made physicians hesitant to give supplemental $O_2$ in amounts greater than 2 L/min. However, if hypoxia remains after low-flow $O_2$ is initiated, the $O_2$ delivery must be increased. Continuous monitoring of spontaneous respiration is required in this setting, and preparations to perform intubation should be made to assist the patient's ventilation if indicated. Note that inspired $O_2$ levels may actually rise with low-flow $O_2$ therapy as respiratory effort wanes, because less room air is entrained with shallow breaths; significant exacerbation of ventilatory failure has been documented with even 1 L/min of $O_2$ by nasal cannula. [101] However, a high $CO_2$ level is not a contraindication to appropriate $O_2$ therapy.

**Equipment**

The two most common sources of $O_2$ for the patient in the hospital are wall outlets linked to a large reservoir of liquid $O_2$ and $O_2$ tanks (green by convention) of various sizes, listed in Table 6-6 . Both provide outlet pressures in the range of 30 to 50 psi. An approximation of the amount of time left in a given tank at a given flow rate may be calculated using the following formula:

Minutes of $O_2$ = gauge pressure (psi) × cylinder factor/flow rate (L/min)

Cylinder factors are shown in Table 6-6 . Alternatively, a simple computer program is available that can estimate the duration of $O_2$ remaining in a tank based on the previous

**TABLE 6-6** -- Oxygen Cylinders (Approximate Values)

| Cylinder Type | Volume (L) | Cylinder Factor | Hours of $O_2$ at 2 L/min |
|---|---|---|---|
| D | 450 | 0.20 | 3.5 |
| E | 650 | 0.28 | 5.0 |
| G | 5600 | 2.41 | 44.0 |
| H, K | 6900 | 3.14 | 58.0 |

variables. [102] The numerous methods available to deliver $O_2$ from the source to the patient are listed in Table 6-7 .

Nasal cannulas are capable of providing low-flow $O_2$ supplementation when accurate control of $O_2$ concentration is unimportant, as inspired concentrations vary considerably depending on patient tidal volume and inspiratory flow rate. [103] Rough guidelines for inspired $O_2$ concentration related to $O_2$ flow rates are listed in Table 6-7 . Similarly, some mask systems are designed to be used without Venturi inserts, and package inserts indicate the range of inspired $O_2$ concentration based on $O_2$ flow rates. As with nasal cannulas, inspired $O_2$ concentrations vary considerably (may be upward of 10% greater than predicted by package inserts) depending on the patient's breathing pattern (rate and tidal volume).

Venturi systems should be used in patients with $CO_2$ retention for whom tight control of inspired oxygen is required. A partial non-rebreathing mask incorporates a 200 mL reservoir that effectively delivers an inspired $Fi_{O_2}$ of 60

**TABLE 6-7** -- Oxygen Delivery Systems

| System | Flow Rate L/min | FIo$_2$ (% Oxygen) | Advantages | Disadvantages |
|---|---|---|---|---|
| Nasal cannulas | 1<br>2<br>3<br>4<br>5<br>6 | 25<br>29<br>33<br>37<br>41<br>45 | Simple, comfortable, inexpensive, allow eating and drinking | Can cause local irritation and drying of mucous membranes (primarily for flow rates >4 L/min) |
| Simple oxygen masks | >5 L/min | 35-50 | Deliver higher flow rates than nasal prongs | Must be removed for airway care, eating, and drinking |
| Masks with reservoir bag | Up to 15 L/min | 60 | Higher FIo$_2$ at lower flow rates | Risk of atelectasis and oxygen toxicity (with prolonged use) |
| Venturi masks | 4<br>6<br>8<br>10 | 24-28<br>31<br>35-40<br>50 | More precise control of final oxygen concentration | Same as above |
| Reservoir nebulizers, includes CPAP, T-tubes | 40 L/min plus: | 60-90 | Can deliver increased humidity, positive pressure | Risk of barotrauma |

to 80% in most patients. The system uses a 200 mL reservoir bag, initially filled with $O_2$. Inspiration at high flow rates generally empties the reservoir and exhalation fills it with the first 200 mL of expired gas, which is mostly derived from the dead space of the upper airway and thus contains a high FiO2. A true non-rebreathing mask contains two types of valves and is capable of delivering close to 100% $O_2$. One valve prevents exhalation gas from entering the reservoir bag. The second type (actually two valves),

are located on the mask and allow exhalation, concurrently preventing ambient air from entering the inhalation circuit. To comply with safety regulations designed to prevent asphyxiation should the $O_2$ supply be interrupted, many manufacturers remove one of the valves located on the mask, somewhat limiting the concentration of $O_2$ delivered.

## Procedure

Supplemental $O_2$ should be given to increase the $Pa_{O2}$ to between 60 and 80 mm Hg or between 90 and 95% $Sa_{O2}$. In attempting to deliver high concentrations of $O_2$ to a non-intubated patient, the challenge is to provide enough $O_2$ at flow rates sufficient to meet the patient's demands. A simple method of assessing the adequacy of $O_2$ delivery with a medium flow system is to add water mist to the $O_2$ (through a humidifier) and to visually observe the pattern of mist flow at the mask. Mist should escape from the side holes of the mask during both inspiration and expiration. If mist is not visualized to escape during inspiration, ambient air is being inspired by the patient (effectively reducing inspired $O_2$ concentration) and a higher flow system is required. Bedside pulse oximetry can provide prompt monitoring of changes in $Sa_{O2}$, allowing adjustments to be made without repeated arterial blood gas determination. Pulse oximetry values generally equilibrate within 5 minutes of an oxygen delivery adjustment. [104]

Evidence of $CO_2$ retention, such as decreasing mental status and failing respiratory drive, indicates the need for an arterial blood gas determination to document the $Pa_{CO2}$; however, the decision to intubate generally should be made on clinical grounds. The need to humidify bedside low-flow $O_2$ is unproven. [100] [105] Providing $O_2$ from cylinders involves several steps that are outlined in Table 6-8 .

## Complications

Three distinct areas of risk accompany supplemental $O_2$ use: (1) respiratory dysfunction, (2) cytotoxic injury, and (3) physical hazards. Respiratory dysfunction results from $CO_2$ retention and atelectasis. Variations in $CO_2$ level provide the main stimulus to breathe in normal subjects. Patients with COPD have a decreased sensitivity to $CO_2$ levels secondary to chronic exposure to higher $CO_2$ levels, and hypoxia provides backup support for the respiratory drive. Oxygen given in sufficient amounts can remove the remaining chemical stimulus to respiration and has the potential to cause respiratory shutdown.

| TABLE 6-8 -- Operation of Oxygen Cylinders |
| --- |
| 1. Secure the tank in an upright position so that it will not move or fall while being manipulated. |

| |
|---|
| 2. Remove the cylinder seal ("E" tank) or cylinder cap ("G," "H" tank). |
| 3. Turn the cylinder valve on and off quickly to clear ("crack") the valve. On the "E" tank, this is done with a wrench; on the "G" or "H" tank, it is done with the cylinder handle. |
| 4. Check the yoke to ensure that it is compatible for use with oxygen, and place it on the cylinder, being sure that the fittings are compatible. |
| 5. Tighten the yoke, making certain that any necessary gasket is in place. |
| 6. Close the needle valve. |
| 7. Slowly open the cylinder valve until the pressure maximizes. |
| 8. Observe that the cylinder contains an adequate supply of gas. |
| 9. Connect the desired secondary delivery system (e.g., nasal cannula). |
| 10. Open the needle valve so that the desired oxygen flow registers on the flow meters. |

The complete abolition of the hypoxic drive has been reported to require a $Pa_{O_2}$ of 200 mm Hg. However, administration of 100% $O_2$ to 22 patients with COPD in acute respiratory distress (mean baseline blood gas values: $Pa_{CO_2}$ = 65 mm Hg, $Pa_{O_2}$ = 38 mm Hg) for 15 minutes resulted in only a transient decrease in minute ventilation. [106] The lowest values for minute ventilation were reached between 20 and 180 seconds from onset of inhalation, followed by a slow rise over the next 12 minutes to within 93% of the control value. The decrease was the result of falls in both tidal volume and

respiratory rate; however, after 15 minutes, these parameters had returned to baseline levels. Despite little difference in these parameters at the 15 minute point, $Pa_{CO_2}$ had risen a mean of 23 mm Hg, and $Pa_{O_2}$ had risen a mean of 225 mm Hg. [107] The mechanism leading to atelectasis is less clear. Elevated $O_2$ levels may affect underperfused or underventilated pulmonary segments by decreasing hypoxic vasoconstriction. The increased perfusion could lead to greater absorption of the remaining gas, destabilizing the alveolar units and bringing on collapse. [108]

Cytotoxic damage, theoretically secondary to free radical production, leads to tracheobronchitis and adult respiratory distress syndrome (ARDS) manifested by pulmonary edema and focal lung collapse with pulmonary fibrosis as a long-term consequence. The mechanism of damage has been shown experimentally to include oxidation of carbohydrates with disruption of cell surface receptors, DNA-RNA alterations, lipid peroxidation, and protein denaturation. Administration of an inappropriately high level of $O_2$ does not usually produce consequences within the time frame of the emergency department visit. The risk of $O_2$ toxicity depends on several factors, including $O_2$ tolerance (the state of biologic resistance to $O_2$ -induced damage, which is itself dependent on antioxidant defenses, age, and nutritional and hormonal factors), concentration of $O_2$ delivered, and duration of treatment. The goal is to deliver the least $O_2$ required to achieve adequate tissue levels. During resuscitation and most emergency care, 100% $O_2$ can be delivered safely to most patients without fear of cytotoxic injury. Healthy adult volunteers have received 100% $O_2$ for up to 6 hours without evidence of pulmonary injury. Except in special circumstances, such as paraquat poisoning, $O_2$ at concentrations of 50% is safe for 2 to 7 days. [100]

Physical risks associated with $O_2$ therapy include trauma associated with tank explosions, fire hazard, local irritation, and drying of mucous membranes.

## NONINVASIVE PRESSURE SUPPORT VENTILATION

Noninvasive pressure support ventilation can be used to deliver increased airway pressure in several modes: (1) pressure increases solely during inspiration (i.e., delivery of inspiratory positive airway pressure [IPAP]) to supplement ventilatory mechanics; (2) continuous steady positive airway pressure maintained throughout the ventilation cycle (i.e., expiratory positive airway pressure [EPAP], also known as continuous positive airway pressure [CPAP]) to improve alveolar oxygen exchange; or (3) a combination of both modalities to achieve both goals. During noninvasive pressure support ventilation, a tight, well-fitting mask is placed over the patient's mouth and nose or just over the nose.

Mask CPAP treatment of cardiogenic pulmonary edema was first described more than 50 years ago [109] and has been shown to be a useful adjunct in the treatment of acute cardiogenic pulmonary edema. Mask CPAP results in early physiologic improvement and reduces the need for intubation and mechanical ventilation in patients with pulmonary edema. [110] [111] [112] It also reduces total hospital costs for patients with severe cardiogenic pulmonary edema. [113] Use of mask CPAP to deliver PEEP has

aided the treatment of other forms of respiratory failure including those due to pulmonary infections, trauma, and obstructive lung disease. Patients with respiratory failure secondary to *Pneumocystis carinii* pneumonia who require mechanical ventilation have in-hospital mortality rates as high as 86 to 94%; however, those who were judged to require mechanical ventilation, but who were instead treated with mask CPAP, have in-hospital mortality rates of 22 to 55%. [114] [115] [116] Mask CPAP may be efficacious in the treatment of respiratory failure secondary to other pulmonary infections. [117] [118] Further, mask CPAP (either by full-face or nasal mask) may obviate the need for intubation and mechanical ventilation in acute exacerbations of COPD. [119] [120] [121]

The general use of IPAP for respiratory failure has recently been reviewed. [122] The use of combined IPAP and EPAP in the ED and critical care setting appears promising. [123] [124] [125] [126] This technique is also known as bi-level positive pressure ventilation, because it allows the clinician to deliver different inspiratory and expiratory pressures. This technology offers the opportunity to both support mechanical ventilation and to improve oxygen exchange through an enhanced functional residual capacity. In essence, a mask device provides the ventilation and PEEP that was once possible only with the use of tracheal intubation and a standard ventilator.

**Indications and Contraindications**

Noninvasive pressure support ventilation is indicated for the treatment of impending ventilatory failure in an attempt to avoid intubation and standard mechanical ventilation, with their associated morbidity and mortality. Noninvasive pressure support ventilation seems best applied to patients whose respiratory failure is expected to quickly respond to medical therapy, as continuous long-term mask CPAP or ventilation requires close attention.

Noninvasive pressure support ventilation for acute respiratory failure requires an alert patient capable of protecting the airway and handling secretions. Other contraindications include an inability to obtain a good mask fit, cutaneous irritation from the mask, and inability to cooperate with the therapy. Finally, intubation and standard ventilation is preferred for patients who require total ventilatory support, because the mask may slip and effective ventilation may cease.

**Equipment**

Although a number of systems that provide CPAP are on the market, this technique is infrequently used in the ED. A small, noninvasive bi-level positive pressure ventilation system (BiPAP System, Respironics Inc., Murrysville, Pa) that permits use of a nasal (rather than facial) mask alone *in lieu* of tracheal intubation may be most useful in the emergency department. [126] Therefore, an overview of the use of the BiPAP System is given below. The advantage of the BiPAP System is that it supplies air or $O_2$ at a pressure and flow rate that are suitable for assisted *inhalation and expiration* by sensing the patient's spontaneous breathing efforts and automatically adjusting to compensate for variations in ventilatory requirements, even in the presence of airway leaks. *Importantly, this device is not intended to be a life support ventilator, because it is used only to temporarily augment spontaneous breathing.* Regardless of the system used,

close attention to manufacturer guidelines is advised.

**Procedure**

Prior to initiation of this technique, the patient must be informed of the purpose of the nasal mask and cooperation ensured. It helps to inform the patient of the alternative therapy should this technique not meet the patient's needs. It also helps to reassure the patient that the operator will stay with the patient until he or she is comfortable with the mask and ventilator system. Baseline vital sign and oxygenation measurements are made.

*The patient should be treated in a closely monitored setting where his or her vital signs and respiratory status are closely monitored.* Despite intervention with this system, emergent intubation and mechanical ventilation may become needed. Equipment for airway support (e.g., $O_2$ source, tubing, bag-valve mask, laryngoscope, suction equipment, and various airways) should be immediately available.

The BiPAP System components are assembled and connected to oxygen--generally at the same flow settings as the patient is currently receiving (generally at 10 to 15 L/min). The pressure tubing is attached to the airway pressure monitor and the pressure monitor is turned on; the main system is then turned on and a ventilation mode is selected. Generally, the system will be used in the spontaneous ventilation mode (i.e., to support spontaneous ventilations) with an initial EPAP setting of 3 to 5 cm $H_2O$ and an IPAP setting of 8 to 10 cm $H_2O$.

Ideally, a mask sizing gauge (Fig. 6-19) (Figure Not Available) is used to choose a mask size that does not place direct pressure on the bridge of the nose, the lateral *ala nasi*, the inferior nasal septum, or the lip. The mask may be initially held in place by the operator as the patient adjusts to the ventilatory support. With the mask in place, the BiPAP System settings are modified to optimize patient ventilatory status (Fig. 6-20) (Figure Not Available) . As the patient becomes more comfortable, the mask is secured in place on the face using self-adhesive binding straps (Fig. 6-21) (Figure Not Available) . If time permits, the headgear straps may be loosely attached prior to placement and the mask slipped over the head as a unit; the straps are then tightened.

The patient is encouraged to breathe with the mouth closed. The facial mask fit should be adjusted for comfort and to minimize air leak, especially about the eyes. When the patient has accepted the mask and the clinical status has stabilized, the patient may be permitted to speak and even to eat small amounts.

Adjustments in the EPAP and IPAP settings are generally made in 2 cm $H_2O$ increments. Near optimal settings can generally be attained within 10 minutes. Periodic blood gas measurements should be coupled with ongoing vital sign and pulse oximetry measurements. When available, a $CO_2$ device side-flow catheter can be placed under the nasal mask for monitoring exhaled $CO_2$ levels. Increases in IPAP settings generally increase tidal volume and decrease $CO_2$ levels, whereas increases in EPAP generally increase functional residual capacity and increase $O_2$ levels. The concentration of inspired oxygen also can be modified by changing the $O_2$ flow rate. High levels of EPAP

or IPAP can induce PEEP-related reductions in cardiac preload. Further, although the system can adjust for some air leak about the mask, higher pressures require a tighter mask fit and can increase patient discomfort.

If the patient's hypoxic drive is diminished, decreased spontaneous ventilatory effort may be noted. If this situation occurs, the BiPAP System should be changed to the "spontaneous/timed" mode, which permits spontaneous breaths but imposes a mandatory ventilation if an extended ventilatory pause is noted. When initiating this mode, the clinician generally sets the respiratory rate at 10 breaths/min. At this setting, when a breath does not occur within 6 seconds of the preceding breath, a mandatory breath is provided.

If the patient requires a brief period of hyperventilation to help coordinate ventilatory efforts, the BiPAP System can be set to the "timed" mode and a respiratory rate of 15 to 20 breaths/min can be initiated. In this mode, the %IPAP time must be set by the operator. A %IPAP of 30% produces an inspiratory-to-expiratory (I:E) ratio of 1:2.3, whereas a %IPAP of 40% produces an I:E ratio of 1:1.5.

Aerosolized medications can be delivered either through in-line "T-pieces" in the BiPAP System circuit or through standard mouthpiece units (see following section).

**Complications**

Complications of these forms of therapy include facial irritation, abrasion, or even facial necrosis; conjunctivitis due to mask air leak; aspiration; and gastric distention. A wound care dressing on the bridge of the nose may reduce skin abrasion. [125] Nasogastric tubes have been used to relieve gastric distention, although a nasal mask system is less likely to produce gastric distention. Additionally, although the pressures used are generally low, all of the complications of positive pressure ventilation may be seen with this technique.

**Conclusions**

Positive pressure ventilation modalities offer the promise of averting the need for intubation and mechanical ventilation in certain groups of patients with acute respiratory failure. The procedure for instituting these therapies is relatively simple and may even have potential in the out-of-hospital setting. Additionally, although best studied in adults, these therapies have been applied to pediatric patients, [127] [128] and pediatric masks are available commercially.


# DELIVERY SYSTEMS FOR INHALED MEDICATIONS

Inhaled substances have been used for medicinal (and social) purposes since the beginnings of recorded history. Among the earliest of such records on the treatment of respiratory disease comes from a monograph written in 2737 B.C. by the Chinese

emperor Shen Nung on the use of inhaled cannabis for the treatment of asthma. [129] Later in the 15th century B.C. the Chinese *Pharmacopeia* refers to cannabis and to *ma huang*, a medicinal that contained ephedrine, as therapy for asthma.

Through the 1900s, atropinic effects were obtained from ingestion and inhalation of medicinals such as *Atropa belladonna* and *Datura stramonium*. Thus, long before the era of modern medicine, beta-adrenergic and antimuscarinic stimulation by inhalation were established in the physician's armamentarium.

When compared with oral or parenteral delivery, the aerosolized delivery of certain medications to patients suffering from bronchospasm has been found to be particularly effective. With fewer side effects and better patient acceptance, aerosolization has become the route of choice in contemporary clinical practice. Aerosolized medications are easily delivered using portable self-contained sprays called metered-dose inhalers (MDIs) and by nebulizer devices propelled by air compressors or wall $O_2$ . Either method can be used in the home, outpatient department, or in-hospital setting. Both modalities also can be delivered in-line with ventilation equipment for intubated patients and by mask for young or weak patients. However, the use of MDIs in intubated patients has not been well defined, and the modality's efficacy remains unproven. [130]

Intermittent positive-pressure breathing (IPPB) as a means of assisting simple nebulizer therapy has received much criticism and is no longer commonly used. Some delivery systems used inhaled dry medication (Ventolin Rotahaler and Bricanyl Turbuhaler). These systems are inspiratory flow driven and eliminate the coordination problems encountered with MDIs. Additionally, the Bricanyl Turbuhaler contains no propellants or lubricants and may reduce the incidence of allergy to the adjuvants commonly found in MDIs. Though they have been used extensively outside the United States, their role in the management of bronchospasm in the acute care setting is uncertain. Hence, these systems are not discussed further in this chapter.

**Indications and Contraindications**

Nebulizers and MDIs deliver medications by the pulmonary route for the treatment of bronchospasm. Metered-dose inhalers also are used to deliver steroids, mucolytics, and ergotamine tartrate for migraine headache. [131] Gentamycin and other antibiotic solutions have been nebulized for antimicrobial therapy. Aerosolized racemic epinephrine is employed to treat croup and aerosolized pentamidine has been developed for the treatment of *Pneumocystis* pneumonia.

Spacer devices permit effective drug delivery with MDIs even in patients with poor hand-inhalation coordination. [132] In the setting of effective pulmonary drug delivery, inhalant therapy is preferred over the intravenous or subcutaneous routes for reasons of convenience, safety, and patient comfort.

Historically, the MDI has been used primarily in the home and nebulizers primarily in the inpatient and outpatient clinical settings; however, this division is becoming less distinct as more patients obtain compressor-driven devices for home nebulizer use and the MDI is restudied for use in the hospital setting. An MDI combined with a spacer increases

efficacy, and recent studies show no detectable difference in effectiveness of albuterol administered by nebulizer or an inhaler system consisting of an MDI combined with a spacer. [133] [134] The same findings have been reproduced in the treatment of children ages 6 to 14 years, [135] and MDIs (combined with spacers) have been successfully used for treating infants (ages 1 to 18 months) with wheezing. [136] Despite these convincing studies, nebulization remains the more widespread technique for beta-agonist delivery in both out-of-hospital and in-hospital settings.

The benefits of replacing nebulizers with MDIs in the hospital setting include decreased costs, quicker treatments, and better patient education in anticipation of later home use. Less nursing and respiratory technician time are required with MDI therapy. [137] Nebulizers have two advantages over the MDI: (1) more than one medication can be given simultaneously; and (2) the system can be used in a hands-off fashion for the young, critically ill, or intubated patient. Spacers are indicated for patients who cannot adequately coordinate activation of the MDI with inhalation, to increase drug delivery, and for the prevention of oral candidiasis in the case of inhaled steroids.

Contraindications to use of these devices are few. The MDI may be difficult for the very young, elderly, or critically ill to use, either because of an inability to cooperate, weakness,or difficulty coordinating breathing with activation of the MDI. Spacer devices make the coordination of MDI use with inspiration less critical and, as noted earlier, have been used in the treatment of young infants. Oxygen-powered nebulization may be contraindicated in the case of $CO_2$ -retaining patients. [138]

## Metered-Dose Inhaler Use

### Equipment and Particle Delivery

Metered-dose inhalers consist of small canisters of highly pressurized liquid with a Freon propellant. An activator valve allows the delivery of consistent doses of medication in an aerosolized form. Particles of 1 to 5 mum in diameter are the most desirable size to achieve efficient peripheral airway deposition. [139] Ideally, the MDI output will have up to 13% of particles in this range, as opposed to only 1 to 5% for the nebulizer. The deposition of aerosol in the lower respiratory tract is a function of inertia and gravitational sedimentation.

Inertia limits the quantity of drug delivered to the therapeutic site because particles in motion, such as the drug suspended in nebulized liquid, have a tendency to move in a straight line, resulting in the majority of the drug bombarding the posterior pharynx. The open-mouth technique (as opposed to lips closed around the inhaler), with the inhaler held a few centimeters from the mouth, has been found to reduce this pharyngeal deposition. [140] By slowing the velocity of the particle, the airflow into the respiratory tract can have proportionately more time to effect transport or "entrain" the drug.

Spacer devices (see the following section) similarly allow for deeper penetration by reducing particle velocity, which in turn reduces droplet size both through the evaporation of the propellant that coats the drug crystal and the sedimentation of the larger particles. More central deposition occurs in patients with severe bronchial asthma

and chronic bronchitis because of the underlying airway obstruction. In this situation, fewer particles remain to be deposited further downstream because of early gravitational sedimentation. Centrally deposited particles are carried by mucociliary transport to the larynx and are swallowed, increasing the systemic absorption. Breath-holding enhances absorption by allowing the deposition of the smaller particles (less than 5 mum) that require up to 2 seconds to settle onto the walls of the terminal bronchioles. [140]

**Procedure**

The most important factor in determining the successful use of the MDI is the patient. Up to 78% of patients use the inhaler incorrectly, [141] and even after careful instruction, 14% still fail to perform efficiently. Furthermore, when housestaff and attending staff were evaluated, half the physicians could not assemble the device correctly, and only 4 of 55 performed all seven steps correctly; 3 of the 4 successes were among asthmatic physicians. [142] Others have confirmed that health care providers lack basic knowledge of inhalation therapy. [143]

Lower temperatures decrease the internal pressure and result in an increase in the size of the particles; therefore, the canister should be kept at room temperature. Lack of use can result in abnormally low doses, up to 50% less than expected. If the device has not been used for several days, it should be activated once or twice before actual use. [131] In the past, patients have been told to activate the inhaler at the beginning of inhalation, immediately after full expiration. Studies of patients with diseased small airways suggest that beginning activation somewhat later in the inspiration period may result in a greater bronchodilator effect by overcoming the requirement for a higher opening pressure, [144] [145] but this finding remains controversial. [146] [147] Spacer devices and inspiration-triggered inhalers exist for the patient who has difficulty coordinating these tasks.

*Before using the MDI*, it is important that the patient or clinician visualize the mouthpiece to ensure the absence of foreign material that might be inhaled with device activation. The aspiration of small coins and other foreign material have occurred when the device has been kept in the patient's pocket or purse. Although the device protective cap should be used during storage and will minimize this adverse event, it is still wise to double-check the mouthpiece prior to MDI activation.

To optimize *maintenance* use of the MDI, the canister is shaken first and held in the inverted position. The patient exhales as much as comfortably possible to functional residual volume. The mouthpiece of the nebulizer is placed approximately 2 to 4 cm from the open mouth or, if a spacer device is used, the lips are placed around the *spacer* mouthpiece. The patient then activates the device at shortly after inspiration inhaling *slowly* (at 10 L/min), with the tongue and teeth as removed as possible to decrease oropharyngeal deposition. Inhalation is continued as long as possible and held 10 seconds, if possible. [148] The inhalation is repeated in 1 to 5 minutes to allow the medication's preliminary effects to enhance the penetration of the second treatment. One pediatric study in which full exhalation and breath-holding subsequent to inhalation were omitted still found improved outcome, probably because of enhanced compliance

with the easier instructions. [149]

In the *acute* exacerbation setting, in which the physician elects to use the MDI rather than nebulizer therapy, the following approach is suggested. A fresh canister is used (or the patient's own MDI can be used if adequate medication remains; see the "float test"-- Fig. 6-22). A spacer device (see below) can be used to maximize the therapeutic effect. The patient is instructed carefully on the correct technique as described previously. (Demonstration of the technique in front of a family member is helpful; often family members are quite eager to ensure optimal home care.) When treating bronchospasm, the patient should initially take up to 4 consecutive puffs of a beta-adrenergic drug, such as albuterol delivered through a spacer. One study found that 90% of subjects reached maximal bronchodilatation after 3 treatments (4 puffs/treatment, 30 minutes apart, 1.2 mg total) of albuterol by MDI with spacer. [134] Patients failing to respond adequately should receive additional doses. The optimal interval of dosing has yet to be determined, and recommendations range considerably.

Failure of home therapy with the same medication does not preclude successful emergency department therapy for at least three reasons: (1) inadequate patient compliance, (2) use of an empty canister, and (3) inadequate dosing. Some patients have been cautioned against overuse of the MDI and may develop a fear of taking more than the ordered 2 puffs. Both physicians and patients should be comforted to know that double the current recommended maintenance dose, much less the occasional extra puff, is quite safe. Nonetheless, the patient and family should be cautioned that increased inhaler requirement indicates an exacerbation of the underlying disease requiring more aggressive therapy; this accelerated disease condition has been associated with increased risk of death or near death from asthma. [150]

**Spacer Devices**

Spacer devices serve as adjuncts to successful use of the MDI for the large subgroup of inhaler users with suboptimal technique. One study demonstrated a nine-fold reduction in oropharyngeal deposition and a greater than double increase in drug deposition over the whole lung with one of these instruments. [139] Additionally, spacers reduce the incidence of oral candidiasis for steroid inhalers by decreasing oropharyngeal deposition. [132]

Essentially, there are two types of spacers: (1) the tube and (2) the airbag type. The simple toilet roll tube, the plastic cola bottle, or the commercially available AeroChamber (Monaghan Medical Corp., Plattsburgh, NY; distributed by Forest Pharmaceuticals, Inc., St. Louis) and Ace Spacer (Center Laboratories, Port Washington, NY) are basically *fixed tubes* of various lengths and shapes (Fig. 6-23) . This type of inhaler can be constructed from common ventilator tubing and, when coupled with a mask (Fig. 6-24) (Figure Not Available) , has been used in the treatment of children as young as 1 month. [136] Recently, the AeroChamber device has been marketed complete with a mask for aiding delivery in the very young (AeroChamber with mask--small) or the old and infirm (AeroChamber with mask--large). This system maximizes drug delivery to those who can tolerate a face mask but cannot use the mouthpiece of the standard AeroChamber

device.

The inhaler mouthpiece is checked for the absence of foreign material, shaken well several times, and placed at one end of the spacer; the patient's mouth is at the other. The patient or parent activates the MDI; only a single puff is recommended. The patient breathes in slowly, exhales to the air, and takes several additional slow breaths from the tube. When the AeroChamber is used, the device makes a whistling noise if the patient breathes in too quickly. When the mask is used with the AeroChamber spacer, the mask is placed gently against the face to cover the nose and mouth (making a good seal), and the same process is followed. Crying children are still able to breathe through the system, and the facial contact should be maintained after MDI activation until several breaths are taken. (When the spacer is used with a corticosteroid MDI, the patient's face should be cleaned with soap and water to remove residue after use.)

Other adjuncts fall into the *airbag* group, wherein the inhaler is either attached to or placed within an air-filled bag. The canister is activated, and the patient subsequently breathes in and out of the bag. An airbag-type spacer can be constructed by cutting a hole in the permanently sealed end of a quart-size plastic bag with a zipper closure, placing the inhaler shell orifice in the small hole, and taping the plastic tightly around the shell. The canister is removed; the bag is inflated with air; and the canister is replaced, activated, and promptly removed. The shell then acts as a mouthpiece, and the patient breathes in and out of the bag four to five times. [151]

### Complications

In the 1960s, MDIs were blamed for excessive deaths in the asthmatic population. There is no proven causal relationship between MDI use and an increased risk of death. [152] However, increased beta-agonist use, suggesting disease exacerbation, has been *associated* with an increase in mortality. [150]

Some patients may be sensitive to the preservatives and lubricants contained in MDIs. Multidose dry powder inhalers, such as the Bricanyl Turbuhaler, may overcome this difficulty. [153]

Some treatment failures also can be attributed to untimely exhaustion of the drug contained in the inhaler, which catches the patient unaware. A simple method to assess drug quantity involves observation of canister flotation in a bowl of water (see Fig. 6-22). One author [154] challenges the efficacy of this technique and recommends calculating the number of inhalations remaining in the canister (200 inhalation capacity for most beta-agonist MDIs).

Oral candidiasis is a complication of inhaled steroids and can be controlled or eliminated by the use of spacers or by activating the MDI at a distance, as discussed previously.

### Handheld Nebulizer Use

**Equipment**

Nebulizers work on two principles. One type, based on the Bernoulli effect, uses $O_2$ or air at a flow rate of 5 to 10 L/min to aerosolize a liquid from a reservoir and carry the particles of medication through a tube and into the patient's upper respiratory tract. A second type of nebulizer produces an aerosol using the ultrasonic action of piezoelectric crystals to create the medicated mist. Those employing the Bernoulli effect are commonly used, and several manufacturers supply acorn-type reservoir nebulizers. Larger reservoir jet nebulizers are available for providing continuous nebulization (e.g., the HEART nebulizer, Vortran Medical Technology Inc., Orangevale, Calif).

**Procedure**

As with the MDI, patient compliance with correct nebulizer technique is poor. In the 1000-patient IPPB Trial Group Study, which compared IPPB and the simple nebulizer, compliance with the assigned regimen was approximately 55%. This degree of noncompliance occurred despite an intensive effort to obtain maximum compliance, wherein patients received repeated instruction and monthly visits by nurse practitioners over the 3 years of the study. [155]

For the procedure, a reservoir is filled with medication (most often a $beta_2$ -agonist, such as albuterol), along with normal saline as a liquid carrier. The reservoir is then connected to the piezoelectric generator or to a source of propellant gas, either air or $O_2$ at 5 to 10 L/min. The aerosol is delivered via a mouthpiece or a ventilation mask to the patient's upper respiratory tract. The patient inhales slowly and deeply, and does so repeatedly, until the onset of audible nebulizer sputtering indicates that medication delivery has ceased [156] or until intolerable side effects intervene. The optimal dosage and frequency of $beta_2$ -agonists via nebulizer is still undetermined. Actual drug delivery depends on many variables, including equipment, technique, and the patient's clinical condition. Continuous beta-agonist nebulization therapy has been used for several years in the pediatric population. However, recent studies [157] [158] suggest only marginal benefits of continuous nebulization of albuterol (at rates of 5 mg/hour and 15 mg/hour, respectively) in patients presenting with severe obstruction. Although no adverse affects of continuous albuterol nebulization were exhibited in these studies, an earlier study noted tachycardia with doses of 0.4 mg/kg/hour (30 mg/hour). [159]

Handheld nebulizers have been found to have less nasopharyngeal effect than the face-mask type. Tapping the sides of the reservoir releases droplets of medication that are trapped on the baffles. Enhanced deposition in the peripheral airways can be achieved using an increased flow rate to raise the fraction of appropriate-sized particles. [139]

**Complications**

Oxygen-powered nebulizers were considered to have contributed to the death of 5 patients who developed hypercarbia after receiving $O_2$ -powered nebulized salbutamol for severe respiratory distress. Two died despite intubation. [138] Transient, unpredictable

decreases in Pa$O_2$ with nebulizer use [160] have been found to be of significance only for the most severely compromised patient and can be recognized and managed by exercising the proper vigilance due in such a critical condition. [161] Cautious administration of supplemental $O_2$ during nebulizer therapy appears to minimize this effect. [162]

## Conclusions

There appears to be little controversy in the current literature as to the safety, efficacy, and superiority of the inhalant route of therapy for acute bronchospasm. Despite strong evidence of the advantages of MDIs (combined with spacers) compared with nebulizers for the treatment of acute reactive airway disease, nebulization remains the most widely used modality in most emergency departments. The MDI, when used correctly, is cheaper, less prone to contamination, and far more convenient for patients both in and out of the hospital.

The appearance of a patient suffering from an exacerbation of reactive airway disease should be viewed as an opportunity for medical personnel to observe and correct the patient's use of the MDI--a powerful therapeutic method when used correctly. Failure of the patient to demonstrate correct use of the MDI suggests the need to implement a spacer device. The spacer is a low-cost, highly efficient device that can pay for itself many times over in reduced future visits. Correction of poor technique and treatment of the acute process with the MDI will reduce the impression that a magical property is inherent in nebulizer use, simplify home therapy for many, and decrease medical costs.

# Chapter 7 - Mechanical Ventilation

**William Durston ,John J. Kelly** , **Barry J. Burton**

Once intubation has been undertaken and the tracheal tube has been secured in proper position, most patients who require prolonged ventilation will be placed on a ventilator. Ventilators have become increasingly technical since the mid-1980s, yet the basic functions prevail. The purpose of this chapter is to provide a usable and practical guide to allow the clinician to properly institute mechanical ventilation. The discussion includes indications for mechanical ventilation, ventilator setup and use, settings and features, and complications that may arise while using mechanical ventilation.

## INDICATIONS FOR MECHANICAL VENTILATION

The principal indication for mechanical ventilation is respiratory failure. Respiratory failure can be defined in terms of arterial blood gases, although criteria based on pulmonary mechanics have also been proposed. The indications for mechanical ventilation are somewhat debatable and must be applied selectively in individual situations, but general indications are summarized in Table 7-1 . Abbreviations used throughout this discussion are found in Table 7-2 .

Respiratory failure in adults is usually defined as either a $Pa_{O2}$ <60 mm Hg while the patient is breathing the maximum oxygen ($O_2$ ) concentration achievable by mask or

| TABLE 7-1 -- Indications for Mechanical Ventilation |
| --- |
| Blood gas criteria |
|    $Pa_{O2}$ <55-60 mm Hg on maximum $Fi_{O2}$ by mask |
|    $Pa_{CO2}$ >50 mm Hg and pH <7.30 |
| Blood gas criteria in neonates |
|    Patient on CPAP of 5-8 cm $H_2$ O and $Fi_{O2}$ up to 0.80 |

| |
|---|
| $Pa_{O_2}$ <55-60 mm Hg |
| $Pa_{CO_2}$ >60 mm Hg |
| pH <7.25 |
| Criteria based on pulmonary mechanics |
| Vital capacity <10 mL/kg |
| $FEV_1$ <10 mL/kg |
| MIF <25 cm $H_2O$ |
| $V_D/V_T$ >0.6 |
| Indications other than respiratory failure |
| Need for hyperventilation |
| Increased intracranial pressure |
| Tricyclic antidepressant overdose |

| |
|---|
| Hypothermia |
| Mechanical ventilation used as means of core rewarming |
| Prophylactic postoperative mechanical ventilation |
| In postoperative patients with shock, morbid obesity, COPD, neuromuscular disease, or other debilitating illness or following cardiothoracic surgery |

a $Pa_{CO2}$ >50 mm Hg with a pH <7.30. These criteria must be modified by the clinical situation. For example, a patient with chronic obstructive pulmonary disease (COPD) who retains carbon dioxide (a *CO2 retainer*) but who has an acute loss of bicarbonate due to a diarrheal illness, might have a $Pa_{O2}$ value of 55 mm Hg, a $Pa_{CO2}$ of 50 mm Hg, and a pH of 7.32 and yet be quite stable from a pulmonary standpoint. A young asthmatic with the same blood gas values might be in need of immediate intubation and mechanical ventilation. [1]

In addition to arterial blood gases, other indices of pulmonary function may be helpful in determining whether a patient requires mechanical ventilation. In adults, a respiratory rate of >35 to 40 breaths/min usually cannot be sustained for prolonged periods, and if this rate is required to maintain a normal pH or $Pa_{CO2}$ , tachypnea may be an indication for mechanical ventilation. A forced expiratory volume in 1 second ($FEV_1$ ) of <1000 mL or <10 mL/kg indicates severe airway obstruction and, if not readily reversible, predicts that the patient may need ventilatory assistance. A vital capacity (VC) <10 mL/kg and a maximum inspiratory force (MIF) <25 cm $H_2$ O are other indications that the patient will not be able to adequately maintain independent ventilation. Similarly, a dead space to tidal volume ratio ($V_D/V_T$) of >0.6 implies a high minute volume requirement and a need for ventilatory assistance in most patients.

Respiratory failure in adults is usually caused by primary pulmonary disease, cardiac disease, neuromuscular disease, drug overdose, or a combination of these conditions. In neonates, respiratory failure usually results from hyaline membrane disease, meconium aspiration, sepsis, or congenital cardiopulmonary anomalies. Respiratory failure in children beyond the neonatal period is most often due to respiratory infections, asthma, or accidents such as near-drowning or chemical aspiration. Indications for mechanical ventilation based on pulmonary mechanics are difficult to apply in infants and children because the patients are usually unable to cooperate in measuring VC, $FEV_1$ , and MIF. Respiratory rates of 35 to 40 breaths/min are normal in neonates and common in febrile children (see Chapter 70) , and tachypnea alone is not an indication

for mechanical ventilation.

The blood gas criteria for respiratory failure in infants are similar to those in adults, except that more deviation from the physiologic norm may be allowed before mechanical ventilation is instituted. Typical blood gas criteria for respiratory failure in infants cited in the literature include a pH <7.25, a $Pa_{CO2}$ >60 mm Hg, and a $Pa_{O2}$ <55 to 60 mm Hg. Infants who are hypoxemic but not hypercapnic may first be tried on continuous positive airway pressure (CPAP) with high inspired $O_2$ concentrations before mechanical ventilation is instituted. [2] [3] [4] As with adults, other clinical factors must be weighed in deciding whether to institute mechanical ventilation. Apneic or bradycardiac periods may warrant ventilatory support, and low-birth-weight or premature infants are more likely to require mechanical ventilation.

In the absence of respiratory failure, there are few other indications for mechanical ventilation. Flail chest was formerly considered one of these. The concept of alkalotic apnea for internal pneumatic stabilization of the critically "crushed chest" was proposed in 1956 by Avery and colleagues, who found that positive-pressure ventilation was more effective and humane in the treatment of flail chest than were the older methods of external stabilization with Hudson traction and towel clips. [5] For 2 decades following

| | |
|---|---|
| **TABLE 7-2** -- Abbreviations Used in This Chapter | |
| A-a$_{DO2}$ | Alveolar-arterial oxygen difference |
| ARDS | Adult respiratory distress syndrome |
| Ca | Oxygen content of systemic arterial blood |
| Cc | Oxygen content of pulmonary capillary blood |
| Cv | Oxygen content of mixed venous blood |

| | |
|---|---|
| COPD | Chronic obstructive pulmonary disease |
| CPAP | Continuous positive airway pressure |
| f | Respiratory rate or frequency |
| $FEV_1$ | Forced expiratory volume in 1 second |
| $FIO_2$ | Percent oxygen content of inspired gas |
| HFV | High-frequency ventilation |
| IMV | Intermittent mandatory ventilation |
| IRV | Inverse ratio ventilation (I:E ratio >1) |
| MIF | Maximum inspiratory force |
| MVV | Maximum voluntary minute volume |
| $P_{ACO_2}$ | Alveolar carbon dioxide tension |
| $Pa_{CO_2}$ | Arterial carbon dioxide tension |

| | |
|---|---|
| $P_{AO_2}$ | Alveolar oxygen tension |
| $Pa_{O_2}$ | Arterial oxygen tension |
| $P_{ECO_2}$ | Tension of carbon dioxide in mixed expired air |
| PEEP | Positive end-expiratory pressure |
| Qs/Qt | Shunt fraction (ratio of right-to-left shunt to total pulmonary blood flow) |
| R | Respiratory quotient (ratio of carbon dioxide produced by the body to oxygen consumed) |
| SIMV | Synchronized intermittent mandatory ventilation |
| $V_A$ | Alveolar ventilation |
| $V_A$ | Alveolar minute ventilation |
| VC | Vital capacity |
| $V_{CO_2}$ | Carbon dioxide production per minute |
| $V_D$ | Dead space volume |

| $V_E$ | Expired minute volume |
|-------|-----------------------|
| $V_T$ | Tidal volume |

Avery's report, it was largely accepted that most patients with flail chest segments should be treated with mechanical ventilation to minimize chest wall motion caused by spontaneous respiratory effort and to allow the fractured ribs to heal. More recently, it has been recognized that patients who have flail segments but who do not meet usual blood gas criteria for respiratory failure do better if treated only with pain control, including epidural and intercostal blocks, than if treated with mechanical ventilation. [6]

Mechanical ventilation may be instituted in the absence of respiratory failure in patients who require hyperventilation. In patients with increased intracranial pressure, hyperventilation to a $Pa_{CO2}$ of 30 to 35 mm Hg has been advocated to modestly reduce cerebral blood volume and intracranial pressure. [7] Hyperventilation has also been proposed to produce a respiratory alkalosis as a means of preventing seizures and ventricular dysrhythmias in patients suffering from tricyclic antidepressant overdose. [8]

In severely hypothermic patients, the administration of warm nebulized air is an adjunctive means of core rewarming. [9] Although such patients usually require mechanical ventilation for other reasons, the need for rewarming might be considered an additional indication. Postoperative mechanical ventilation is also used "prophylactically" in certain surgical patients who are at high risk for the development of respiratory failure, atelectasis, or pneumonia. Conditions that may place a patient in a high-risk category include shock, morbid obesity, COPD, neuromuscular disease, or other debilitating illnesses. Patients also are at increased risk for the development of respiratory complications following cardiothoracic surgery. [10]

There are no absolute contraindications to mechanical ventilation. Some consider COPD with chronic $CO_2$ retention to be a relative contraindication, because patients with this condition are notoriously difficult to wean back to spontaneous breathing. When the COPD patient presents with respiratory failure, however, the condition has usually been precipitated by an acute process, such as a pulmonary infection, which can be reversed while the patient receives ventilatory assistance. The complication rate is unusually high in patients with asthma during mechanical ventilation as a result of the high pressures required to ventilate these patients. [11] Hence, combining positive-pressure ventilation with pharmacologic sedation and/or muscle relaxation may be required. Although barotrauma should be anticipated in treating severe asthmatics with mechanical ventilation, mechanical ventilation nonetheless may be life saving. In some patients with terminal illness or chronic debilitating disease without hope for clinical recovery, a decision not to initiate mechanical ventilation may be made on moral grounds.

# TYPES OF VENTILATORS

Mechanical ventilators manufactured by more than a dozen companies are currently available in the United States; most manufacturers offer several different models. Despite the diversity of these machines, they may be grouped and classified according to a few basic characteristics that describe their operation.

## Inspiratory Flow

There are basically 2 types of inspiratory flow patterns built into modern ventilators. The inspiratory flow either is constant during the inspiratory cycle or varies from the start to the end of the cycle. In general, ventilators with constant flow are called *flow generators*, whereas ventilators with variable flow are classified as *pressure generators*. [12] In a constant- *flow* generator, a high-pressure gradient is established between the ventilator and the patient. The machine is built with a high internal resistance so that changes in the resistance and compliance of the patient's airways make relatively little contribution to the total resistance of the system. The result is a constant, or square wave, flow pattern (Fig. 7-1 (Figure Not Available) *A*). In a constant- *pressure* generator, the machine develops a constant pressure that is only slightly above the pressure in the patient's airways. An exponential flow pattern results, with flow approaching zero as the patient's airway pressure approaches the pressure from the ventilator (Fig. 7-1 (Figure Not Available) *B*).

It is apparent that constant-flow generators and constant-pressure generators are not conceptually different but rather are the opposite ends of a spectrum. By reducing internal resistance and machine pressure, a constant-flow generator can be converted into a constant-pressure generator. A sine wave flow pattern is also available on some ventilators (Fig. 7-1 (Figure Not Available) *C*). Such a flow pattern is produced by gas being driven by a piston on an eccentric cam.

The flow patterns of ventilators are of more interest to bioengineers than to clinicians, except that some modern ventilators have controls that allow the operator to switch from one flow pattern to another. As discussed in the section on ventilator settings, by changing the flow pattern, one may sometimes produce small but significant improvements in ventilation of the patient.

## Cycling

In addition to being classified according to their inspiratory flow characteristics, ventilators are classified according to the factor that determines when the ventilator cycles from the inspiratory phase to the expiratory phase. Basically, ventilators can be described as pressure-cycled, volume-cycled, or time-cycled.

### Pressure-Cycled

In pressure-cycled ventilators, the inspiratory phase is terminated, and expiration begins

when a preset pressure limit is reached. The tidal volume ($V_T$) received by the patient is not set directly but depends on the set pressure limit and the patient's chest and lung compliance and airway resistance. As long as the patient's compliance and resistance do not change, the $V_T$ will be the same with each breath. If the patient's compliance falls or resistance increases, the $V_T$ will also fall, and hypoventilation may result.

Because of the problem of a changing $V_T$ caused by changing patient compliance, pressure-cycled ventilators have been largely replaced by volume- or time-cycled ventilators for mechanical ventilation in adults. Pressure-cycled ventilators still have certain advantages, however. They are less expensive than volume- and time-cycled machines. They are more compact and can be run by compressed gas sources without the need for an electrical source, making them well suited for ambulance transport. For reasons discussed later, pressure-cycled machines also remain popular for ventilating infants and neonates.

**Volume-Cycled**

In volume-cycled ventilators, inspiration is terminated and expiration begins when a preset $V_T$ is delivered. The gas is usually delivered from a compressible bellows. Since the introduction of the Puritan-Bennett MA-1 volume ventilator in 1968, volume ventilators have become the standard for mechanical ventilation in adults. They have an important advantage over pressure-cycled ventilators: delivery of a relatively constant $V_T$ despite changes in the patient's compliance. Even with volume-cycled machines, however, the delivered $V_T$ falls slightly if the patient's compliance falls. This is because although a constant volume is delivered from the ventilator bellows, as the patient's lungs become stiffer, more of this gas is lost to expansion of the ventilator tubing. This phenomenon becomes very important in infants. The compliance of the child's chest and lungs may be less than the compliance of the ventilator tubing, and more gas will go to expansion of the tubing than to ventilation of the patient.

Most modern volume-cycled ventilators have adjustable secondary pressure limits such that when the airway pressure exceeds the set limit, inspiration is terminated. Thus, volume-cycled ventilators may function as pressure-cycled ventilators when the pressure limit is set at a low enough level.

**Time-Cycled**

In time-cycled ventilators, inspiration is terminated and expiration begins after a preset time has elapsed. The $V_T$ that is delivered is determined by the integral of the inspiration flow-inspiratory time curve. Time-cycled ventilators resemble volume-cycled machines in that they deliver a relatively constant $V_T$ despite changes in the patient's compliance. They may also function as pressure-limited ventilators when

the secondary pressure limits are adjusted. Time-cycled ventilators are becoming increasingly popular. They allow great flexibility in adjustment of the inspiratory-to-expiratory ratio, and their internal circuitry is such that they can be

manufactured at a lower cost than most volume-cycled machines.

## CHOOSING VENTILATOR SETTINGS

To optimize ventilator therapy, the physician must understand the capabilities of the ventilator, the pathophysiology of the patient, and how to match the ventilator settings to the patient's condition to achieve the desired results. In this section, the rational choice of ventilator settings is discussed. Not all of the settings described in this section are available on every ventilator. To learn the capabilities of a given ventilator, the physician should consult the operating manual supplied with the particular machine. A summary of recommended initial ventilator settings is provided in Table 7-3 .

### Rate and Tidal Volume

The two most important settings on a volume-cycled ventilator (and those that are usually set first) are the rate and $V_T$. To provide an understanding of the way in which the rate and $V_T$ determine alveolar ventilation and the arterial $CO_2$ tension ($Pa_{CO_2}$ ), it is necessary to briefly review some aspects of pulmonary physiology.

#### Minute Volume and Alveolar Ventilation

The volume of air that moves in and out of a patient's lungs per minute is termed the minute volume ($V_E$). Minute volume is the product of $V_T$ and respiratory frequency or rate (f):

$$V_E = V_T \times f \; [1]$$

Tidal volume can be further broken down into alveolar ventilation ($V_A$) and dead space ventilation ($V_D$):

$$V_T = V_A + V_D \; [2]$$

In healthy young persons, the anatomic dead space can be accounted for by the trachea and the larger airways and is approximately 2.2 mL/kg of lean body weight. In disease states, in addition to the anatomic dead space, there is also a variable amount of "pathologic" dead space corresponding to ventilated alveoli and respiratory bronchioles that are not adequately perfused. The sum of the anatomic and pathologic dead spaces is often referred to as the physiologic dead space.

Alveolar minute ventilation ($V_A$) is the product of rate times $V_T$ minus dead space:

$$V_A = (V_T - V_D) \times f \; [3]$$

Alveolar minute ventilation and the rate of $CO_2$ production by the body ($V_{CO_2}$ ) determine the partial pressure of $CO_2$ in the alveoli ($PA_{CO_2}$ ), which is approximately equal to the systemic arterial $CO_2$ tension ($Pa_{CO_2}$ ). This relationship is shown in

equation 4:

$$PaCO_2 \; PACO_2 = k \times (V_{CO_2} / V_A) \; [4]$$

where the value of the constant (k) is 0.863 when the partial pressure of $CO_2$ is measured in millimeters of mercury at 37 °C saturated with water vapor, $V_{CO_2}$ is measured in milliliters per minute, and $V_A$ is measured in liters per minute.

Using equations 3 and 4, one can work through the example of an average 70-kg man with a typical spontaneous $V_T$ of 500 mL, respiratory rate of 12 breaths/min, $CO_2$ production of 200 mL/min, and dead space of 150 mL, and see that the result is a normal $Pa_{CO_2}$ :

$$V_A = (0.500 - 0.150) \times 12 \; 4.2 \; L/min$$

and

$$Pa_{CO_2} = 0.863 \times (200/4.2) = 41.0 \text{ mm Hg}$$

If the same 70-kg man were to develop respiratory failure purely on a neurologic basis, without any change in

| TABLE 7-3 -- Recommended Initial Ventilation Settings | | |
| --- | --- | --- |
| **Parameter** | **Recommended Setting** | **Comment** |
| Tidal volume | 10-15 mL/kg | 8 to 12 mL/kg in infants (see section on rate and tidal volume) |
| Rate | 12-14 breaths /min | Starting rates of 25 to 30/min used in infants; rates of 18/min used in adults when hyperventilatation is indicated or when $V_D/V_T$ is very high (see section on rate and tidal volume) |

| | | |
|---|---|---|
| Fi$_{O_2}$ | 0.5-1.0 | To avoid oxygen toxicity, reduce Fi$_{O_2}$ to <0.5 as soon as possible, guided by blood gas analysis results |
| Ventilator mode | Assist control, IMV, or SIMV | (See section on ventilator mode) |
| PEEP | None | Start with PEEP of 5 cm $H_2O$ and increase in 5 cm $H_2O$ increments if Pa$_{O_2}$ <60 mm Hg with Fi$_{O_2}$ 0.50 (see section on PEEP and CPAP) |
| Inspiratory waveform | Square wave | (See section in inspiratory flow) |
| Inspiratory flow | 50 L/min | 8 to 15 L/min in infants (see section on inspiratory flow rate) |
| Inspiratory pause | None | (See section on inspiratory pause) |
| I:E ratio | 1:2 | (See section on I:E ratio) |
| Peak pressure | 50 cm $H_2O$ | With pressure-cycled ventilators, starting pressures of 20-30 cm $H_2O$ should be used in infants and in adults with normal compliance (see section on peak pressure) |
| Expiratory retard | None | (See section on expiratory retard) |

| | | |
|---|---|---|
| Sighs | None | (See section on sighs) |
| Humidifier temperature | 35°C | Use higher temperature (40°C) for rewarming hypothermic patients, lower temperature for cooling febrile patients |

his dead space or $CO_2$ production, one might assume that the appropriate ventilator settings would be a $V_T$ of 500 mL and a rate of 12 breaths/min. It has been found empirically, however, that when patients with $V_T$ values that are in the normal range for spontaneous breathing are ventilated, atelectasis and hypoxemia develop. [10] This can be prevented by ventilating patients at higher $V_T$ levels, in the range of 10 to 15 mL/kg. Also, mechanical ventilation alters the normal ventilation-perfusion relationship in the lungs, causing relatively greater ventilation of the less-well-perfused upper lung regions, which in turn results in an increase in physiologic dead space. [13] The magnitude of this effect is equivalent to roughly doubling the predicted dead space. [14]

Returning to the case of our 70-kg man, assuming a physiologic dead space on the ventilator of 300 mL, a desired $V_T$ of 12 mL/kg (850 mL), and a desired $Pa_{CO_2}$ of 40 mm Hg, one can use equations 3 and 4 to determine the desired ventilator rate:

$Pa_{CO_2} = 0.863 \, (V_{CO_2} / V_A)$

$40 = 0.863 \times (200/V_A)$

To determine $V_A$:

$V_A = (0.863 \times 200)/40$

$= 4.3 \, L/min$

By putting this value for $V_A$ into equation 3, the value of f can be determined:

$V_A = (V_T - V_D) \times f$

$4.3 = (0.850 - 0.300) \times f$

$f = 4.3/(0.850 - 0.300)$

= 7.8 breaths/min

8 breaths/min

In practice, such a low rate is rarely prescribed as the initial ventilator setting. Most patients requiring mechanical ventilation have more than normal dead space, and it is usually safer to risk mild hyperventilation than hypoventilation. Starting rates of 12 to 14 breaths/min are recommended for most adult patients.

**Changes in $PaCO_2$ as a Result of Changes in Rate and Tidal Volume**

Once a patient has been placed on a ventilator at a rate and $V_T$ that are deemed appropriate, it is necessary to check arterial blood gas values to be sure that the patient is being adequately ventilated and oxygenated. Arterial samples for blood gas analyses are customarily drawn 15 minutes after the initiation of mechanical ventilation or after changes in ventilator settings. This interval is supported by indirect evidence suggesting that blood gases reach equilibrium within 15 minutes after ventilator changes in most patients with severe pulmonary disease and much sooner in patients with normal lungs. [15] [16] In most patients, achieving a normal $Pa_{CO2}$ (36 to 44 mm Hg) is the goal. In some cases, however, a higher or lower $Pa_{CO2}$ is desired. For example, most experts warn against rapid reduction of $Pa_{CO2}$ to normal in patients with chronic $CO_2$ retention. The resultant alkalemia may have many undesirable consequences, including diminished cardiac output, diminished cerebral blood flow, hypokalemia, hypocalcemia with associated seizures, increased airway resistance, and a shift in the hemoglobin-oxygen dissociation curve to the left with impaired release of $O_2$ to the tissues. [13] However, in patients with increased intracranial pressure due to trauma, infection, or cerebrovascular accident, a slightly lower than normal $Pa_{CO2}$ may be desired to help lower intracranial pressure. [7]

Whatever the desired $PaCO_2$, if the measured $Pa_{CO2}$ differs from the desired level, the ventilator rate or $V_T$ must be adjusted accordingly. One can easily calculate the amount of change in ventilator settings needed to produce the desired change in $Pa_{CO2}$ using the following relationship:

$$V_{E2} = (Pa_{CO2}\ 1\ /Pa_{CO2}\ 2\ ) \times V_{E1}\ [5]$$

where $V_{E2}$ is the desired minute volume, $V_{E1}$ is the present minute volume, $Pa_{CO2}\ 2$ is the desired $Pa_{CO2}$, and $Pa_{CO2}\ 1$ is the present $Pa_{CO2}$. Strictly speaking, $Pa_{CO2}$ varies inversely with alveolar minute ventilation and not with total minute ventilation, which includes both alveolar ventilation and wasted, dead space ventilation. It has been observed empirically, however, that the ratio of $V_D/V_T$ remains relatively constant despite changes in $V_T$. [17] Thus, equation 5 holds true whether rate or $V_T$ or both are altered. As an example, suppose that a 70-kg patient is being ventilated at a rate of 10 breaths/min with a $V_T$ of 900 mL. The observed $Pa_{CO2}$ is 50 mm Hg, and the desired $PaCO_2$ is 30 mm Hg. The desired minute volume is calculated as follows:

$V_{E2}$ = (50/30) × 9.0 L/min

ISOdia = 15 L/min

To obtain a $Pa_{CO_2}$ of 30 mm Hg, the rate could be increased to 16 and the $V_T$ to 940 mL, giving a new minute volume of 15 L/min.

**Adding Dead Space**

Some patients who are placed on ventilators and who are allowed to trigger the machine themselves, as in the assist control mode, will spontaneously hyperventilate. If the $Pa_{CO_2}$ drops as low as 25 to 30 mm Hg in a patient who is used to a normal $Pa_{CO_2}$ , the same complications may develop as described previously for rapidly lowering the $CO_2$ to normal in a chronically hypercapnic patient. An approach that may be useful in dealing with patients who spontaneously hyperventilate is to add dead space to the ventilator tubing so that the patient rebreathes some of the expired air. A formula has been developed that predicts the amount of dead space that must be added to give a desired increase in $Pa_{CO_2}$ . [18] This formula is somewhat complicated, though, and requires determination of the concentration of $CO_2$ in the expired air. Empirically, it has been found that the addition of 50 mL of dead space leads to an increase in $Pa_{CO_2}$ of approximately 5 mm Hg in most patients.

Another approach to raising the $Pa_{CO_2}$ in a patient who spontaneously hyperventilates is to add $CO_2$ to the inspired air. A rise of 1% in the inspired $CO_2$ concentration leads to a rise in $Pa_{CO_2}$ of approximately 5 mm Hg. [19] The disadvantage of using this method rather than adding dead space is that it requires an expensive piece of additional equipment, the $CO_2$ mixer. Paradoxically, it has been found that increasing the $Pa_{CO_2}$ by either method leads to a rise, rather than a decline, in $Pa_{O_2}$ .

The paradoxical rise in $Pa_{O_2}$ seems to be a result of the

increased cardiac output that occurs with normalization of the $Pa_{CO_2}$ . The problem with adding either dead space or inspired $CO_2$ is that the patient may hyperventilate even more, returning the $Pa_{CO_2}$ to harmfully low levels. If this occurs, the patient should be sedated or changed to a different ventilator mode, as will be discussed subsequently.

**The Dead Space-to-Tidal Volume Ratio**

As noted earlier, in addition to their anatomic dead space, patients with pulmonary disease have variable amounts of pathologic dead space. The $V_D/V_T$ ratio is useful to follow as an index of the severity of a patient's pulmonary disease. The normal $V_D/V_T$ ratio is 0.20 to 0.35. [20] Patients with $V_D/V_T$ ratios 0.6 usually need ventilatory assistance, because they require large minute volumes to maintain adequate alveolar ventilation. The $V_D/V_T$ ratio may be calculated using the Bohr equation, as follows:

$$V_D/V_T = (P_{ACO2} - P_{ECO2})/P_{ACO2} \quad [6]$$

where $P_{ECO2}$ is the partial pressure of $CO_2$ in mixed expired air and $P_{ACO2}$ is the partial pressure of $CO_2$ in alveolar air, which is assumed to be the same as $Pa_{CO2}$. To use this equation, one must have access to a $CO_2$ analyzer. Although these analyzers are commercially available, they are not standard equipment in all emergency departments or intensive care units. For this reason, a graph has been constructed from which $V_D/V_T$ can be determined if the patient's minute ventilation and $Pa_{CO2}$ (Fig. 7-2) (Figure Not Available) are known. [21] The graph assumes a normal $CO_2$ production of 200 mL/min. Although critically ill patients tend to have higher rates of $CO_2$ production, it has been found empirically that $V_D/V_T$ ratios determined by the graph correlate well with $V_D/V_T$ ratios measured directly in patients in intensive care units. [21] The graph can also be used to predict the necessary minute ventilation to produce a desired $Pa_{CO2}$ in a patient with a known $V_D/V_T$ ratio.

**Compliance**

Measurement of pulmonary compliance is useful in following the progression of a patient's pulmonary disease as well as in determining the optimal $V_T$ and level of positive end-expiratory pressure (PEEP--see the following discussion). Compliance is defined as change in volume over change in pressure:

$$Compliance = DeltaV/DeltaP \quad [7]$$

Total pulmonary compliance can be subdivided into the compliance of the lung and the compliance of the chest wall. For practical purposes in managing most ventilator patients, only the total compliance need be considered. Total compliance in a ventilator patient may be measured as either static compliance, which is the $V_T$ delivered divided by the plateau airway pressure after the patient has been held in full inspiration for 1 second, or as dynamic compliance, which is the $V_T$ divided by the peak airway pressure. (When measuring compliance in a patient on PEEP, one considers the change in pressure to be the difference between the peak or plateau inspiratory pressure and the PEEP level.)

In monitoring ventilator therapy, measurements of static compliance are usually preferred, because dynamic compliance is affected not only by the elastic recoil properties of the patient's chest wall and lungs but also by the patient's airway resistance. Normal static compliance in a healthy young adult undergoing general anesthesia is approximately 50 mL/cm $H_2O$. [22] It is believed that the $V_T$ that leads to the greatest total compliance is that which results in maximal recruitment of alveoli without overdistention. In most patients, the best compliance is found with $V_T$ values of 12 to 15 mL/kg. [23] In addition to being affected by $V_T$, compliance is also affected by PEEP and characteristics of inspiratory flow, as is discussed later.

**Set Tidal Volume vs. Delivered Tidal Volume**

As discussed, an advantage of volume- and time-cycled ventilators is that they deliver a relatively constant volume despite changes in the patient's compliance. One must remember that even with volume- and time-cycled ventilators, delivered $V_T$ will drop slightly if the patient's compliance goes down. This drop in delivered volume occurs because the ventilator tubing has compliance of its own. Although most of the gas mixture delivered by the ventilator bellows goes to expansion of the patient's lungs, a certain amount is lost to expansion of the ventilator tubing.

The compliance of the tubing varies from ventilator to ventilator. For a typical modern volume ventilator, such as the Bear-2, the compliance of the inflow circuit is 3 mL/cm $H_2O$. [24] To calculate the difference between the set $V_T$ and that actually received by the patient, one can multiply the pressure read from the inspiratory pressure gauge at the end of inspiration by the tubing compliance. For example, if the set $V_T$ is 850 mL and the peak inspiratory pressure is 30 cm $H_2O$, then the volume delivered to the patient is as follows:

Delivered volume = set volume - (tubing compliance × peak pressure)

= 850 - (3 × 30)

= 760 mL

The lower the patient's compliance, the higher the peak inspiratory pressure and the greater the amount of gas mixture that will be captured in the ventilator tubing.

**Tidal Volume and Rate in Infants**

Some controversy remains concerning the appropriate rate and $V_T$ for mechanical ventilation of infants. Some researchers recommend lower $V_T$ values, in the range of 8 to 12 mL/ kg, and correspondingly rapid rates of 25 to 30 breaths/min. [2] In neonates with hyaline membrane disease, rates as high as 60 to 100 breaths/min may be used in an attempt to synchronize the ventilator with the infant's own breathing pattern. [25] Other investigators recommend higher $V_T$ values (15 to 20 mL/kg) and lower respiratory rates (16 to 22 breaths/min). [26] Most ventilators used for adults and larger children are not capable of delivering the small $V_T$ values used in infants and neonates. (An exception is the Siemens-Elema Servo ventilator. [27] ) Therefore, special ventilators have been developed for neonatal intensive care. The compliance and dead space of the ventilator tubing and the valves have special importance in neonates. The total pulmonary compliance of a newborn with hyaline membrane disease may be 1 mL/cm $H_2O$, which is the same as the compliance of some ventilators designed especially for use in neonates. Thus, with each breath delivered by the machine, half of the volume expands the patient's lungs and the other half expands the ventilator tubing. This technical problem has to some extent hampered the development of volume ventilators for infants. [28] Another problem in infants and children is that leaks around uncuffed endotracheal tubes prevent accurate determination of volumes delivered to the lungs. Adequacy of ventilation must be ensured by clinical observation of chest excursions,

auscultation of breath sounds, and blood gas determinations. [29]

## Inspired Oxygen Concentration

After rate and $V_T$ have been set, the next variable that the physician usually sets is the inspired $O_2$ concentration ($Fi_{O2}$ ). The goal should be to deliver the lowest $O_2$ concentration that provides adequate arterial oxygenation. Adequate arterial oxygenation is difficult to define and may vary from patient to patient. Most experts recommend maintaining a $Pa_{O2}$ of 60 mm Hg, because this measurement corresponds to the shoulder on the normal hemoglobin-oxygen dissociation curve at which hemoglobin is 90% saturated with $O_2$ . Beyond this point, increases in $Pa_{O2}$ lead to relatively little improvement in hemoglobin saturation, whereas below this point, small decrements in $Pa_{O2}$ cause large drops in hemoglobin saturation. Conditions such as acidemia, fever, hypercarbia, and certain hemoglobinopathies result in a shift in the curve to the right so that at a $Pa_{O2}$ of 60 mm Hg, the hemoglobin will be <90% saturated. Thus a target $Pa_{O2}$ of 60 mm Hg may not be adequate in such compromised patients. [30]

### Oxygen Toxicity

When one is initiating mechanical ventilation, it is better to err on the side of a higher than necessary $Fi_{O2}$ than to risk making a patient hypoxemic. Values of $Fi_{O2}$ in the range of 0.50 to 1.00 are commonly prescribed as initial settings. With long-term ventilator support, however, a higher than necessary $Fi_{O2}$ may have serious adverse effects. The phenomenon of pulmonary $O_2$ toxicity has been recognized since the turn of the century, although the pathophysiology is still not completely understood. [31] The syndrome begins with tracheal irritation, cough, and chest pain, followed by diminished vital capacity and dyspnea. In the later stages, hypoxemia develops with associated alveolar edema and infiltrates on chest radiographs. Finally, as the process becomes irreversible, fibrosis of alveoli occurs.

Although the exact dose-time relationship of pulmonary $O_2$ toxicity has not been established, it is known that at atmospheric pressure, an $Fi_{O2}$ of 0.40 is well tolerated for 30 days or more, an $Fi_{O2}$ of 0.70 will lead to signs and symptoms of toxicity by 2 days, and an $Fi_{O2}$ of 1.00 leads to toxicity within 30 hours. [31] In general, an $Fi_{O2}$ of 0.50 should be considered potentially toxic if used for more than a few days.

In neonates, two other manifestations of $O_2$ toxicity may occur: retrolental fibroplasia and bronchopulmonary dysplasia. [32] [33] The dose-time relationship of $O_2$ administration to the development of these two conditions is even less well established than $O_2$ toxicity in adults. A multicenter cooperative study designed to develop guidelines for the safe administration of $O_2$ in neonates was unable to yield any firm recommendations. [34]

### The Alveolar-Arterial $O_2$ Difference

After the patient has been on a given $FI_{O2}$ for 15 minutes, arterial blood gases should be checked. Besides confirming the adequacy of arterial oxygenation, blood gas results

can be used to measure the efficiency of the lungs in oxygenating venous blood. One way to quantify how well the lungs are doing their job is to compare the $O_2$ concentration delivered to the alveoli with the $O_2$ tension in arterial blood. The difference between these 2 values is known as the alveolar-arterial $O_2$ difference (A-$aDO_2$ ). The arterial $O_2$ tension ($PaO_2$ ) is easily obtained by blood gas analysis, and the alveolar $O_2$ tension $PAO_2$ ) is calculated using the following alveolar air equation:

$$P_{AO_2} [F_{iO_2} \times (P_B - P_{H_2O})] - P_{ACO_2} \times F_{iO_2} /R \quad [8]$$

where $P_B$ is barometric pressure (760 mm Hg at sea level), $P_{H_2O}$ is the pressure of water vapor in the patient's lungs (47 mm Hg at 37 °C), and R is the respiratory quotient (assumed to be 0.8 unless it has been directly measured). The alveolar $CO_2$ partial pressure, $P_{ACO_2}$ , is assumed to be the same as the arterial $CO_2$ tension, $P_{aCO_2}$ . When the patient is breathing 100% $O_2$ , this equation simplifies to the following:

$$P_{AO_2} 713 - P_{aCO_2} /R \quad [9]$$

At room air, a further approximation that is easy to remember (for sea level calculations) is as follows:

$$P_{AO_2} 150 - 1.2 \times P_{aCO_2} \quad [10]$$

The normal A-$aDO_2$ is approximately 8 mm Hg in healthy young adults and 16 mm Hg in healthy 60 year olds with

the subjects breathing room air at atmospheric pressure. (The formula of 10 mm Hg plus the age in decades can be used to estimate the normal gradient). With increasing inspired $O_2$ concentrations, the normal value for A-$aDO_2$ increases, although not in a strictly linear fashion. The normal A-$aDO_2$ for a healthy young adult breathing 100% $O_2$ is 30 to 50 mm Hg. [35]

## Ventilator Mode

After rate, $V_T$, and $F_{iO_2}$ have been set, the next priority is to set the mode of ventilation. Although not all of the modes discussed in this section are available on all ventilators, most modern machines allow the operator to choose more than one mode. The airway pressure and flow characteristics of the different modes are shown diagrammatically in Figure 7-3 .

### Controlled Ventilation

In this mode, the patient is ventilated at the rate set by the operator. The patient cannot breathe between machine breaths. This mode may be used in an unconscious patient with depressed respiratory drive, in a heavily sedated patient, in a patient who has been paralyzed with drugs, or in a patient who is being deliberately hyperventilated. In this mode, alert patients may attempt to inhale against the closed inspiratory valve, resulting in apprehension, asynchronous chest movement, increased $O_2$ consumption and $CO_2$

production, and high peak pressures.

**Assist Control**

In this mode, the operator sets the minimum rate for the patient to be ventilated. If the patient makes no respiratory effort, the prescribed number of breaths will be delivered and no more. If the patient does try to breathe, the machine will deliver an extra breath with the same $V_T$ as the others, when the patient generates a sufficient inspiratory effort. The amount of negative inspiratory pressure that the patient must generate to trigger the machine is controlled by the operator. The sensitivity is set so that it is neither too difficult for the patient to initiate a breath (in which case the ventilator would actually be functioning in the control mode) nor too easy (in which case small movements by the patient might trigger the machine). The advantage of assist control over the control mode is that it allows the patient to regulate the minute volume in part. This mode is usually preferred in conscious patients. A disadvantage of assist control is that some patients spontaneously hyperventilate to an undesirable degree in this mode.

**IMV and SIMV**

Intermittent mandatory ventilation (IMV) was introduced in the early 1970s. [36] As in the assist control mode, the patient may breathe at a rate faster than the set ventilator frequency. With IMV, however, when the patient initiates a spontaneous breath, the machine offers no assistance, and the patient receives only the $V_T$ that is self-generated. This is usually smaller than the $V_T$ delivered by the ventilator. As in the assist control mode, the patient may determine the minute volume in part. An advantage of IMV is that there is less tendency for the patient to hyperventilate spontaneously. Also, because the patient must work harder to generate a spontaneous breath, it is believed that IMV may help to maintain the tone of the respiratory muscles. An additional potential advantage of IMV over assist control and controlled ventilation is that mean airway pressures tend to be lower in the IMV mode, resulting in less impedance to return of blood to the right side of the heart. [13] (See Complications of Mechanical Ventilation later in the chapter.) The IMV mode is particularly well suited for weaning a patient from mechanical ventilation. Over a period of hours to days, the IMV rate can be turned down gradually, so that the patient makes a smooth transition from depending mainly on the ventilator to breathing entirely independently. Some researchers have claimed that the use of IMV facilitates weaning in difficult patients, [36] although others have questioned the assertion that it speeds the weaning process. [37]

Older ventilators, such as the Puritan-Bennett MA-1, do not have IMV circuits built into them, but they may be converted to the IMV mode through the use of additional tubing and an independent $O_2$ source. This system wastes $O_2$, because the patient may breathe only a small fraction of the air-$O_2$ mixture that is continuously flowing through the IMV circuit. Newer ventilators have IMV circuits built into them so that gas flows through the IMV circuit only when the patient initiates a breath. Another problem that occurred with older IMV systems was that the ventilator sometimes delivered a $V_T$ just as the patient had completed a spontaneous inspiration. This so-called *stacking* of a machine breath on top of a spontaneous breath could lead to overdistention of the lungs and dangerously high peak pressures. This problem has been overcome by the

development of synchronized IMV (SIMV). This mode is the same as IMV except that the ventilator times the machine breaths to fall in a pause in the patient's spontaneous respiratory cycle or to coincide with the initiation of a spontaneous breath.

**Pressure Support**

This mode of ventilation, which was the basic mode on the old intermittent positive-pressure breathing machines, has been reintroduced, with some refinements, on some of the newer ventilators such as the Siemens Servo 900C. When the patient initiates a breath, the ventilator provides a constant inspiratory pressure, until inspiratory flow drops to 25% of the peak level. The patient determines the rate, and $V_T$ depends on the patient's compliance and the inspiratory pressure set by the operator. If the patient's compliance goes down, the $V_T$ will go down. The pressure support mode is obviously not desirable in patients with fluctuating ventilatory drive or pulmonary mechanics. It has been found useful, however, in patients with stable pulmonary disease and in patients being weaned from mechanical ventilation. In such patients, pressure support results in better patient comfort, slower spontaneous ventilatory rates, less patient work, and lower peak airway pressures than the SIMV mode. [38]

# PEEP

The acronym PEEP is used to denote positive end-expiratory pressure in a patient who is receiving assisted ventilation. In this mode, airway pressure is positive not only at end expiration but also throughout the respiratory cycle. When using PEEP, the ventilator can be in the control, assist control, IMV, SIMV, or pressure support modes. In older ventilators, application of PEEP involved cumbersome extra circuitry. In newer machines, the capability for PEEP is built in, and the level is set merely by turning a knob.

Theoretically, PEEP improves oxygenation by keeping alveoli open during expiration. Whether it actually works by this mechanism is a moot point. [39] What is known is that it leads to improved oxygenation and narrowing of the A-a$_{DO2}$ in most patients.

A generally accepted indication for PEEP is failure to achieve adequate oxygenation (i.e., Pa$_{O2}$ <60 mm Hg) with safe levels of inspired $O_2$ (i.e., Fi$_{O2}$ = 0.50). Other indications are more controversial. There is a prevalent philosophy in the surgical literature that PEEP has prophylactic value in preventing the respiratory distress syndrome or that it hastens its resolution. [40] One basis for this belief is that PEEP alters the appearance of infiltrates on chest films, making them appear less dense. In one study, skilled radiologists interpreted pre- and post-PEEP chest films differently in 25% of cases after patients had been on 10 to 20 cm $H_2$ O PEEP for only 15 minutes. [41] Thus some surgical intensive care units routinely use low levels of PEEP (5 to 8 cm $H_2$ O) in patients who require postoperative ventilation but who are not hypoxemic on safe levels of Fi$_{O2}$ . The prophylactic and therapeutic value of PEEP has been questioned in the medical literature, however. [42] In one large retrospective study, use of PEEP did not seem to improve survival in critically ill patients. [43] Animal studies designed to assess the influence of PEEP on pulmonary edema have shown either no change or an

increase in lung water after institution of PEEP. [44]

Determination of the optimal level of PEEP is also controversial. A conservative approach is to use a level of PEEP that is just enough to provide adequate arterial oxygenation with an $Fi_{O2}$ <50%. In patients requiring higher $Fi_{O2}$ levels, PEEP may be started at 5 cm $H_2O$ and may be increased in increments of 3 to 5 cm $H_2O$ at 15-minute intervals. At each new level, pulse and blood pressure, static compliance, peak airway pressure, arterial blood gas tension, and cardiac output should be measured and recorded. The pulmonary artery wedge pressure (PAWP) should probably also be measured, although it may not accurately reflect left ventricular filling pressure in patients on PEEP. [42] The $Fi_{O2}$ can usually be gradually turned down as PEEP is increased and the $A\text{-}a_{DO2}$ narrows. [45]

Most patients exhibit a fall in cardiac output at levels of PEEP above 12 to 15 cm $H_2O$. This drop is due in part to decreased venous return and can be at least partially overcome by expansion of intravascular volume. [46] Diminished myocardial blood flow at higher levels of PEEP may also contribute to diminished cardiac output. [47] A useful method of determining when a fall in cardiac output negates the effect of a rise in $Pa_{O2}$ is to calculate the peripheral $O_2$ delivery at different levels of PEEP. [45] [48] One can calculate the peripheral $O_2$ delivery by multiplying the cardiac output by the arterial $O_2$ content as follows:

$O_2$ delivery to periphery = cardiac output × arterial $O_2$ content [11]

One calculates the arterial $O_2$ content by using equation 12 (shown below). The $O_2$ content C (expressed in milliliters of $O_2$ per 100 mL of blood) equals the product:

$C = 0.003 \times PO_2 + 1.34 \times SO_2 \times Hb$ [12]

Here, $SO_2$ is the $O_2$ saturation (available on a blood gas reading) estimated for a given $PO_2$ and Hb is the patient's hemoglobin level (expressed in grams per deciliter).

Measurement of static compliance also has been advocated as a means of determining the optimal level of PEEP. One small study found that the best PEEP from the point of view of $O_2$ delivery to the periphery coincided with the level at which static compliance was highest, usually in the range of 6 to 12 cm $H_2O$. [49] Other researchers have questioned the reproducibility of this association.

**Dual Synchronous Ventilation**

In some patients with severe unilateral lung disease, adequate oxygenation, ventilation, or both, cannot be maintained with safe levels of $Fi_{O2}$ and reasonable pressures. In such patients, PEEP may actually worsen ventilation perfusion mismatch by causing overdistention of the more normal lung and shunting of blood flow to the more diseased lung. A novel approach to ventilating such patients is to use a different ventilator, with different $V_T$ values and pressures, for each lung. Dual-lumen endotracheal tubes are available for this purpose, and some ventilators, such as the Servo 900C, are

specifically designed to be used synchronously with another twin ventilator. Patients with unilateral lung disease due to pneumonia and bronchopleural fistulas have been ventilated successfully with this technique. [50]

## Characteristics of Inspiratory and

## Expiratory Flow

On modern ventilators, many variables of inspiratory and expiratory flow can be altered by the operator. These include inspiratory flow waveform, inspiratory flow rate, inspiratory time, inspiratory-to-expiratory (I:E) ratio, peak inspiratory pressure, and expiratory resistance. These variables, along with rate and $V_T$, are interrelated, so that a change in one affects the others. Which variable is set by the operator and which variable is secondarily determined depends on the ventilator design. For example, in a pressure-cycled ventilator, flow and pressure are set and $V_T$ is determined, whereas in a volume-cycled ventilator, flow and $V_T$ are set and pressure is determined. With a time-cycled ventilator, the I:E ratio is set, whereas with pressure- and volume-cycled ventilators, the I:E ratio is determined. Although changes in the characteristics of inspiratory and expiratory flow tend to result in relatively small changes in the final measures of oxygenation and $CO_2$ elimination, by adjusting these variables appropriately, one can fine-tune the ventilator to optimize the therapy of critically ill patients.

### Inspiratory Flow

The basic flow pattern built into most ventilators is a constant flow (square wave). In many modern ventilators, this basic pattern can be altered by the operator to produce an accelerating (tapered wave) pattern. In most cases, a square wave is chosen as the initial setting, since this leads to lower peak pressures for a given inspiratory time and $V_T$. In some patients with very uneven ventilation of different regions of the lungs, however, use of a tapered inspiratory waveform may result in more even ventilation by allowing more time for the inspired gas to pass through airways with increased resistance to flow. [51]

### Inspiratory Flow Rate

In volume- and pressure-cycled ventilators, the inspiratory flow rate is initially set in the range of 40 to 50 L/min in adults and 8 to 15 L/min in infants. In time-cycled ventilators, one may secondarily determine the inspiratory flow by setting the $V_T$ and inspiratory time. An inspiratory flow rate that is too rapid may lead to dangerously high peak pressures or unequal ventilation of lung units with different time constants, whereas an inspiratory flow rate that is too slow may lead to an undesirably long inspiratory time with inadequate time for exhalation.

### Inspiratory Pause

Some ventilators allow the operator to add a pause to the end of inspiration, during

which time airway pressure is held constant and the patient cannot exhale. Like other maneuvers that prolong the inspiratory phase, an inspiratory pause leads to more even ventilation. [51] The pause may also lead to air trapping. An inspiratory pause is also analogous to PEEP in that by increasing mean airway pressure, it may lead to decreased venous return and decreased cardiac output. An inspiratory pause is usually not initially prescribed, but it may be added when hypoxemia due to uneven ventilation is a problem.

**Inspiratory-to-Expiratory Ratio**

To allow complete exhalation, the expiratory phase of the ventricular cycle should usually be at least twice the length of the inspiratory phase. Higher I:E ratios may lead to air trapping, overdistention, and high peak pressures. It was found in the late 1970s, however, that in infants with hyaline membrane disease, so-called inverse ratio ventilation (IRV) with I:E ratios of 2:1 improved oxygenation and reduced the time infants had to be on high inspired $O_2$ concentrations and PEEP. [52] Inverse I:E ratios as great as 4:1 have also been used in adults with severe hypoxemia due to adult respiratory distress syndrome (ARDS), pneumonia, and cardiogenic pulmonary edema. Inverse ratio ventilation is similar to PEEP in that it improves oxygenation, increases mean airway pressure, reduces $V_D/V_T$ and pulmonary shunt, and may reduce cardiac output. It leads to lower peak pressures than PEEP, however, with less risk of barotrauma. The mechanism by which IRV achieves its beneficial effects is not known for certain, but it is theorized that the long inspiratory time may lead to more even ventilation, and the short expiration time may prevent collapse of diseased alveoli.

**Peak Pressure**

With pressure-cycled ventilators, peak pressure is a set variable and determines $V_T$. Starting pressures of 20 to 25 cm $H_2 O$ are commonly used in adults with normal compliance, although pressures 40 cm $H_2 O$ are usually required in patients with a pathologic pulmonary condition.

With time- and volume-cycled ventilators, peak pressure is determined mainly by $V_T$. Typical peak pressures for patients with respiratory failure are in the range of 40 to 50 cm $H_2 O$. Peak pressures >60 cm $H_2 O$ are not uncommon but are associated with a higher incidence of barotrauma. In exceptional cases, peak pressures of 100 cm $H_2 O$ are required. Modern volume- and time-cycled ventilators have

adjustable peak pressure limits such that when the airway pressure exceeds the set limit, inspiration is terminated. The limit is usually set at 10 cm $H_2 O$ above the peak pressure that is observed when the patient is initially placed on the ventilator. If the peak pressure alarm sounds later, it is a sign that the patient's compliance has dropped or that resistance has increased, and the cause must be sought. Typical causes of acute increases in peak pressure include attempts by the patient to override, or "buck," the ventilator, development of pneumothorax, migration of the tip of the endotracheal tube into the right main stem bronchus, kinking of the tube, or plugging of the tube or a major airway.

**Expiratory Retard**

Many ventilators have an adjustable expiratory resistance, or retard. The development of such a mechanism was inspired by the observation of COPD patients who breathe through pursed lips, apparently in an effort to increase their own expiratory resistance. Theoretically, this resistance to expiration may prevent the premature collapse of small airways and may paradoxically lead to more complete exhalation. Whether this is actually the reason for pursed-lip breathing and whether expiratory retard is a useful setting on a ventilator remain controversial.

**Sighs**

In the early 1960s, it was shown that patients who were being mechanically ventilated during anesthesia showed a progressive decline in compliance and widening of their A-a$DO_2$ . [56] These changes were thought to be caused by atelectasis and were reversed by intermittent deep breaths. Based on this work, "sigh" functions have been built into mechanical ventilators, allowing the operator to introduce a breath 1.5 to 2.0 times the usual $V_T$ at regular intervals. A problem with the original study that demonstrated the benefit of "sighs," however, was that patients were ventilated with low $V_T$ levels. With the $V_T$ levels that are used today, which are in the range of 10 to 15 mL/kg, it has not been demonstrated that progressive atelectasis occurs. The optimal rate and volume of sighs are unknown, nor is it known whether sighs are beneficial. Indeed, it has been argued that incorporation of a sigh function into a ventilator adds only needless extra expense. [57]

**Permissive Hypercapnia**

The concept of permissive hypercapnia represents an alternate approach to conventional mechanical ventilation concepts. While $V_T$ values of 10 to 15 mL/kg (regardless of the inspiratory pressures required) are usually recommended as initial settings, high peak pressures have been shown to produce lung injury in numerous settings (e.g., ARDS, asthma). The avoidance of "volutrauma" by pressure or volume limitation theoretically prevents ventilation-induced lung injury. With permissive hypercapnia a lower $V_T$ of 5 to 8 mL/kg is used to prevent excessive alveolar distention, but the tradeoff is a rise in $Pa_{CO_2}$ and a decrease in serum pH. Both hypercapnia and acidemia are apparently well tolerated in critically ill patients. Although previously believed to be detrimental, good outcomes have been described despite previously "unacceptably high" $Pa_{CO_2}$ . [58] [59] Some authors advocate adjusting the serum pH with sodium bicarbonate whereas others also allow a mild "permissive acidemia." [59]

Permissive hypercapnia allows arterial $p_{CO_2}$ to rise above 50 to 60 mm Hg. Using low $V_T$ values and peak inspiratory pressures <40 cm $H_2$ O, Hickling and colleagues  report significant decreases in mortality rates in patients with ARDS whose mean $Pa_{CO_2}$ was in the range of 62 to 66 mm Hg and mean serum pH was 7.23 (range, 6.8 to 7.45). Neuromuscular blockade is usually required to achieve pressure-volume limitation. Permissive hypercapnia is easy to perform and appears free of complications, but it is contraindicated in those with intracranial hypertension in which a high $p_{CO_2}$ may further

elevate intracranial pressure.

## Humidification

Inspired air is normally humidified and warmed in the oropharynx and the nasopharynx before reaching the lower airway. In an intubated patient, inspired air bypasses the nasopharynx and the oropharynx and is injected directly into the trachea. To prevent drying of the mucosa of the lower airways, all modern ventilators are equipped with systems for humidifying and warming inspired air. The setup of these systems varies from ventilator to ventilator and is described in the operating manuals supplied by the manufacturers. A commonly overlooked setting, however, is the temperature of the inspired air. Normally, the air temperature should be 35 °C at the point at which the gas enters the endotracheal tube. One should remember that in patients who are hypothermic, heating the inspired air is an adjunctive means of core rewarming. [9] Likewise, cooling the inspired air will lower the body temperature in febrile patients.

## Medication Nebulizers

Most modern ventilators have in-line medication nebulizers for the administration of bronchodilator drugs. The dosages are the same as for medication nebulizers for patients who are breathing spontaneously. Special mention should be made of ribavirin, a synthetic nucleoside analogue approved for aerosol treatments of infants and young children with severe respiratory syncytial virus infections. Ribavirin has been reported to cause malfunction of ventilators because of precipitation of the drug on the valves and in the ventilator tubing. Special filters must be used when this drug is delivered via the nebulizer on a mechanical ventilator

# VENTILATOR USE

## Specific Situations Requiring Ventilation

### Acute Respiratory Distress Syndrome

An acceptable arterial oxygen saturation (i.e., 90%) should be targeted in patients with ARDS, but the $Fi_{O2}$ should be minimized (0.6) whenever possible to decrease $O_2$ toxicity. A plateau pressure of 35 cm $H_2O$ is of concern, and it may be desirable to decrease $V_T$ to as low as 5 mL/kg to alleviate excessive pressures. Such patients may be candidates for permissive hypercapnia. While PEEP is quite useful in ARDS because it permits the reduction of $Fi_{O2}$ to less toxic levels, the level of PEEP should be applied judiciously to avoid high peak airway pressure barotrauma. When oxygenation is inadequate, sedation, paralysis, and position change are parameters that should be carefully reviewed. Because a

depression in cardiac output commonly occurs in hypovolemic patients placed on PEEP, volume repletion may be required. Because effects on cardiac output are complex and quite unpredictable, most patients with ARDS require pulmonary artery

catheter placement to assess hemodynamic parameters.

**Severe Acute Asthma**

Mechanical ventilation of the severe asthmatic is both difficult and hazardous. A major concern is ventilator-induced hyperinflation due to high plateau pressures (35 cm $H_2O$) and high peak airway pressure (PAP) (50 cm $H_2O$). It is therefore desirable to minimize plateau pressures and PAP in patients with asthma. Increasing the lumen of the tracheal tube may help to minimize peak airway pressures, and other approaches specific to mechanical ventilation in acute asthma are outlined in Table 7-4 (Table Not Available) . The clinician should aim to decrease minute ventilation and increase expiratory time. In many instances, it is permissible to accept an elevated $Pa_{CO_2}$ as long as pH can be maintained in an acceptable range. Paralysis and/or sedation are usually necessary to effectively achieve ventilatory goals in severe asthma.

## Choosing a Ventilator

With the large variety of commercial ventilators available today, choosing the best ventilator for one's hospital or hospital department can be a difficult decision. One factor in deciding which ventilator is best is the setting in which the ventilator will be used. In an emergency department in which mechanical ventilation is done infrequently and for short periods, a relatively simple ventilator that is easy to set up and to operate may be preferable to a more complex machine that offers more controls and monitors. Although some ventilators, such as the Siemens Servo 900C, can be used for both infants and adults, ventilators designed specifically for infants are usually preferred in pediatric and neonatal intensive care units. Safety and reliability are major concerns in choosing a ventilator. Fortunately, most modern ventilators have both features. Cost is another factor to be considered. The improvements in ventilator technology since the mid-1980s have not come cheaply. Many ventilators today cost more than $20,000. Table 7-5 (Table Not Available) lists some features that are most desirable in a ventilator and some other features that may add significantly to the cost but may not be as necessary.

## Ventilator Setup

The actual setup of a ventilator is usually done by a respiratory therapist under the direction of a physician. In some cases, a respiratory therapist or other knowledgeable nonphysician may not be available immediately, and the physician may need to do the actual ventilator setup. Unfortunately, it is not practical to describe in this chapter the step-by-step setup of every ventilator currently in use. Instead, setup of a state-of-the-art infant ventilator, the Infant Star, and the versatile Siemens Servo 900C, which can be used in both infants and adults, is described. The descriptions of ventilator setup that follow are not intended to provide everything one needs to know to operate either of these ventilators safely. Rather, these descriptions are intended to

serve as examples of the typical steps in the setup of modern ventilators. For complete instructions regarding the setup of these or other ventilators, the operator should consult the operating manuals supplied by the manufacturers. Copies of these manuals should

be available in any departments in which the ventilators are in use.

If at any point during ventilator setup the patient appears to have inadequate ventilation, the patient should be taken off the ventilator and should be bag ventilated. Troubleshooting of ventilator setup should *not* occur while the patient is dependent on the ventilator.

**Setup of the Infant Star**

The Infant Star is a time-cycled ventilator, designed especially for mechanical ventilation of infants and children. The actual ventilator is shown in Figure 7-4 (Figure Not Available) and the control and display panels are depicted schematically in Figure 7-5 (Figure Not Available) . The ventilator is controlled by 2 microprocessors that allow precise tailoring of ventilator settings and provide alarms and monitors not found on most other pediatric ventilators. The ventilator may be connected by a standard computer cable to an IBM-compatible personal computer for making printouts of monitored parameters and trends. The ventilator software can be updated, and high-frequency ventilation is an enhancement that is currently available.

Two modes are basically available on the Infant Star--CPAP and IMV. With either mode, continuous or demand flow is possible. Demand flow conserves the hospital $O_2$ supply with minimal, if any, increase in the work of breathing as a result of the rapid response time of the microprocessor-controlled inspiratory flow valves. Setup in the IMV mode with demand flow is described here.

**Step-by-step setup.**

1. Air, $O_2$ , and electrical power hookups at the back of the ventilator are connected to hospital sources.
2. Inspiratory and expiratory arms of patient breathing tubes are connected to the ports labeled TO PATIENT and FROM PATIENT on the compressor front panel. (Note that a separate humidifier must be used.) Special 1/8-inch tubing is connected from the patient Y to the PROXIMAL AIRWAY PRESSURE port.
3. The power switch at the back of the ventilator is turned on, and the operator checks that all digital displays are illuminated and bright, that an audible alarm sounds momentarily, and that all light-emitting diode indicators are illuminated, except POWER LOSS, VENT INOP, and INTERNAL BATTERY.
4. Ventilator settings are made by turning the appropriate knobs on the control panel and compressor. Although the appropriate settings will be determined by the clinical situation, typical settings for an infant would be FLOW RATE 20 L/min, RATE 30 breaths/min, PEAK INSPIRATORY PRESSURE 50 cm $H_2$ O, INSPIRATORY TIME 1 second, and LOW INSPIRATORY PRESSURE 10 cm $H_2$ O.
5. Connect the patient Y to a test lung to check inflation and deflation at the appropriate rate.
6. Disconnect the patient Y from the test lung and check that the LOW INSPIRATORY PRESSURE ALARM is activated. Obstruct the tube at the patient Y to be sure that the OBSTRUCTED TUBE alarm is activated. Partially obstruct

the patient Y and rotate the pop-off valve on the front of the compressor until gas is felt to escape from the valve at a pressure of about 60 cm $H_2O$ as read off the analogue pressure gauge. Reconnect the patient Y to the test lung and push the ALARM SILENCE and VISUAL RESET buttons.

7.  Set the desired $Fi_{O2}$ on the front of the ventilator compressor.
8.  Connect the ventilator to the patient and observe for rise and fall of the patient's chest and appropriate pressures, I:E ratio, and expiration time. Readjust the PEAK INSPIRATORY PRESSURE to 5 to 10 cm $H_2O$ above the observed peak inspiratory pressure and the LOW INSPIRATORY PRESSURE to 5 to 10 cm $H_2O$ below the observed peak inspiratory pressure. Readjust the pop-off valve to 13 cm $H_2O$ above the set peak inspiratory pressure limit.

**Alarms and monitors on the Infant Star.**

The Infant Star has 9 alarm systems plus an indicator to signal that the ventilator is running on its internal battery if the outside power source fails. All alarms are both audible and visual, except for a portion of the OBSTRUCTED TUBE alarm. Alarm lights are located on the lower right-hand corner of the ventilator control panel (see Fig. 7-5) (Figure Not Available) . Audible alarms can be silenced for 60 seconds by pushing the ALARM SILENCE button, with the exception of the VENTILATOR INOPERATIVE and POWER LOSS alarms, which cannot be silenced.

**LOW INSPIRATORY PRESSURE.**

This alarm is activated when the peak inspiratory pressure of a given breath does not reach the set alarm limit. By setting the alarm limit within 5 to 10 cm $H_2O$ of the observed peak inspiratory pressure, the alarm will detect any significant leaks in the inspiratory circuit. Setting the alarm limit closer to the observed peak inspiratory pressure will lead to frequent false alarms.

**LOW PEEP/CPAP.**

The microprocessor automatically sets the low PEEP and CPAP limits to 2 to 5 cm $H_2O$ below the set PEEP and CPAP levels. The alarm is activated if PEEP or CPAP falls below the automatic limits.

**AIRWAY LEAK.**

This alarm is activated when demand flow exceeds background flow for 4 seconds. It supplements the low inspiratory pressure and low PEEP/CPAP alarms.

**OBSTRUCTED TUBE.**

This alarm is activated when peak inspiratory pressure or PEEP or CPAP significantly exceeds the set levels, usually indicating an obstructed breathing tube. The alarm is actually sensitive to 5 different specific violations, and the exact violation is displayed in

coded form in the window above the alarm lights. Explanations of the 5 different codes and the most likely malfunction associated with each alarm code are described in detail in the operating instructions provided with the ventilator.

**INSUFFICIENT EXPIRATORY TIME.**

This alarm is activated when exhalation time for mandatory breaths is <0.3 seconds.

**LOW O$_2$ PRESSURE and LOW AIR PRESSURE.**

These alarms are activated when the pressure of gases into the ventilator drops below 45 psi. Below this pressure, the blender may be affected, and inaccurate F$_{IO2}$ settings may result.

**POWER LOSS and INTERNAL BATTERY.**

The internal battery light comes on whenever outside power fails and the ventilator is running on its internal battery. Approximately 5-10 minutes before the internal battery is exhausted, the internal battery and power loss lights alternately flash. When fully charged, the internal battery provides up to 30 minutes of operation.

**VENTILATOR INOPERATIVE.**

This alarm signals a major malfunction, which makes the ventilator unsafe. When this alarm activates, the ventilator shuts down and all valves open, allowing the patient to breathe ambient air. Possible causes of ventilator malfunction are detailed in the operating instructions.

Monitors on the Infant Star include an analogue proximal airway pressure manometer; digital displays for PEEP and CPAP, mean airway pressure, and peak inspiratory pressure; and a digital display with a 3-way switch that may be turned to read PEEP/CPAP, I:E RATIO, or EXPIRATION TIME. The monitors are for the most part self-explanatory. The MEAN AIRWAY PRESSURE monitor is a particularly useful feature of the Infant Star, because mean airway pressure has a direct bearing on the risk of barotrauma and on oxygenation and cardiac output.

**Setup of the Siemens Servo 900C**

The Siemens Servo 900C is a compact yet versatile and dependable modern ventilator, which operates almost silently. With its many accessories, it can be used as a ventilator for infants, children, or adults; as an anesthesia machine; or as a diagnostic instrument. Although it is basically a time-cycled ventilator, volume and pressure limits can be set to make it function like either a volume-cycled or a pressure-controlled machine.

The modes available on the Siemens Servo 900C deserve special mention, because they are somewhat different from those available on most other ventilators. In the

VOLUME CONTROL mode, the ventilator functions much like a typical volume-cycled ventilator, except that the operator sets the desired minute volume rather than the desired $V_T$. The

operator then sets the desired respiratory rate and I:E ratio, and through its internal electronic circuitry, the ventilator determines the inspiratory flow necessary to deliver the set minute volume. A VOLUME CONTROL PLUS SIGH mode, in which every 100th breath is delivered with double the basic $V_T$, is also available. By adjusting the TRIGGER SENSITIVITY appropriately, the VOLUME CONTROL mode becomes analogous to "assist control" in a volume-cycled ventilator. In the PRESSURE CONTROL mode, the ventilator delivers a constant pressure, with a decelerating flow pattern, during a set inspiratory time. The inspiratory pressure, respiratory rate, and inspiratory time determine the $V_T$ received by the patient. PRESSURE SUPPORT is a unique mode in which the patient must initiate all breaths independently, but assistance is provided by positive pressure from the ventilator each time a sufficient inspiratory effort is made. The $V_T$ of each breath depends in part on the set inspiratory pressure and in part on the amount of effort made by the patient. This mode is suggested by the manufacturer for weaning patients from anesthesia or from mechanical ventilation.

The SIMV mode is the same as that in a volume-cycled ventilator except the minute volume rather than $V_T$ is set. SIMV PLUS PRESSURE SUPPORT combines these 2 modes, providing the patient with a set minute volume at a set rate but allowing the patient to breathe spontaneously between machine breaths, with the ventilator applying positive pressure during the spontaneous inhalations. The CPAP mode is the same as that in a volume ventilator. A MANUAL mode is also available for providing manual ventilation during the administration of anesthesia or while a patient is being suctioned.

The external appearance of the Servo 900C is shown in Figure 7-6 (Figure Not Available) . The assembly of the ventilator to this point as well as the attachment of the humidifier, the $O_2$ -air mixer, and the patient tubing are discussed in detail in the manufacturer's instruction manual. The power supply cord (which must be plugged into a standard 110-volt AC outlet) and the on/off switch are located at the rear of the electronic unit. The further setup of the ventilator is illustrated in Figure 7-7 (Figure Not Available) and is discussed here. Setup is described for the ASSIST CONTROL mode, because this is the mode that is most appropriate for initial ventilation of most adult patients in the emergency department or the critical care unit. The setup in other modes varies slightly and is covered in the manufacturer's manual.

**Step-by-step setup.**

1. The WORKING PRESSURE adjustment on the pneumatic unit, which is mounted above the electronic unit, determines the maximum pressure that the ventilator will deliver and provides a secondary safeguard against the inadvertent exposure of the patient to dangerously high pressures. (The primary safeguard is the UPPER PRESSURE LIMIT setting on the ventilator control panel, described in step 12.) The recommended working pressure for most adult patients is 60 cm $H_2$ O. Higher pressures, up to 120 cm $H_2$ O, may be required in rare cases.
2. The mode selector switch may be turned to either VOLUME CONTROL or

VOLUME CONTROL PLUS SIGH. As discussed previously, the sigh function is not recommended.

3. The PRESET INSPIRATORY MINUTE VOLUME control determines the minute volume that the ventilator will deliver if the patient initiates no breaths independently. As discussed previously, not all of the volume that is delivered by the ventilator actually reaches the patient, beause

    some is lost to compression within the humidifier and the patient tubing. The difference between the preset inspiratory minute volume and the minute volume that the patient actually receives is on the order of 400 to 600 mL/min for a typical adult patient. A method of calculating the exact difference is given in the operating manual.

4. Either a square wave or an accelerating inspiratory flow pattern may be selected. In most cases, a square wave is the appropriate initial setting (see discussion in the section on inspiratory flow).

5. The BREATHS/MINUTE knob determines the minimum number of breaths that the patient will receive. One determines the $V_T$ of each breath by dividing the preset inspiratory minute volume by the number of breaths per minute. The patient may trigger the ventilator at a faster rate than the breaths per minute setting, in which case the patient will receive more than the preset inspiratory minute volume. The $V_T$ of the ventilator breaths will not change, however.

6. 6 and 7. The INSPIRATORY TIME PERCENT setting determines the fraction of each respiratory cycle that is spent in inspiration. The PAUSE TIME PERCENT setting may be used to provide a pause at the end of inspiration that is up to 20% of the respiratory cycle. In combination, the inspiratory time percent and pause time percent settings determine the I:E ratio, which may be set anywhere from 1:4 to 4:1. The recommended initial setting is an inspiratory time of 33% with no pause, resulting in an I:E ratio of 1:2. (See the sections on inspiratory pause and I:E ratio for a discussion of the use of an inspiratory pause and different I:E ratios.)

7. The inspired $O_2$ concentration is set by a control on the air-$O_2$ mixer attached to the right side of the ventilator.

8. The UPPER and LOWER ALARM LIMIT controls should be set approximately 6% above and below the desired $O_2$ concentration. When the $F_{IO_2}$ varies outside the set limits, as detected by the internal $O_2$ analyzer on the ventilator, visible and audible alarms are activated.

9. The PARAMETER SELECTOR knob determines which parameter is displayed in the digital readout window on the control panel above the knob. The available parameters include breathing rate (sum of spontaneous and mechanical), actual $FIO_2$ , inspiratory $V_T$, expiratory $V_T$, expired minute volume, peak pressure, pause pressure, and mean airway pressure.

10. If PEEP is required, one sets the desired level by turning the PEEP knob.

11. The UPPER PRESSURE LIMIT knob allows the operator to set a ceiling for airway pressure above which inspiration is terminated, and visible and audible alarms are activated. The limit should be set at approximately 50 cm $H_2O$ initially and should be readjusted with the patient on the ventilator to 10 to 15 cm $H_2O$ above the observed peak pressure. If the upper pressure limit is set above the working pressure (see step 1), inspiration will end when the working pressure is reached, but no alarms will be activated.

12. The TRIGGER SENSITIVITY control determines how much inspiratory effort the patient must make to trigger an assisted ventilator breath. The sensitivity is usually set so that an inspiratory effort of -1 to -3 cm $H_2O$ is required.
13. The EXPIRED MINUTE VOLUME meter at the top left corner of the ventilator control panel has dual scales: from 0 to 40 L/min for adults and from 0 to 4 L/min for children. The proper scale is selected with the INFANTS/ADULTS switch at the lower left corner of the control panel. This switch also sets the scale for the UPPER and LOWER ALARM LIMIT controls (see step 16).
14. The ventilator is now connected to the patient. In addition to observing the clinical response of the patient, one should use the PARAMETER SELECTOR control and EXPIRED MINUTE VOLUME and AIRWAY PRESSURE meters to be sure that the rate, $V_T$, minute volume, $Fi_{O2}$, and airway pressures are in the desired range.
15. After the patient has been on the ventilator for a few minutes and it has been determined that the patient is receiving an appropriate minute volume, the UPPER and LOWER ALARM LIMIT controls should be set approximately 20% above and below the desired minute volume. When the expired minute volume deviates from this range, visible and audible alarms are activated.

The alarms that are included for patient safety are depicted in Figure 7-8 (Figure Not Available) . Alarms are given in the form of audible signals as well as flashing red lights. One can switch off most of the audible alarms for 2 minutes by depressing the ALARM SILENCE button on the control panel.

In addition to the features discussed previously, available options on the Servo 900C include a $CO_2$ monitor, which analyzes end-tidal $CO_2$ and has alarms for levels of $CO_2$ above or below set limits; a lung mechanics calculator, which calculates airway resistance and compliance; and a paper strip recorder, which can print pressure-flow curves or graphically present the digital displays from the ventilator control panel.

**Sedation and Paralysis**

Whereas many patients adapt readily to mechanical ventilation and synchronize their own breathing with the ventilator breaths, other patients "fight the machine." By coughing, bucking, and breathing out of phase with the ventilator, they generate high peak airway pressures and increase their $O_2$ consumption and $CO_2$ production. When their airway pressures exceed the peak pressure limits, they receive less than the prescribed $V_T$, and hypoventilation results. In such cases, *a complication or a mechanical problem must first be ruled out*. One should check the inspired $O_2$ concentration with an $O_2$ analyzer to be sure that the set $Fi_{O2}$ is really being delivered. While the patient is being "bagged" by hand, increased resistance to inflow of air, suggesting a plugged or kinked endotracheal tube, can be sensed. Malposition of the endotracheal tube and pneumothorax should be ruled out by auscultation and a chest film. The patient should be suctioned to remove any large airway obstruction due to mucus, blood, or other debris. Other causes for agitation, such as hypotension or pain, should be considered. If coherent, the patient should be reassured.

When all of these measures have been taken, no complication or malfunction has been

found, and the patient continues to fight the ventilator, sedation should be considered. Diazepam is a useful drug in this setting. Diazepam acts rapidly and provides excellent relaxation, sedation, and amnesia. The usual starting dose in adults is 2.5 to 5.0 mg IV, given at a rate of 2.5 mg/min. Because the main side effect is respiratory depression, much larger doses, up to 1 mg/kg, can be given in a mechanically ventilated patient, although some cardiac depression occurs at very high doses (>3 mg/kg).

An alternative to diazepam is morphine sulfate. Morphine is usually given in 2- to 4-mg increments IV and titrated to effect. As with diazepam, the main side effect of morphine is respiratory depression, which is not a problem in the mechanically ventilated patient with a secure airway. The drug also causes a small drop in blood pressure, probably a result of peripheral vasodilation rather than a direct cardiac depressant effect. [64] Morphine is also known to cause histamine release, which could theoretically lead to increased bronchospasm. [64] Whether this effect is of clinical significance is unknown. Advantages of morphine over diazepam are that it is a potent analgesic and its effect is readily reversible with naloxone.

When sedation and analgesia are ineffective in preventing the patient from fighting the ventilator, a paralyzing drug may be used. Pancuronium bromide is the drug of choice for inducing paralysis in ventilator patients. Pancuronium is a nondepolarizing blocker of neuromuscular transmission. The main side effects of the drug are a mild increase in pulse and blood pressure, although it has also been reported to cause severe hypertension, ventricular dysrhythmias, and anaphylactic reactions on rare occasions (also see Chapter 3) .

Pancuronium is preferred over *d*-tubocurarine, which commonly causes hypotension, and the depolarizing agent succinylcholine, which is short acting and causes fasciculations and cholinergic side effects. [65] The dose of pancuronium is 0.02 to 0.06 mg/kg IV. Paralysis occurs within 1 to 3 minutes and lasts 1 to 2 hours, after which time repeated doses may be given. Paralysis induced by pancuronium can be reversed by neostigmine, 0.06 to 0.08 mg/kg IV up to a total of 2.5 mg. Physostigmine should *not* be used for reversal, because it crosses the blood-brain barrier and may induce seizures. Atropine, 0.01 to 0.02 mg/kg up to a total of 1 mg, should be given in the same syringe to block the cholinergic side effects of neostigmine. Paralysis with pancuronium has been reported to be particularly effective in asthmatics. [66] This agent is also used frequently in neonates with hyaline membrane disease, in whom it has been shown to increase compliance and $Pa_{O_2}$ . [67]

It is important to remember that although a patient who is paralyzed with pancuronium or other newer agents (see Chapter 3) may appear asleep and calm, the drug has no sedative or analgesic properties. A paralyzed patient must be given liberal doses of sedatives and analgesics at regular intervals. Hospital personnel should treat the patient as if fully awake, talking to the patient in a reassuring manner and avoiding bedside discussion of the case. Finally, the patient must be continually observed and ventilator function and alarms must be checked frequently, because the patient will be entirely unable to breathe independently should the ventilator fail.

# COMPLICATIONS OF MECHANICAL VENTILATION

In a prospective study of 354 episodes of mechanical ventilation at the University of Colorado from 1972 to 1973, there were 400 complications. [68] Although most were minor, some were associated with increased patient mortality. It is probably not surprising that there is a high incidence of complications with mechanical ventilation, because it is an invasive form of therapy using complex equipment in critically ill patients for prolonged periods. To some extent, however, the complications are preventable. Others that are not preventable at present should at least be anticipated so that when they occur, they can be recognized and dealt with promptly.

The potential complications of ventilator therapy are listed in Table 7-6 . Complications related to intubation and the presence of an endotracheal tube are dealt with in more detail elsewhere in this text but deserve mention here. Intubation

| **TABLE 7-6** -- Potential Complications of Mechanical Ventilation |
| --- |
| Complications related to endotracheal or tracheostomy tube |
| Tube malfunction (leaking cuff, kinked tube, obstruction caused by herniation of balloon over end of tube) |
| Pressure phenomena (nasal and tongue necrosis due to pressure from the tube, laryngeal ulceration and polyps, tracheal stenosis and malacia, fistulas into esophagus and innominate artery) |
| Complications resulting from machine malfunction and operator error |
| Failure of ventilator to deliver set tidal volume, rate, $Fi_{O_2}$ , etc |
| Inappropriate settings ordered |

| |
|---|
| Settings not fixed as ordered (commonly includes wrong rate, $V_T$, and $F_{IO_2}$ ; assist control sensitivity too high or too low) |
| Alarm failure |
| Alarm turned off and left off |
| Inadequate humidification |
| Overheating or underheating of inspired air |
| Patient accidentally disconnected from ventilator |
| Direct effects of positive-pressure ventilation |
| Barotrauma (pneumothorax, tension pneumothorax, pulmonary interstitial emphysema, subcutaneous emphysema, pneumomediastinum, pneumoperitoneum, air embolism) |
| Decreased venous return and cardiac output |
| Other complications |
| Ventilator-associated pneumonia |
| Oxygen toxicity and retrolental fibroplasia |

of the right main stem bronchus was one of the most common complications in the University of Colorado study and was associated with other problems, including pneumothorax and atelectasis, as well as with decreased survival. Complications related to pressure from the endotracheal tube cuff have declined since the introduction of high-volume, low-pressure cuffs, which require <25 mm Hg to produce a seal. Whereas it was recommended in the older literature that a tracheostomy be performed in patients requiring mechanical ventilation for >1 to 2 weeks, more recent studies have reported fewer complications with soft cuff endotracheal tubes in place for up to 3 weeks than with tracheostomies. [69] There has been one report of orotracheal intubation for 2 months without complications. [70] The safety of orotracheal vs. nasotracheal intubation has not been studied systematically. In general, nasotracheal intubation is better tolerated by the patient, is more secure, and allows better mouth care. On the other hand, it leads to a higher incidence of sinusitis and necrosis of the nasal mucosa and cartilage.

The incidence of complications related to ventilator malfunction has declined as ventilators have become more reliable, but operator errors remain a significant problem. One of the most common yet potentially serious errors is to turn off a ventilator alarm and to forget to turn it back on. This happens most frequently when the patient is disconnected briefly from the ventilator for suctioning. With many older ventilators, such as the Puritan-Bennett MA-1, one can permanently silence the alarm that signals when the patient has failed to receive a full $V_T$ by turning off a single switch. If this alarm is not turned back on after the patient has been suctioned, the patient, by turning the head, may later cause the ventilator tubing to become disconnected from the endotracheal tube. The next indication that something is wrong may be the development of a ventricular dysrhythmia on the cardiac monitor. To prevent this occurrence, many modern ventilators have been designed with alarms that can be turned off by the operator only for 1 to 2 minutes before they reactivate themselves.

Barotrauma remains a relatively common complication of ventilator therapy. Pneumothorax is the most common form of barotrauma and may be preceded or accompanied by pulmonary interstitial emphysema, pneumomediastinum, subcutaneous emphysema, and pneumoperitoneum. In patients receiving ventilatory assistance, pneumothorax is more often than not of the tension type. The reported incidence of pneumothorax in mechanically ventilated patients varies in the literature from 0.5 to 14%. The highest incidence of barotrauma occurs with the use of levels of PEEP >24 cm $H_2O$, [74] whereas levels of PEEP of 5 cm $H_2O$ and below do not seem to be associated with increased risk. [73] The incidence of pneumothorax is also particularly high in patients with asthma. [11]

It has been reported that the incidence of barotrauma is lower with pressure-cycled than with volume-cycled ventilators. [72] This is not surprising when it is considered that higher pressures are commonly used with volume-cycled ventilators. Judicious use of sedation

and paralysis may help reduce pressures and thereby lower the risk of barotrauma. It has been argued that it is safer to allow patients with asthma to remain hypercapnic for periods of hours to days rather than to use peak pressures above 50 cm $H_2O$. [75]

In patients who are on high levels of PEEP or who require high peak pressures, the development of barotrauma should be anticipated. A pneumothorax should be suspected whenever there is a sudden deterioration in compliance and blood gas values. Pneumothorax should be confirmed by auscultation, palpation of the position of the trachea, and, if time permits, a chest film. In the patient whose condition is deteriorating rapidly, needle thoracostomy is both diagnostic and therapeutic (see Chapter 8) , but it should be followed promptly by tube thoracostomy (see Chapter 9) .

Pneumoperitoneum is a form of barotrauma that deserves special mention. The incidence of pneumoperitoneum and pneumoretroperitoneum has been reported to be as high as 4% in patients on PEEP. [76] Pneumoperitoneum is typically preceded by pulmonary interstitial emphysema, subcutaneous emphysema, and pneumomediastinum and is usually, but not always, accompanied by pneumothorax. Patients with ventilator-induced pneumoperitoneum have been subjected to needless laparotomies in a search for the cause of abdominal free air. [77] Another unusual form of barotrauma is fatal arterial and venous air embolism, which has been reported in association with tension pneumothorax in premature infants receiving mechanical ventilation. [78]

Another potential problem that is a direct effect of positive-pressure ventilation is diminished cardiac output. As discussed in the section on PEEP, a clinically significant fall in cardiac output usually occurs at levels of PEEP of 12 to 15 cm $H_2O$. A drop in cardiac output has also been shown to be a result of positive-pressure ventilation without PEEP. [17] The fall in cardiac output seen with mechanical ventilation is thought to be caused by decreased venous return as a result of increased intrathoracic pressure. This effect is greatest when mean airway pressures are highest, as in controlled ventilation with PEEP, and is negligible when low mean airway pressures are used, as in the IMV mode with a low

mechanical breathing frequency. [13] It has been shown in the case of PEEP that the fall in cardiac output is at least partly reversible with blood volume expansion. [46] The influence of positive-pressure ventilation on venous return also depends on the extent to which airway pressure is transmitted to the pleural space. When lung compliance is high and chest wall compliance is low, as in COPD, much of the airway pressure is transmitted to the pleural space, and venous return is impaired more than in conditions such as ARDS, in which lung compliance is low.

It should not be assumed that the effect of positive-pressure ventilation on cardiac function is always detrimental. It has been shown that patients with respiratory distress may generate highly negative intrathoracic pressures during spontaneous breathing and that this negative pressure acts as increased afterload on the heart. [79] Although venous return into the thorax is enhanced by negative intrathoracic pressure, ejection of blood out of the thorax is impeded, and the more negative the intrathoracic pressure, the harder the left ventricle must pump to reach a given systemic arterial pressure. [80]

Mechanical ventilation substitutes positive for negative intrathoracic pressure, thereby decreasing afterload on the left side of the heart. In most cases, the effect of positive-pressure ventilation in decreasing venous return to the heart outweighs its effect in decreasing afterload, and decreased cardiac output results. In patients breathing spontaneously on PEEP as compared with those breathing spontaneously without PEEP, however, an increased cardiac output has been demonstrated. [81]

Pneumonia is one of the more common complications of intubation and mechanical ventilation. In a review of infections related to medical devices, it was found that ventilator-associated pneumonia was second in frequency only to catheter-related cystitis, and it was estimated that 75,000 ventilator patients a year acquire nosocomial pneumonia, with a fatality rate of 40%. [82] In another large-scale retrospective study, the incidence of nosocomial pneumonia was 0.3% in patients not on ventilators, 1.3% in patients ventilated by endotracheal tube, and a surprising 66% in patients ventilated by tracheostomy. [83] No pneumonias developed in patients ventilated <24 hours, whereas there was an abrupt rise in risk for patients ventilated >5 days.

Most cases of ventilator-associated pneumonia resulted from enteric gram-negative organisms. Ventilator-associated pneumonias may be preventable in part by strict adherence to sterile technique during suctioning and by avoidance of prolonged ventilation or tracheostomy whenever possible. Ventilator humidification systems are a potential source of bacterial contamination, and it is the policy at many hospitals to change the ventilator tubing and humidification system every 24 hours. With the cascade humidifiers used on most modern ventilators, however, there is probably little risk of introducing bacteria into the inspired gas mixture, and it has been shown that the system need not be changed more often than every 48 hours. [84]

Oxygen toxicity was discussed as a potential complication of mechanical ventilation in the section on inspired $O_2$ concentration. Although the exact dose-time relationship of $O_2$ toxicity has not been worked out, it should be assumed that an $F_{IO_2}$ 0.50 has the potential to produce toxic effects. In many cases, $O_2$ toxicity can be avoided by using measures such as PEEP, which improves the A-a$_{DO_2}$ and allows use of lower inspired $O_2$ concentrations.

A final condition that should be included as a potential complication of mechanical ventilation is bronchopulmonary dysplasia. Bronchopulmonary dysplasia is a form of chronic lung disease that occurs in infants who have been mechanically ventilated for severe hyaline membrane disease. It is characterized radiographically by cystic enlargement of the airways with intermingled dense, strand-like infiltrates. The incidence of bronchopulmonary dysplasia is 6 to 11% in survivors of hyaline membrane disease. [33] It is not known whether bronchopulmonary dysplasia results from high inspired $O_2$ concentrations, high airway pressures, or the evolution of hyaline membrane disease itself. One small study has suggested that intramuscular administration of vitamin E may modify the development of bronchopulmonary dysplasia. [85] Until more is known about the actual cause of this condition, however, it cannot be considered preventable.

## HIGH-FREQUENCY VENTILATION

More than 800 articles have been published on high-frequency ventilation (HFV) since the technique was introduced in Sweden in 1967, and it has been the subject of several recent reviews.  High-frequency ventilation differs radically from the traditional approach of using $V_T$ values and frequencies in the physiologic range. Instead, $V_T$ values of a few milliliters per kilogram are used at rates from 60 to 3000 breaths/min. The chest does not rise and fall in patients on HFV unless they breathe spontaneously around the ventilator. One might expect that only the dead space gas would be exchanged and that no alveolar ventilation would occur at all. In fact, however, it has been found that adequate ventilation and $O_2$ exchange do occur by mechanisms that are still not completely understood but that include facilitated diffusion of gases along the vibrating column of air.

High-frequency ventilation has found practical application in laryngeal surgery, in which it allows the surgeon a clear view of a motionless larynx. It has also been used successfully to ventilate patients with large bronchopleural fistulas in whom conventional mechanical ventilation has failed because of massive air leaks. High-frequency ventilation has been tried in adults with ARDS and in infants with hyaline membrane disease in hopes that adequate oxygenation and ventilation could be achieved with lower airway pressures than with conventional mechanical ventilation and PEEP. Conflicting results have been reported in these situations, and whether HFV offers a definite advantage remains to be established.

Problems that have been encountered with high-frequency ventilation include difficulties in humidifying the inspired air adequately. Mucosal injury and increased pulmonary secretions have been encountered. Conventional alarm systems for detecting apnea, inadequate $V_T$, and excess airway pressure are not applicable in HFV. Although there are now ventilators available commercially that are capable of delivering HFV, there are still no well-established guidelines to aid the clinician in deciding which of the several different ventilator types to choose from or what $V_T$ or rate is best for a given patient.

At this point, it seems unlikely that HFV will replace conventional mechanical ventilation in the majority of patients. With further refinements in technology and greater understanding of the physiology involved, however, it is

probable that the number of indications for HFV will expand and that ventilator designs and settings will become more standardized.

# Chapter 8 - Thoracentesis

**David S. Ross**

The term *thoracentesis* is derived from the Greek *thorakos* (chest) and *kentesis* (to pierce). Although a broad definition could include the introduction of any object into the chest, including thoracostomy tubes, common usage is confined to the temporary insertion of a needle or small catheter into the pleural space. Traditionally, thoracentesis is considered to be the method of removing a pleural effusion for diagnostic or therapeutic purposes. In the emergency situation, the removal of air from the pleural space (e.g., from a tension pneumothorax) may also be referred to as thoracentesis. In this chapter, both aspects are discussed. (Tube thoracostomy and the use of catheter drainage for longer periods of time are discussed in Chapter 9) .

Thoracentesis may be performed whenever the appropriate indications are present and suitable equipment is available. Because a tension pneumothorax may quickly cause death, all physicians should be familiar with its clinical appearance and the appropriate indications and techniques for relief of this life-threatening complication. In many communities, paramedical and nursing personnel are also trained in needle or catheter thoracentesis for treatment of tension pneumothorax. Removal of pleural fluid by thoracentesis may be performed by any physician experienced with its indications, precautions, and techniques. It may be performed in the emergency department (ED) or on medical and surgical floors.

The pleural space is a potential space between the visceral and parietal pleura that contains a thin physiologic layer of pleural fluid. With normal inspiration, a negative pressure is developed within the thorax and is transmitted through the pleural space to the pulmonary parenchyma, allowing a normal influx of air. During normal expiration, the elasticity of the pulmonary parenchyma and chest wall allows exhalation.

If fluid, blood, or air accumulates in the pleural space, normal ventilatory mechanisms may be affected. If the volume of fluid or air is large, respiratory compromise may be the result. If the accumulation is rapid and progressive (e.g., tension pneumothorax), there may be cardiovascular compromise in addition to severe respiratory effects. The underlying etiology of an effusion also influences the severity of symptoms.

## BACKGROUND

Thoracentesis (originally called *paracentesis thoracis*) was first described by Hippocrates for relief of empyema. [1] Various operative approaches using trocars or open drainage were advocated during the ensuing centuries. Professional wound suckers who assisted with drainage were replaced by the aspiration syringe, described in 1674. Anel reported the first

successful evacuation of a pneumothorax in 1707. [2] Laennec used thoracentesis for the relief of an apparent pneumomediastinum with a possible associated tension

pneumothorax. [3]

In the 19th century, more interest developed in thoracentesis. Boerhaave advocated the use of a flexible tube for evacuation of a hemothorax. [2] Bowditch provided the first description of thoracentesis in the American medical literature in 1851. [4] Hunter adapted the newly developed hypodermic needle into a modern instrument for thoracentesis in 1859. An underwater seal was later designed by Playfair. [2] Hewett described continuous pleural drainage for empyema in 1876. [5]

Antiseptics and improved sterile technique enabled widespread successful use of thoracentesis in the 20th century. In World War II, thoracentesis and chest drainage replaced routine thoracotomy for most chest injuries, and during the Korean conflict, repeated thoracentesis was advocated for penetrating chest wounds. [3] [6] At the time of the war in Vietnam, the improvement in thoracostomy tubes made them more effective, and tube thoracostomy came to be preferred to simple or repeated thoracentesis.

# INDICATIONS

In the emergency setting, thoracentesis is most often indicated as a life-saving intervention in cases of suspected tension pneumothorax. It is usually done as a temporizing measure before thoracostomy tube placement. Occasionally, a pleural effusion may require emergency evacuation for therapeutic reasons in the compromised patient. More often, thoracentesis will be done semielectively for diagnostic purposes. Thoracentesis has also been performed for evacuation of a small, stable pneumothorax.

There are two general approaches to performing thoracentesis. The anterior approach is most often recommended for removal of air. The posterior approach is chosen most commonly for removal of pleural fluid. Each is discussed separately with further detail regarding clinical indications. The current indications for each approach are summarized in Table 8-1 .

**Anterior Approach for Evacuation of Air**

**Stable Pneumothorax**

**Etiology.**

Pneumothorax is defined as air within the pleural space. It usually enters from a rent in the airway, but it may also enter through the chest wall or from a perforated hollow viscus. Many pneumothoraces occur spontaneously

---

**TABLE 8-1** -- Indications for Thoracentesis

| |
|---|
| **Evacuation of Air: Anterior Approach** |
| Emergency diagnosis and treatment of suspected tension pneumothorax (temporary treatment before tube thoracostomy) |
| Evacuation of simple stable pneumothorax |
| **Evacuation of Fluid: Posterior Approach** |
| Diagnostic analysis of pleural effusion |
| Acute treatment of large symptomatic pleural effusion and tension hydrothorax |

in patients predisposed by the presence of such conditions as chronic obstructive pulmonary disease (COPD), asthma, aspiration pneumonia, or malignancy. Others occur without evidence of preexisting lung disease. These idiopathic spontaneous pneumothoraces typically occur in young tall males, and they may be recurrent. [8] [9] They occur more frequently in smokers. [10] Catamenial pneumothorax may occur in women at the time of menses but is rare. [11] Bilateral spontaneous pneumothoraces have been reported. [12] In the emergency department, most pneumothoraces occur secondary to trauma, either blunt or penetrating, and may be associated with a hemothorax. Pneumothorax may also occur iatrogenically secondary to central venous catheterization; cardiopulmonary resuscitation; intubation; mechanical ventilation; positive end-expiratory pressure (PEEP); and any needle insertion into the chest wall, such as thoracentesis, electromyography, and breast or lymph node biopsy. [13] [14] [15] [16] [17] Drug abusers may induce a pneumothorax from "pocket shooting" drugs into the internal jugular veins as well as from a Valsalva maneuver performed to enhance a "high" while smoking marijuana or cocaine. [18] [19]

**Diagnosis.**

Patients with a pneumothorax typically experience dyspnea and pleuritic pain that is localized to the involved hemithorax. Cough may also be present. Symptoms vary greatly depending on the size of the pneumothorax and its etiology as well as any underlying lung disease. Physical findings also vary and may include tachypnea,

tachycardia, diminished breath sounds, and increased hyperresonance to percussion. [9] Subcutaneous emphysema may occur. Increased resonance to percussion of the ipsilateral clavicle has been reported. [20]

Radiographic diagnosis of a pneumothorax is made by noting the presence of a visceral pleural line and the absence of pulmonary markings peripheral to that line. An upright expiratory chest film or a lateral decubitus view with the involved side up may enhance the visibility of a small pneumothorax. In the absence of pleural adhesions, the pleural air is free flowing, so a pneumothorax will assume the apical-lateral position on an upright chest radiograph. Pneumothorax must be differentiated from bullae in patients with COPD. [8]

In the supine patient, a small pneumothorax may collect in several recesses. The most common is an anteromedial collection in which a radiolucent band is seen parallel to and enhancing the heart border. Another common location is subpulmonic, in which a radiolucent band is seen superior and parallel to the diaphragm, outlined by the visceral pleura. [21] [22] Other locations include the costophrenic sulcus and the posteromedial recess. In the former, the sulcus appears deepened ( *deep sulcus sign*), often with an oblique radiolucent band projected across the upper abdomen. In the latter, retrocardiac air is seen outlining a collapsed lower lobe and adjacent structures. Other signs of pneumothorax on the supine chest film include hyperlucency of the hemithorax, depressed diaphragm, double diaphragmatic contour, increased delineation along the inferior cardiac border, and visualization of lobulated pericardial fat at the cardiac apex. [21]

Any of these small pneumothoraces may proceed to a tension pneumothorax. [22] Several additional views may help with their diagnosis, including cross-table lateral, abdominal, and oblique views of the chest. If the patient can be repositioned, the free pleural air will rise, as can be demonstrated

on a semirecumbent, upright, or lateral decubitus film. Computed tomography (CT) of the abdomen may demonstrate the pneumothorax. [23] Some suggest performing a limited CT of the lower chest of the major trauma patient, particularly when the patient is having a CT for evaluation of other trauma. [24]

The size of a pneumothorax is often underestimated because volume is a cubic measurement and is not always appreciated by viewing its linear dimensions. An accurate nomogram (see Chapter 9) has been designed for determining pneumothorax size. [25] However, in the ED, from a practical standpoint, categorization of pneumothorax size may be adequate. Pneumothoraces may be functionally categorized as small (marginal), moderate, and large (massive). [26]

**Treatment.**

The usual treatment for a pneumothorax is tube thoracostomy, as discussed in Chapter 9 . Conservative management has been advocated in selected cases. [27] Stable spontaneous pneumothoraces (<20%) are commonly absorbed gradually at the rate of 1.25% daily. [28] This absorption will be enhanced by the administration of oxygen. Small

or marginal stable spontaneous pneumothoraces may be observed for resolution, [8] although some advocate thoracentesis to hasten the resorption and increase patient comfort. [29] In a Detroit study, patients with simple pneumothoraces of varying sizes were treated successfully with catheter aspiration, followed by a 6-hour observation period and discharge from the ED; this was successful in 76% of cases. [30] Similar catheter aspiration and outpatient management proved successful in self-inflicted pneumothoraces among "pocket shooters." [18] Small pigtail catheters and small-bore thoracostomy tubes have also been used successfully to treat pneumothoraces, with the use of the Seldinger (guide wire) technique. [31] [32] [33] [34] (For further discussion of the Seldinger technique for vascular access see Chapter 21.)

**Tension Pneumothorax**

**Mechanism.**

A tension pneumothorax is marked by the progressive collection of air in the pleural space through a rent in the airway, with subsequent increasing pleural pressures and often a shift of the mediastinum away from the side of the pneumothorax. During inspiration, negative intrapleural pressure facilitates flow into the pleural space. During expiration, air is less able to exit the pleural space in the patient with a tension pneumothorax, because of the relative compression of the bronchioli and alveoli and subsequent collapse of the rent in the airway (a result of positive intrathoracic pressure). This creates a one-way valve mechanism, which favors collection and trapping of additional air in the pleural space.

As the volume of intrapleural air continues to expand, the intrapleural pressure rises. This pressure is transmitted against the lungs, causing a continued decrease in functioning lung volume. First the ipsilateral and then the contralateral lung becomes compressed. Resultant parenchymal collapse leads to respiratory compromise as ventilation-perfusion mismatch develops and hypoxia and acidosis ensue. In addition, the increase in the volume of the intrapleural air causes a shift of the mediastinum away from the side of the tension pneumothorax. There may be some decrease in the systemic venous return because of a combination of increased intrathoracic pressure and mechanical collapse of the venae cavae. Originally, it was felt that compromised venous return was the primary cause of diminished cardiac output that led to cardiovascular collapse. Animal studies, however, suggest that progressive hypoxia and $CO_2$ retention may be the primary mediators of cardiovascular depression and collapse. [35] [36] The rapidity with which these events occur is variable. They can proceed quickly and lead to death in a matter of minutes. Therapeutic intervention may often be necessary before full diagnostic evaluation can be carried out.

**Etiology.**

A tension pneumothorax may develop as a result of any of the usual causes of pneumothorax described earlier (see also Chapter 9) . It may develop either primarily as a complication of a previously stable pneumothorax or as a result of positive-pressure ventilation. Although occasionally occurring in the patient with spontaneous pneumothorax, it is seen more often in patients following trauma and may occur

following instrumentation of the chest, such as during central line insertion. It has been reported following incorrect placement of a nasogastric tube in an elderly patient. [37] Certain factors may predispose to the development of a tension pneumothorax in a patient with an otherwise stable pneumothorax. A major predisposing factor is the use of positive-pressure ventilation, either by bag-valve devices or by mechanical ventilators. [38] The addition of PEEP may further increase the risk of development of a tension pneumothorax. [39] Likewise, cardiopulmonary resuscitation may predispose to the development of a tension pneumothorax. [8]

**Clinical diagnosis.**

Tension pneumothorax should always be suspected in any patient in whom sudden respiratory or cardiac deterioration occurs. Development of sudden difficulty ventilating a patient should alert the clinician to the possibility of tension pneumothorax. [8] The clinical presentation in the patient who is awake includes the sudden development of dyspnea, agitation, or diminished consciousness. Tachypnea, tachycardia, hypotension, cyanosis, and diaphoresis may be present and may progress to cardiac or respiratory arrest. The following classic constellation of findings may be seen, although they are not necessarily uniformly present: tympany to percussion, decreased breath sounds over the involved hemithorax, tracheal deviation toward the contralateral side, and the presence of an overinflated, immobile, ipsilateral hemithorax. Jugular venous distention may also be seen. Other signs of chest trauma or respiratory distress should prompt one to consider the possibility of an underlying pneumothorax with potential tension.

In the patient whose condition suddenly deteriorates, other diagnostic considerations should include massive pulmonary embolus, pericardial tamponade, pneumomediastinum, respiratory failure from reactive airway disease, and myocardial infarction. Traumatic diaphragmatic hernia has also been reported to simulate a tension pneumothorax. [40] The patient with a tension pneumothorax who is being ventilated mechanically will show signs of increased airway resistance, evidenced by increased ventilatory pressures, prolonged inspiratory times, elevated central venous pressure, and elevated pulmonary artery pressure. [39]

When the patient is in extremis, the diagnosis should be made by needle or catheter thoracentesis. The patient will show clinical improvement following thoracentesis. The successful relief of a tension pneumothorax is also marked by the rapid efflux of air through the thoracentesis needle during both inspiration and expiration. If a syringe (ideally premoistened) is attached to the thoracentesis needle, the plunger may be driven outward if sufficient pressure has developed. Likewise any drainage apparatus that directs the

efflux of air through a water seal will reveal vigorous bubbling of air in inspiration as well as expiration until the high-pressure air has been released. In a similar manner, a flutter-valve apparatus attached to the needle may continue to show evidence of massive air efflux. If an intubated patient is being ventilated, *diagnostic tube thoracostomy is* preferred to thoracentesis when time permits. There is a theoretical risk of creating a tension pneumothorax by indiscriminate thoracentesis in a patient who is

receiving positive-pressure ventilation.

**Radiologic diagnosis.**

Withholding treatment of a tension pneumothorax until radiographic confirmation is made is not advised except in the following circumstances: in patients whose conditions are stable with only moderate respiratory compromise; in patients in whom tension pneumothorax is suspected but physical evidence is lacking; in patients in whom other likely conditions are being considered, such as a ruptured diaphragm, in which needle decompression may be relatively contraindicated; and in patients in whom portable chest radiographs may be completed immediately.

A patient who has even the most remote possibility of a tension pneumothorax should *never be sent to the radiology suite unless accompanied by a physician* who is prepared to perform immediate thoracentesis or tube thoracostomy. Despite an increased awareness of the possibility of a tension pneumothorax on clinical grounds, the diagnosis is often not recognized until a chest film has been taken. [41]

Radiographic diagnosis is made by noting the presence of the usual signs of pneumothorax described previously, combined with a flattening of the diaphragm and a shift of the mediastinum away from the side of the pneumothorax. [8] In most cases, even in the supine patient, tension pneumothorax is easily diagnosed. Several exceptions exist. In the case of bilateral pneumothoraces, no mediastinal shift may be seen. In this case, tension pneumothorax may be diagnosed clinically on decompression of the chest. Localized areas of tension pneumothorax may occur secondary to adhesions. In patients with adult respiratory distress syndrome (ARDS) on PEEP, localized subpulmonic or paracardiac tension pneumothoraces have been reported despite the presence of thoracostomy tubes. [42] These cases will be suspected clinically because of the presence of increased respiratory pressures and diagnostic criteria for localized pneumothorax discussed earlier, with flattening of the cardiac border and a depression or contour change of the diaphragm.

**Treatment.**

Treatment of a tension pneumothorax should begin as soon as it is recognized clinically. It should be emphasized that although *the preferred and definitive treatment of a tension pneumothorax is immediate tube thoracostomy*, [43] this may not be possible in many clinical settings. If a patient's condition deteriorates suddenly and rapid tube thoracostomy is not possible immediately, thoracentesis is an appropriate temporary treatment and may be life saving. Thoracentesis is most commonly and safely performed by simple needle or catheter aspiration in the midclavicular line, second or third intercostal space. Once a tension pneumothorax is confirmed by this method, a thoracostomy tube should be placed as soon as possible (see Chapter 9) . This is particularly important in the patient on positive-pressure ventilation.

Thoracentesis may also be used in the out-of-hospital care setting, when a patient in extremis is suspected of having a tension pneumothorax. To facilitate the continued

drainage of the pneumothorax, an expedient flutter valve, underwater seal, or commercially available one-way valve may be attached. [44]

McSwain has developed a self-retaining catheter and trocar (McSwain dart, Medical Dynamics, Inc.) that can be placed in the chest and attached to a drainage system. [45] Although this apparatus has been suggested as an alternative to needle or catheter placement, it may be associated with increased risk of pulmonary injury, particularly if placed incorrectly when a pneumothorax is not present [46] (see Chapter 9) . Use of a guide wire-directed minicatheter is currently preferred to use of a minitrocar system. [32] [33] [34]

## Contraindications

In clinical situations in which a tension pneumothorax is suspected, several relative contraindications to the use of thoracentesis are important to remember. If tube thoracostomy is readily available, it may be the preferred procedure, and thoracentesis may only serve to delay the more definitive treatment. In patients who are being ventilated manually or by respirator, extreme caution should be exercised when performing thoracentesis. If the presumptive diagnosis of a tension pneumothorax is incorrect, the insertion of a thoracentesis needle may actually create a pneumothorax, which may be converted into a tension pneumothorax by the positive-pressure ventilation.

An absolute contraindication to thoracentesis is the insertion of a needle through an area of infection. [8] In such cases, an alternate insertion site should be selected. In patients who have bleeding diatheses or who are on anticoagulants, thoracentesis for evacuation of a stable, small pneumothorax may be relatively contraindicated, with observation being the preferred treatment.

## Equipment

The equipment needed to perform rapid thoracentesis for tension pneumothorax or aspiration of a spontaneous pneumothorax is listed in Table 8-2 . Because several techniques are described, the necessary equipment for all techniques is included in this list. Evacuation of a tension pneumothorax can be accomplished by insertion of a needle only, a through-the-needle catheter, or an over-the-needle catheter. Although the needle insertion technique can be performed most rapidly, the catheter insertion techniques may theoretically be safer, allowing continued drainage of the relieved tension pneumothorax without the presence of a rigid needle in the thorax. The drawbacks to the use of a catheter are the possibility of the catheter's crimping or collapsing as well as the increased resistance to drainage caused by the catheter's length. The catheter technique is recommended in relief of tension pneumothorax in the out-of-hospital setting. [47] Likewise, when evacuating a simple stable pneumothorax, the catheter techniques may be preferred because they are less likely to pierce the expanding lung. Small thoracostomy tubes and pigtail catheters may be used for the stable pneumothorax and are placed using the Seldinger technique (see also Chapter 21) .

## Procedure

The anterior thoracentesis approach is used primarily for relief of a tension pneumothorax and evacuation of a simple

pneumothorax. Because treatment is urgently required in the first case, the procedure chosen should be performed in an appropriately expedient manner. Likewise, the amount of preparation and equipment needed is dictated by each particular clinical situation. Informed consent is likely to be impossible to obtain in cases of tension pneumothorax, but an attempt to secure it may be considered in patients with a stable pneumothorax.

### Insertion Site and Patient Position

The conventional approach has been to evacuate tension pneumothoraces by using the anterior approach, with the patient in a supine position and the head of the stretcher elevated 30°. The recommended insertion site is the second intercostal space in the midclavicular line (Fig. 8-1) (Figure Not Available) . The rationale for this approach is that free pleural air will rise to the anterior upper chest. With a tension pneumothorax, however, the collapsed lung is moved away from the entire ipsilateral chest wall, making a lateral approach also possible. If the patient is in a supine position and if the anterior chest is obscured (e.g., subclavian vein catheter bandage, chest monitoring leads, subcutaneous emphysema) or if a chest wall infection is present, a lateral approach may be more practical. The lateral approach is accomplished by inserting the needle into the fourth or fifth intercostal space in the midaxillary or anterior axillary line. This location can be identified quickly in males by extending the horizontal nipple line laterally into the axilla. In females who have large breasts, reliable approximation can be made by extending an

| **TABLE 8-2** -- Equipment Needed for Thoracentesis Evacuation of Air |
| --- |
| **Tension Pneumothorax** |
| Antiseptic solution and sterile gauze sponges (if condition allows) |
| Catheter or needle (either) |

| |
|---|
| Over-the-needle catheter (14- to 16-ga needle) |
| Through-the-needle catheter (14-ga needle) |
| Hypodermic needle (14 to 20 ga) |
| Drainage equipment (either) |
| 5- to 10-mL syringe (premoistened if condition allows) |
| Flutter value (commercial or fashioned from sterile glove fingertip) |
| Sterile intravenous tubing with water-filled basin |
| **Stable Pneumothorax** |
| Antiseptic solution and sterile basin |
| Sterile gauze sponges |
| Sterile towels |
| Syringes (5 to 10 mL) for anesthetic infiltration |

| |
|---|
| Needles (22 and 25 ga) for anesthetic infiltration |
| Catheter (either) |
|    Over-the-needle catheter (16- to 18-ga needle) |
|    Through-the-needle catheter (14- to 16-ga needle) |
|    Pigtail catheter (6.0 to 8.5 Fr) * |
|    Small chest tube (8 Fr) * |
| Drainage equipment (either) |
|    Flutter valve (commercial or fashioned from sterile glove fingertip) |
|    Sterile intravenous tubing with water-filled basin |
|    Syringe (30 to 50 mL) with 3-way stopcock |

* Requires additional equipment for Seldinger technique (see Chapters 9 and 21).

**Figure 8-1** (Figure Not Available) For relief of a pneumothorax, the second intercostal space in the midclavicular line is commonly used. The head of the stretcher is elevated 30°. The midaxillary line, fourth or fifth intercostal space site, has been used for thoracentesis of pleural fluid (see text). *(From Fishman NH: Thoracic Drainage: A Manual of Procedures. Chicago, Year Book Medical Publishers, 1983, p 26. Reproduced by permission.)*

imaginary horizontal line between the inferior tips of the scapulae laterally into the axilla.

There are two major problems when the lateral approach is chosen. The first is the greater risk of parenchymal injury if neither a tension nor a large pneumothorax is present. The second problem is the danger of adhesions. Previous empyema, hemothorax, tuberculosis, and other inflammatory processes may cause pleural adhesions, which frequently occur in dependent portions of the thorax. Therefore, the anterior approach is theoretically safer and is recommended for most cases in which relief of tension pneumothorax is required. Likewise, for the evacuation of a simple stable pneumothorax, the anterior approach is recommended unless a loculated pneumothorax dictates an alternate approach.

**Needle or Catheter Insertion for Evacuation of Air**

**Patient preparation.**

The patient is positioned as previously discussed. An explanation of the procedure is appropriate if the patient is awake, but it should not delay the procedure. Because the patient is usually in extremis, sedation is contraindicated. Restraining the patient may be necessary if the patient is hypoxic and confused. Oxygen should be administered. When time permits, the insertion site is swabbed rapidly with povidone-iodine or another suitable antiseptic. If a stable pneumothorax is being evacuated, an appropriate explanation and a careful preparation of the sterile field is indicated.

**Anesthesia.**

Local anesthesia is usually unnecessary in the case of relieving a tension pneumothorax and will only delay the procedure. If the patient has a slowly progressive tension pneumothorax or a stable pneumothorax and is not in extremis, local anesthesia with 1% lidocaine or its equivalent may be used. It is administered through a 5- to 10-mL

syringe with a 25- to 27-ga needle. An intradermal wheal is raised over the upper edge of the third or fourth rib after localization with palpation. Anesthetic is infiltrated down to the periosteum. The needle is then withdrawn.

**Insertion techniques.**

If time allows, a 14- to 20-ga needle is selected and attached to a 5- to 10-mL syringe premoistened with saline. The third rib is again identified by palpation. The needle is inserted perpendicularly in the midclavicular line over the upper edge of the rib. As the rib is encountered, the needle is "walked" over the superior aspect of the rib and into the lower portion of the second intercostal space. This approach should avoid the intercostal vessels positioned near the lower border of each rib, as indicated in Figure 8-2 (Figure Not Available) . The syringe is gently aspirated as the needle is advanced. A "pop" may be felt as the pleural space is entered and air is encountered. A pneumothorax under tension may create enough pressure to drive the plunger of a premoistened syringe the length of the barrel without manually withdrawing it. If the needle has been inserted without a syringe attached, a rush of air exiting the chest may

be heard in inspiration as well as during expiration. Either of these findings will confirm the presence of a tension pneumothorax. Inserting a needle without an attached syringe or other drainage device invites the possibility of creating a pneumothorax if none is already present. If any pneumothorax is confirmed, a one-way drainage device should be attached as soon as possible.

Alternate methods of relief of a tension or stable pneumothorax include the use of standard intravenous needle and catheter insertion sets (Fig. 8-3) (Figure Not Available) . This can be accomplished by attaching the syringe to the hub of an *over-the-needle catheter* (with a 14- to 18-ga needle). Larger sizes should be chosen for a tension pneumothorax. A small scalpel blade may be used to pierce the skin at the entry site to facilitate catheter entry. The pleura is entered through the second or third intercostal space, as previously described,

**Figure 8-2** (Figure Not Available) "Walking" the anesthetic needle over the superior aspect of the rib. *(From Fishman NH: Thoracic Drainage: A Manual of Procedures. Chicago, Year Book Medical Publishers, 1983, p 28. Reproduced by permission.)*

**Figure 8-3** (Figure Not Available) Insertion technique for through-the-needle and over-the-needle catheters. Separate intercostal spaces depict the steps *(A-C)* that occur at a single intercostal space. *(From Fishman NH: Thoracic Drainage: A Manual of Procedures. Chicago, Year Book Medical Publishers, 1983, p 30. Reproduced by permission.)*

and the catheter is advanced over the needle into the pleural space. The needle is then removed, and the syringe or another drainage device is attached to the hub of the catheter. A tension pneumothorax is confirmed by the same findings as those discussed earlier. The catheter may be secured with sutures. A drainage system is then attached.

A *through-the-needle catheter* (with a 14- to 16-ga needle) of the shortest available length may also be used. The larger diameter should be chosen for a tension pneumothorax. Once the needle has entered the pleural space, the catheter is advanced fully into the thorax through the needle. The needle is then withdrawn, and a drainage device is attached to the catheter hub. The needle guard should be attached to prevent a laceration of the catheter by the surrounding needle. The catheter may be secured by suturing it to the chest wall. A one-way drainage system should be attached.

For evacuation of a *simple stable pneumothorax*, either the over-the-needle or through-the-needle technique may be used. In these cases, it is important to attach a one-way drainage system before piercing the chest, because the pneumothorax will not be under pressure, and an exposed catheter lumen may enlarge the pneumothorax. Commercial thoracentesis kits and specialized equipment may also be used (see Needle or Catheter Insertion for Evacuation of Fluid).

The Seldinger technique may also be used to insert a pigtail catheter (6.5 to 8.5 Fr) or a small chest tube (approximately 8 Fr) for evacuating a stable pneumothorax. A 16- to 18-ga needle is attached to a syringe and inserted into the

pleural space until air is encountered, as described previously. While holding the needle securely, the syringe is removed, and the hub of the needle is covered with a gloved

finger. A fine, flexible-tipped guide wire is inserted through the needle and advanced into the pleural space. The needle is withdrawn, leaving the wire in place. A small scalpel blade is used to pierce the skin adjacent to the guide wire. As an option, a dilator may be threaded over the wire and then removed after enlarging the opening. A pigtail catheter or small chest tube is threaded over the exposed guide wire and gently advanced into the pleura to an adequate depth such that all side openings are well within the chest. The wire is then removed, and the catheter should be secured to the chest wall. The catheter should be attached to a drainage device as soon as possible.

Initially, air may be evacuated with a 60-mL syringe and a three-way stopcock. If expansion is incomplete, continuous suction may be used (see Chapter 9) . The expanding lung may kink the catheter, requiring placement of a larger catheter. The physician may also instruct the patient to change position (e.g., supine to sitting) or cough to help free the catheter and permit complete lung expansion.

**Drainage.**

After the diagnosis of a pneumothorax has been made, drainage should be instituted. In the case of a tension pneumothorax, drainage should be continued until a thoracostomy tube can be placed. Continuous drainage can be accomplished by attaching the distal end of an intravenous tubing set without one-way valves to the needle or catheter hub. The proximal end of the intravenous tubing is placed under water in a basin to create an underwater seal. This prevents air from entering the pleural space. An alternative is to attach a commercial flutter valve, such as the Heimlich valve. An expedient drainage device may also be made using a premoistened finger cut from a sterile examination glove (Fig. 8-4) (Figure Not Available) . The cut edge of the glove finger will act as a one-way flutter-valve.

Any of the previously mentioned techniques may be used for drainage of a stable pneumothorax. Evacuation by attaching a three-way valve to a large (30 to 50 mL) syringe may facilitate the removal of air without creating a danger of increasing the pneumothorax size. Care must be taken to exert gentle negative pressure when aspirating air, because greater negative pressure may cause postexpansion pulmonary edema (see Complications). If a pigtail catheter or small chest tube has been placed, a standard pleural drainage system may be used (see Chapter 9) .

If a tension pneumothorax is found and confirmed by any of the aforementioned methods, a thoracostomy tube should be placed as soon as possible. A chest film is also indicated following thoracentesis or tube thoracostomy to confirm the successful relief of the pneumothorax, absence of hemothorax or other complications, and (if performed) thoracostomy tube placement. This confirmation is best obtained with an upright expiratory chest film.

## Posterior Approach for Evacuation of Fluid

**Pleural Effusion**

**Physiology.**

Pleural fluid is normally created as a result of several physiologic mechanisms, including hydrostatic pressure, colloid oncotic pressure, and intrapleural pressure. [8] [48] With normal pleural homeostasis, the hydrostatic pressure generated by the systemic capillaries across the parietal pleura is greater than the hydrostatic pressure generated by the pulmonary capillaries across the visceral pleura. Because the colloid oncotic and intrapleural pressures exert symmetrical forces, a gradient is created that allows for the formation of normal fluid at the parietal pleura and absorption by the visceral pleura. Lymphatic flow also facilitates absorption. A summary of the homeostatic forces involved is shown in Figure 8-5 (Figure Not Available) .

The integrity of this system can be disrupted by changes in hydrostatic pressure, colloid oncotic pressure, intrapleural pressure, lymphatic flow, capillary permeability, and pleural fluid surfactant composition. [48] [49] Fluid can accumulate because of two processes, transudation and exudation. Transudation may occur from increased pulmonary capillary pressure, as in left ventricular failure, or from decreased colloid oncotic pressure secondary to hypoalbuminemic states, as in

**Figure 8-4** (Figure Not Available) Use of a needle and a sterile finger cot or a finger from a sterile glove to fashion a one-way (flutter) valve for emergency evacuation of a tension pneumothorax. A small opening is made in the free end of the glove to allow air to escape during expiration. *(Redrawn from Cosgniff JH: An Atlas of Diagnostic and Therapeutic Procedures for Emergency Personnel. Philadelphia, JB Lippincott, 1978, p 243.)*

**Figure 8-5** (Figure Not Available) Diagrammatic representation of the pressures involved in the formation and absorption of pleural fluid. *(From Fraser RG, Pare JAP: Diagnosis of Diseases of the Chest. Philadelphia. WB Saunders, 1970, p 65. Reproduced by permission.)*

the nephrotic syndrome and cirrhosis. Structural abnormalities, such as peritoneal-pleural communications in cirrhotic patients with ascites, have been reported. [50]

The exudation of fluid into the pleural space may occur as the result of abnormalities, such as changes in surfactant or altered capillary permeability seen with inflammatory disease, infection, pulmonary infarction, respiratory distress syndrome, and neoplasm. [8] [48] [49] Lymphatic malignancy may decrease fluid resorption. Likewise, trauma to the thoracic duct, esophagus, or vascular structures leads to the direct flow of the contents of these structures into the pleural space.

**Etiology.**

Numerous etiologies may account for the formation of abnormal pleural fluid. Rapid or relatively acute accumulations may be seen with hemothorax, esophageal rupture, pulmonary infarction, infection, empyema, obstructive uropathy, thoracic duct injury, or iatrogenic causes (such as intrapleural placement of a subclavian vein catheter). Chronic effusions are also commonly seen in many disease states. The most common atraumatic causes of effusion are congestive heart failure, infection, and neoplasm. [8]

Effusions due to pneumonia are referred to as *parapneumonic.* Massive effusions causing mediastinal shift (tension hydrothorax) due to malignancy, dialysis, and following iatrogenic intrapleural infusion of parenteral hyperalimentation solution have been reported. [51] [52] [53] Traditionally, effusions have been classified as transudates or exudates. Transudates typically have a low specific gravity and protein concentration. Exudates usually have an increased specific gravity and high protein concentration (see Pleural Fluid Analysis). Common causes of effusions are listed in Table 8-3 .

**Clinical diagnosis.**

A pleural effusion may be asymptomatic or may produce varying degrees of dyspnea or pleuritic pain. Other symptoms, such as cough, fever, weight loss, or edema, may be present and may be related to the underlying cause of the effusion.

The physical signs of pleural effusion vary according to its size. Small effusions do not usually produce physical findings. Moderate-sized effusions may produce physical findings over the area of the effusion. A pleural friction rub may be present. Breath sounds may be diminished, there may be dullness to percussion, and tactile fremitus may be decreased. Occasionally, decreased chest excursions are evident. In very large effusions, breath sounds may change from vesicular to bronchovesicular, gradually becoming absent as compression of the underlying lung occurs. A tension hydrothorax may produce mediastinal shift with tracheal deviation, displacement of the cardiac impulse, and bulging of the intercostal spaces. [52] [53]

**Radiographic diagnosis.**

The radiographic findings of pleural effusions vary with the location and amount of fluid present. Slight elevation of the hemidiaphragm or lateral displacement of the diaphragmatic dome on the posteroanterior chest film suggests a small subpulmonic effusion. Blunting of the costophrenic angle may be seen on an upright chest film and represents at least 175 mL of fluid. [54] A fluid level with an upwardly concave meniscus indicates a

| **TABLE 8-3** -- Causes of Pleural Effusion |
| --- |
| **Transudates** |
| Congestive heart failure |

| |
|---|
| Cirrhosis with ascites |
| Nephrotic syndrome |
| Hypoproteinemia |
| Acute glomerulonephritis |
| Peritoneal dialysis |
| Urinary obstruction (urinothorax) |
| Superior vena caval obstruction |
| Acute atelectasis |
| Myxedema |
| **Exudates** |
| Neoplasm |
| Pulmonary infarction (embolus) |

| |
|---|
| Bacterial pneumonia (parapneumonic effusion) |
| Empyema |
| Lung abscess |
| Other pneumonias |
|   Viral |
|   Tuberculous |
|   Fungal |
|   Rickettsial |
|   Parasitic |
| Collagen vascular disease (lupus erythematosus, dermatomyositis, rheumatoid pleuritis) |
| Pancreatitis |
| Drug reactions (nitrofurantoin, methysergide, practolol) |

| |
|---|
| Asbestosis |
| Meigs syndrome |
| Dressler syndrome |
| Lymphatic disease (chylothorax) |
| Trapped lung (fibrothorax) |
| Subphrenic and hepatic abscess |
| Sarcoidosis |
| Chronic atelectasis |
| Uremia |
| Urinary obstruction (urinothorax) |
| Pulmonary or vascular disruption (hemothorax) |
| Esophageal rupture |

considerably larger effusion. The higher level of the meniscus laterally is due partly to the fact that the lung exerts pressure outward from the hilum, pushing the fluid toward the periphery. [55] In addition, the increased density of the fluid viewed tangentially at the edge of the chest renders it visible, whereas the fluid located medially may not be of sufficient depth to be visualized. A fluid level without a meniscus is indicative of a coexisting pneumothorax. A massive pleural effusion with contralateral shifting of the mediastinum and diaphragmatic inversion indicates a tension hydrothorax. [53]

Lateral decubitus views of the chest with the involved side down may help to identify small amounts of pleural fluid. [56] Comparison of bilateral decubitus views may help to differentiate fluid from other densities as well as enable viewing underlying lung previously obscured by pleural fluid. [57]

On a supine anteroposterior film, a generalized increased density over the lung field(s), costophrenic blunting, apical capping, widening of the minor fissure, and obscuring of the hemidiaphragm may be suggestive of a pleural effusion. [58] A pleural effusion may be differentiated from an infiltrate by the lack of air bronchograms and the absence of a "silhouette sign" obscuring the heart border. [8] A lateral decubitus or upright chest film helps to confirm this diagnosis.

Other findings may be seen in various views, including thickening of the pleural fissures. A middle lobe "step" has been described on the upright lateral film in which the major and minor fissures outline the middle lobe while the lower lobe is compressed. [8] Loculated effusions along the pleural border due to adhesions from previous hemothorax or empyema may be seen occasionally. Elliptical thickening of the fissures is suggestive of loculated effusions. Ultrasonic examination of the chest may help differentiate loculated effusions from solid tumors as well as help localize fluid for thoracentesis. [59] A CT of the chest may help evaluate underlying pathologic findings in the presence of an effusion. [60] It may also help identify subpulmonic effusion, atelectasis, and subphrenic fluid. [61] [62]

**Treatment.**

Management of pleural effusions must be directed toward treatment of the underlying condition. In most cases, thoracentesis is indicated for diagnostic purposes. Exceptions to the need for diagnostic thoracentesis may be made for congestive heart failure or for the stable, previously diagnosed effusion in which the cause is clear. Thoracentesis may be withheld in these situations if the effusion responds to medical management. [63] In cases in which tuberculosis or malignancy is suspected, pleural biopsy may also be indicated. The timing of the thoracentesis and biopsy should be coordinated.

Repeated thoracentesis has been recommended by some to monitor the pH of a parapneumonic effusion; a decreasing pH may indicate progression to a complicated parapneumonic effusion that requires thoracostomy tube drainage. [64] Other conditions diagnosed by thoracentesis, such as empyema or hemothorax, usually require thoracostomy tube insertion to facilitate more thorough evacuation. Likewise, chylothorax due to thoracic duct trauma is often managed by thoracostomy drainage

and total parenteral hyperalimentation. [65] A ruptured esophagus usually requires thoracotomy and surgical repair.

Another major indication for thoracentesis is evacuation of a large symptomatic pleural effusion. If the effusion is quite large, has accumulated rapidly, or causes inversion of the diaphragm, the effusion itself may seriously impair respiratory function by decreasing lung volume and increasing shunting. [66] Tension hydrothorax likewise causes significant respiratory compromise and may impair venous return. [67]

In the conditions mentioned previously, removing a large amount of pleural fluid may provide significant therapeutic benefit by improving oxygen saturation, improving the alveolar-arterial oxygen gradient, decreasing shunting, and improving vital capacity. [68] [69] Although removing a maximum of 1000 to 1500 mL has been a general rule of thumb, as it was thought to prevent postevacuation pulmonary edema, larger amounts may be removed safely by monitoring the pleural pressures and maintaining gentle negative pressure (see Complications). In cases of intractable malignant effusions, placement of a pleuroperitoneal shunt may be useful. [70]

**Hemothorax**

Historically, repeated thoracentesis was used initially to evacuate hemothoraces. With the advent of effective tube thoracostomy, however, this method has become nearly obsolete. Several major arguments against this practice can explain its decline. Repeated violation of the pleura and persistence of the hematoma may increase the risk of infection. The effectiveness of evacuation is inferior to that of tube thoracostomy with continuous suction. Likewise, thoracostomy tube drainage allows better monitoring of the rate of bleeding. Failure of complete resorption of a hemothorax may create a "fibrous peel," or fibrothorax, which can lead to restricted ventilation and may subsequently require open thoracotomy and decortication. Finally, tamponade of the bleeding source may theoretically be enhanced by complete expansion of the lung.

Nevertheless, some investigators suggest that thoracentesis may be used safely in select cases, such as a small stable traumatic hemothorax. [71] In one series, thoracentesis was used in traumatic hemothorax for 104 of 502 patients, with only 1 patient requiring subsequent thoracotomy for fibrothorax. [72] In a second series of 130 patients, thoracentesis was used for hemothorax evacuation in 48 patients, with 10% developing fibrothorax. [73] The results were poorest in patients with hemopneumothorax. Other isolated successful uses of thoracentesis in cases of nontraumatic hemothorax have been reported, including in Osler-Weber-Rendu disease and spontaneous hemothorax in the newborn. [74] [75] (Traumatic hemothorax is discussed in Chapter 9.)

## Contraindications

The removal of pleural fluid by thoracentesis should be avoided in patients with a bleeding diathesis and in those on anticoagulants before correction of the clotting deficits. [8] Thoracentesis is also contraindicated unless performed under ultrasound or fluoroscopic guidance if the patient has an ipsilateral ruptured diaphragm. Extreme

caution should be used when thoracentesis is being performed in a patient who has pleural adhesions, such as from previous tuberculosis, hemopneumothorax, or empyema, because of the danger of piercing the closely approximated visceral pleura and lung. Should there be an infection of the chest wall, thoracentesis is contraindicated unless an alternate puncture site without soft-tissue infection can be selected. In patients who require removal of large quantities of fluid, needle insertion techniques

may be relatively contraindicated because of an increased risk of parenchymal damage. [8] Of note, Godwin and Sahn report a thoracentesis series in mechanically ventilated patients with a mean fluid withdrawal of 840 mL. [76] Only 2 of 32 procedures resulted in a pneumothorax. Hence, mechanical ventilation alone should not represent an absolute contraindication. Nonetheless, the operator must closely monitor the patient in such situations, because any pneumothorax is at great risk for becoming a tension pneumothorax.

## Equipment

For removal of fluids, more equipment is necessary than that required for aspiration of a pneumothorax. The additional equipment permits thorough patient preparation, more adequate anesthesia, controlled evacuation of fluid, and careful collection of specimens. In Table 8-4 , the basic and alternative equipment is listed. The simple needle method has the potential risk of perforating the visceral pleura as the fluid is withdrawn, whereas the catheter of narrow diameter and long length may limit the removal of thick, tenacious fluids. Catheters also have the drawback of crimping if not handled carefully.

Either needle insertion or catheter insertion techniques may be used for diagnostic thoracentesis when only a relatively small amount of fluid is being withdrawn. Needle insertion techniques may increase the risk of parenchymal injury when large quantities of fluid are removed for therapeutic purposes. [8] Likewise, catheter insertion techniques may be preferable when only a small amount of fluid is available and the risk of contact with the lung is greater.

Pigtail catheters also have been used successfully to remove effusions. [32] [33] [77] The Seldinger technique is used to aid in their insertion.

## Procedure

Thoracentesis is generally an elective procedure and should be performed after an adequate number of diagnostic radiographic

| **TABLE 8-4** -- Equipment Needed for Thoracentesis and Fluid Collection |
| --- |
| Basin for preparation solution |

| |
|---|
| Antiseptic solution (povidone-iodine) |
| Sterile gauze sponges |
| Sterile towels |
| Syringes (5 to 10 mL) for anesthetic |
| Needles (22 and 25 ga) for infiltration |
| Local anesthetic (e.g., 1% lidocaine)--10 mL |
| Syringe (50 mL) for aspiration |
| Catheter or needle (either) |
| Hypodermic needle (18 to 22 ga, 1½ to 2 inch) |
| Over-the-needle catheter (16- to 20-ga needle) |
| Through-the-needle catheter (14- to 18-ga needle) |
| * Pigtail catheter (6.0 to 8.5 Fr) |

| |
|---|
| 2 Curved hemostats |
| 3-Way stopcock |
| Sterile intravenous extension tubing |
| Specimen bowl (may be calibrated for volume measurement) or sterile vacuum bottle with intravenous tubing |
| Sterile dressing and adhesive tape |

\* Requires additional equipment for Seldinger technque (see Chapters 9 and 21)

**Figure 8-6** (Figure Not Available) Upright positioning of patient for drainage of pleural fluid. Note the use of the hemostat to limit the depth of penetration of the thoracentesis needle. *(From Nealon TF Jr: Fundamental Skills in Surgery. 3rd ed. Philadelphia, WB Saunders, 1979, p 291. Reproduced by permission.)*

studies have been obtained. The procedure should be carried out with sufficient preparation after all available equipment has been tested and under properly controlled circumstances. Informed consent should usually be obtained and documented, according to hospital policy.

The posterior approach is most often recommended when performing thoracentesis for removal of pleural effusions for either diagnostic or therapeutic purposes. A lateral approach may be used in certain circumstances that are described later.

**Insertion Site and Patient Position**

The choice of insertion site for removal of pleural fluid depends on many clinical factors. If the fluid collection is large and if the patient is able to sit upright for a prolonged period, the following approach is recommended: The patient is seated, leaning forward slightly, supported by the back of a chair or a table. Some physicians recommend that the patient's back be vertical to keep the fluid in the dependent posterior chest (Fig. 8-6) (Figure Not Available) . [8] The site chosen for aspiration is the midscapular line or the posterior axillary line at a level below the top of the fluid. This level is best determined clinically at the time of the procedure by the height of dullness to percussion and the decrease in tactile fremitus. [78] A single interspace below this level should be selected.

[8]

A chest film of the patient in an upright position should be taken to diagnose and locate the general position of the effusion, but reliance on the film is less precise than clinical assessment. Radiographic determination of fluid levels may occasionally be misleading, because the position of the fluid changes with respiration and patient position. In addition, one should be reminded that the fluid level seen on a chest film of a patient in an upright position is actually higher

than the bottom of the fluid meniscus. [55] The raised lateral edge of the fluid level represents the height of fluid available at the periphery. An exception to this distribution of pleural fluid occurs when a combined pneumothorax and pleural effusion produces a flat air-fluid level. In all cases, the lowest level recommended for thoracentesis is the eighth intercostal space. The highest level clinically indicated is generally chosen to minimize inadvertent abdominal insertion of the needle. If a catheter has been inserted and insufficient fluid is obtained, the patient can subsequently be repositioned to make the insertion site more dependent. [78]

The following alternative approaches have been described: If the patient is unable to remain seated, a catheter (through-the-needle, over-the-needle, or pigtail) may be inserted into the midaxillary or posterior axillary line in the fourth or fifth intercostal space while the patient is in a supine position (see Fig. 8-1) (Figure Not Available) . [55] It may be prudent to evaluate the height of the fluid both clinically and radiographically in this position before attempting this approach. If the patient can tolerate some elevation, it has been suggested that the patient recline against the raised head of the bed for support while the needle is inserted in the midaxillary or posterior axillary line at the appropriate level. [8] A lower interspace also may be used in these cases. Another suggestion is that the patient lie in a lateral decubitus position with a posterior site chosen for insertion. [8] If the amount of fluid is small and the need for diagnostic analysis is considerable, fluid can be aspirated from beneath the patient in the midaxillary line with the patient in the lateral decubitus position. [79] This technique requires that the patient be placed across an open space between stretchers in such a way that the physician can aspirate from below the patient and between the supporting structures.

For small or loculated effusions, ultrasound-guided thoracentesis may be preferable. [8] [80] Fluoroscopic and CT scan guidance have also been used successfully. [81]

**Needle or Catheter Insertion for Evacuation of Fluid**

**Patient preparation.**

The patient should be placed in the appropriate position, as described previously. Proper explanation of the procedure is essential, when there is adequate time. If the patient is comfortable and cooperative, sedation is not needed. When the patient is uncooperative and in extremis, restraint may be necessary. If the patient is restrained, access to the insertion site must be clear, and one's ability to manage the patient and the airway must not be compromised. Sedation should be avoided if possible. In cases

in which some sedation is essential, extreme caution and careful monitoring must be done; a rapid-acting or reversible sedative agent should be used. Supplementary oxygen by nasal prongs or mask should be given if it is clinically indicated, and it may minimize postevacuation hypoxia (see Complications). A wide area around the thoracentesis site should be prepared, using povidone-iodine or another suitable antiseptic, to allow selection of several intercostal spaces. Sterile towels should be draped around the site. A patent intravenous line should be established, and atropine sulfate for intravenous administration should be available in the event that the patient has a vasovagal reaction.

**Anesthesia and pleural fluid localization.**

Local anesthesia should be used before removal of pleural fluid. A 1% solution of lidocaine or equivalent anesthetic is usually chosen. The anesthetic (5-10 mL) is drawn into a syringe, and a 25- or 27-ga needle is attached. The previously selected insertion site is relocated by palpation. A small skin wheal is raised at the upper edge of the rib. The syringe is withdrawn, and a 3.75-cm, 22-ga needle is attached and then inserted through the wheal toward the upper border of the rib. The subcutaneous tissue and muscle are alternately aspirated and infiltrated as the needle is advanced down to the periosteum of the rib. At this point, the needle is "walked" above the superior edge of the rib. It is then held perpendicular to the chest and, while the aspiration-infiltration process is continued, is advanced through the intercostal space until the pleura is entered (see Fig. 8-2) (Figure Not Available) . A pop may be felt, and fluid should be aspirated to ensure that the pleural space has been reached. If no fluid is encountered, the chosen intercostal space may be too low, and a higher site may be indicated. If air bubbles are encountered, the lung parenchyma may have been entered; the chosen intercostal space may be too high, and a lower site may be indicated. Once fluid is aspirated, a curved hemostat may be applied to the needle at the skin surface, or the needle may be grasped with the thumb and index finger to indicate the proper depth of penetration. [78] The needle and hemostat or fingers should be removed together.

**Needle insertion techniques.**

The following technique is used for diagnostic thoracentesis when a moderate to large amount of fluid is present. A 3.75- to 5-cm, 18- to 22-ga needle is selected. The smaller-diameter needles may decrease the risk of hemothorax and are usually adequate to remove pleural fluid. [8] The needle and a 50-mL syringe are attached to a three-way stopcock. The syringe and needle may be premoistened with heparin. A drainage tube is attached to the stopcock. The lever of the stopcock is set to allow passage of fluid between the needle and the syringe. The depth of the pleural space as determined from the anesthetic needle is now marked on the larger aspiration needle by gently placing a second hemostat or grasping the needle with the index finger and thumb. The pleural space is again entered through the previous anesthetic site while gentle negative pressure is applied. Attention to the indicated depth will prevent insertion of the needle farther than necessary, thus decreasing the chance of lacerating the underlying lung. Fluid is first aspirated with the syringe. The stopcock lever is then turned, and the fluid is expelled through the drainage tube into a sterile container or sterile vacuum bottle or into open specimen tubes. An assistant is needed to handle the

specimen tubes. The process of aspirating and ejecting the fluid through the drainage tubing is repeated until an adequate amount of fluid to accommodate all needed specimens has been drained.

Thoracentesis performed for diagnostic purposes requires removal of 50 to 100 mL. The specimens obtained are listed in Table 8-5 . It is suggested that the appropriate laboratories be contacted to ensure the optimal collection technique for a specific analysis. It may, for example, be best to collect fluid for cytologic analysis in the morning, to ensure examination of freshly obtained cells. Once the desired amount of fluid has been removed, the needle is withdrawn. A sterile dressing is applied over the insertion site. An upright expiratory chest film is taken to ensure that an iatrogenic pneumothorax was not created.

**Catheter insertion techniques.**

An alternative method

| **TABLE 8-5** -- Diagnostic Pleural Fluid Specimens |
|---|
| **In All Cases:** |
| 6.5-mL plain (red-top) specimen tube |
| Lactate dehydrogenase (LDH) |
| Glucose |
| Protein |
| 5-mL EDTA (lavender-top) specimen tube |

| |
|---|
| Appearance |
| Color |
| Specific gravity |
| Cell counts |
| Differential counts |
| Crystals |
| **If Exudate or Clinically Indicated:** |
| 6.5-mL plain (red-top) specimen tubes |
| Amylase |
| Triglycerides, cholesterol (lipoprotein electrophoresis) |
| Complement levels |
| Rheumatoid factor |

| |
|---|
| Countercurrent immunoelectrophoresis (CIE) |
| Carcinoembryonic antigen (CEA) |
| 10-mL sterile container |
| Gram stain |
| Aerobic cultures |
| Anaerobic cultures |
| Acid-fast bacilli culture and stain |
| Fungal culture and stain |
| 10- to 50-mL plain bottle or (red-top) specimen tubes |
| Cytology 5-mL heparinized (green-top) specimen tube |
| Lupus erythematosus cells |
| 2-mL iced heparinized syringe with air expelled pH |

of removing the pleural fluid can be accomplished by either of the following techniques.

Catheter insertion techniques (see Fig. 8-3) (Figure Not Available) may be preferable when either small amounts of fluid are present and needed for diagnostic analysis or when large quantities are to be removed for therapeutic relief. [8] An *over-the-needle catheter* (16- to 20-ga needle) is attached to the 50-mL syringe. The syringe may be premoistened with heparin. A small scalpel blade to pierce the skin at the selected insertion site may ease entry of the catheter through the skin. The proper depth of the pleural space is indicated on the catheter by grasping it between a gloved index finger and thumb. The needle and catheter are inserted into the pleural space, as previously described. As fluid is encountered, the needle and the catheter are angled slightly caudally. The catheter is advanced into the pleural space, and the needle is withdrawn. The exposed lumen of the catheter hub is covered with a gloved finger to prevent the entry of air. A three-way stopcock with an attached 50-mL syringe and a drainage tube are again attached to the catheter hub. Fluid is removed following the same process for the needle insertion technique. For diagnostic purposes, 50 to 100 mL of fluid should be adequate. If the purpose of the thoracentesis is therapeutic drainage, the fluid is removed in 50-mL aliquots until the respiratory distress appears to be relieved. Although it has usually been recommended that no more than 1000 to 1500 mL of pleural fluid be removed because of the potential for excessive loss of protein or the risk of postevacuation pulmonary edema, [8] [78] more fluid may be removed with gentle negative pressure. Measuring the pleural pressure frequently, using a manometer, may be appropriate if larger quantities are removed [8] [82] (see Complications). Aftercare is the same for catheter drainage as for needle drainage.

A second alternative method involves the use of a *through-the-needle catheter* (14- to 18-ga needle). The catheter is withdrawn from the needle before the procedure. A three-way stopcock is attached to the catheter hub and adjusted to close the catheter to the passage of air or fluid. The catheter and stopcock are temporarily set aside with sterile technique. The empty outer needle is attached to a 50-mL syringe. The syringe may be premoistened with heparin. The depth is again marked on the needle with a hemostat or index finger and thumb, as previously described, to indicate the depth of the pleural fluid as determined from the anesthetic needle. The needle is inserted into the pleural space through the anesthetized area while constant, gentle, negative pressure is applied. Once fluid is encountered, the needle is held securely as the syringe is detached. Again, it is essential to cover the needle hub with a gloved finger. The needle is then held securely and angled caudally as the catheter is inserted through the needle into the pleural space and advanced its full distance. The needle is withdrawn, leaving the catheter within the chest wall. The needle guard is then attached to the needle tip to prevent shearing off of the catheter. The catheter *must not be drawn back through the needle*, because such action may lacerate the catheter and allow its free entry into the pleural space. The catheter is then held securely within the chest wall without bending or kinking. The syringe is reattached to the stopcock, and the stopcock lever is turned to allow passage of fluid into the syringe. Fluid is withdrawn according to the guidelines described for the previous over-the-needle catheter technique. Should repositioning be needed, care must be taken to avoid crimping the catheter. After an adequate amount of fluid has been withdrawn, the catheter is removed and the entry site is covered with a sterile bandage. A follow-up chest film is again indicated.

**Commercial thoracentesis kits and miscellaneous equipment.**

Commercial prepackaged kits have been developed that provide the general equipment needed to perform thoracentesis for the removal of pleural effusion. Most include the basic equipment previously described. Use of a kit saves time. Some kits include equipment such as drainage bags or applicators for the preparation solution. Others may provide unique equipment such as automatic two-way valves, self-sealing diaphragms, or collection apparatus. [83] In general, their drawbacks may include increased cost and limited equipment within the kit. Some may provide brief instructions on technique. It is recommended that each manufacturer's description of thoracentesis technique be consulted and that the equipment be reviewed to allow assembly of any additional equipment before beginning the procedure. The techniques used should follow the guidelines described earlier, depending on the general type of needle or catheter included in the kits.

Occasionally, specialized equipment that has been developed for other purposes can be used for thoracentesis. An example is an "on-off flow switch" produced in England (Viggo) that has been incorporated into a 13-cm over-the-needle catheter for central venous cannulation. The flow through this catheter may be turned off while the introducing needle is removed and a syringe is attached. [84] If the

catheter is used for thoracentesis, the flow switch may serve to prevent the inadvertent creation of a pneumothorax.

**Pigtail catheter insertion.**

A new method of catheter insertion using the Seldinger technique allows the insertion of small pigtail catheters (6.0 to 8.5 Fr) to evacuate fluid. A 16- to 18-ga needle is attached to a syringe and inserted through the appropriate interspace into the pleural space until fluid is encountered. The needle is held securely as the syringe is removed and the exposed hub of the needle is covered. A small, flexible guide wire is then threaded through the needle, which is angled caudally. The needle is removed while the wire remains within the chest. A puncture is made through the skin next to the guide wire with a small scalpel blade. An optional dilator may be threaded over the wire and then removed to enlarge the opening. The pigtail catheter is inserted over the wire and advanced gently into the chest so that all side openings are well within the pleura. The wire is removed and a stopcock and drainage system are attached to the catheter. The catheter should be attached to the chest wall.

## Pleural Fluid Analysis

Whether fluid is removed for diagnostic or for therapeutic purposes, certain specimens should be sent for laboratory analysis based on clinical indications. [85] Of diagnostic thoracenteses, 92% provide clinically useful information. [86]

Whenever thoracentesis is performed, visual inspection of the fluid should be performed. Bloody or blood-tinged fluid most commonly indicates hemothorax, malignancy, pulmonary infarction, or tuberculosis. White or milky fluid suggests a

chylothorax or chyliform effusion (described later). A thick, purulent fluid, often with a foul odor, indicates an empyema. Other effusions may range from clear, to straw-colored, to turbid (Table 8-6) (Table Not Available) .

Establishing whether an effusion is a transudate or an

| TABLE 8-6 -- Diagnostic Features of Pleural Fluid<br>*From Ross DS: Pleural effusion. In Harwood-Nuss AL, Linden CH, Luten RC, et al (eds): The Clinical Practice of Emergency Medicine. JB Lippincott, Philadelphia, 1990. Reproduced by permission.* |
| --- |
| (Not Available) |

exudate provides useful information. If a transudate is diagnosed, it has been suggested that minimal additional testing will be needed. [87] Typically, transudates have protein levels <3 g/100 mL and a specific gravity <1.016. Exudates have values above these levels. Additional criteria have been advocated that provide a more accurate differentiation. These include (1) a ratio of pleural fluid protein to serum protein of 0.5, (2) a pleural lactate dehydrogenase (LDH) level of 200 IU/mL, and (3) a ratio of pleural fluid LDH to serum LDH of 0.6. Transudates have values below these levels, and exudates have at least one finding above these levels (Table 8-7) (Table Not Available) . [88] These recommendations should be tempered by clinical suspicion. In all cases, fluid should be held for possible additional analysis in the appropriate specimen container (see Table 8-5) . [89]

Cytologic analysis is an extremely important diagnostic tool. If abnormal cellular components of pleural fluid are found, it is highly significant. Cytologic analysis may identify malignancies in >60% of cases, [90] [91] although a repeat tap for repeat cytologic analysis along with assay of tumor markers (e.g., carcinoembryonic antigen [CEA]) may be needed. [92] Many specimens may be needed if the suspicion is high. White blood cell (WBC), differential, and red blood cell (RBC) counts on pleural fluid, however, may provide some general information, but their usefulness in differentiating the source of effusions is limited. [90] [91] The highest WBC levels are usually seen with empyema, with values often >50,000/mm [3] . [64] Differential cell counts may be grouped by the predominant cell type, usually reported as polymorphonuclear cells or mononuclear cells (which include lymphocytes). [8] Typical WBC and differential findings in pleural fluid are summarized in Table 8-6 (Table Not Available) .

Other patterns may include eosinophil counts >10% with blood or air within the pleural space as well as with parasitic and fungal disease. [8] An RBC count >100,000/mm3 suggests trauma, malignancy, or pulmonary infarction. [88] A pleural fluid hematocrit may

be performed on bloody effusions. Values greater than half the peripheral hematocrit should indicate a hemothorax. [8]

Numerous chemical analyses of pleural fluid are available and may help differentiate the causes of effusions. Glucose levels are usually low (<60 mg/dL) in malignancy, rheumatoid pleuritis, empyema, or, occasionally, tuberculosis. [8] Amylase levels >160 Somogyi units suggest pancreatitis, pancreatic malignancy, esophageal rupture, or, occasionally, lung cancer. [93] Creatinine levels elevated above the serum

| TABLE 8-7 -- Classification of Pleural Effusions |
| --- |
| *From Ross DS: Pleural effusion. In Harwood-Nuss AL, Linden CH, Luten RC, et al (eds): The Clinical Practice of Emergency Medicine. JB Lippincott Co, 1990. Reproduced by permission.* |
| (Not Available) |

level may be found in pleural effusions due to obstructive uropathy. [94] [95] Pleural fluid lactate 10 mmol/L above blood lactate suggests a pyogenic pleural effusion. [96] The pH of the pleural fluid may be decreased below 7.30 in cases of malignancy, rheumatoid disease, and, occasionally, tuberculosis. This is also particularly helpful in delineating empyemas and complicated parapneumonic effusions that may need thoracostomy tube drainage. Findings of pH <7.2 to 7.3 are considered significant. [8] [64]

Milky fluids due to thoracic duct disruption from trauma or lymphoma are known as *chylous effusions,* which may be differentiated from chyliform effusions seen in tuberculosis, rheumatoid disease, and trapped lung. [88] This is a most important distinction, because chylothorax may require tube thoracostomy. Triglyceride levels >110 mg/dL and chylomicrons, which stain with Sudan III stain, are seen in chylous effusions. Chyliform effusions have elevated cholesterol levels, may demonstrate cholesterol crystals on microscopic examination, and do not stain with Sudan III stain. The previously mentioned lipid values may also be obtained by lipoprotein electrophoresis. It has also been suggested that cholesterol levels >60 mg/dL may help define an exudate. [97]

If clinical suspicion warrants, further laboratory tests of pleural fluid may provide useful information. Reduced complement levels may be found in pleural effusions due to rheumatoid arthritis and systemic lupus erythematosus. Lupus erythematosus cells may be seen on a Wright stain. Rheumatoid factor titers >1:320 suggest a rheumatoid pleural effusion. Elevated CEA levels may be suggestive of adenocarcinoma. [8]

The presence of bacteria may be identified in Gram stains and anaerobic and aerobic cultures of empyema and parapneumonic effusions. Countercurrent

immunoelectrophoresis may also detect the presence of bacterial antigens in parapneumonic effusions. [8] Stains for acid-fast bacillus and tuberculosis cultures may identify tuberculous pleural effusions. Fungal and mycoplasmal cultures may also be obtaine.

# THORACENTESIS IN PEDIATRIC PATIENTS

## Indications

The indications for performing thoracentesis are much the same in children as in adults: relief of a suspected tension pneumothorax, diagnostic tap of a pleural effusion, and therapeutic drainage of a large symptomatic pleural effusion.

Pneumothorax and tension pneumothorax may develop in children for similar reasons as in adults. In all ages, mechanical ventilation and PEEP increase the risk of developing pneumothorax. During the neonatal period, however, the incidence of pneumothorax seems to increase. [98] It occurs with increasing frequency with fetal distress, difficult deliveries, meconium aspiration, cardiopulmonary resuscitation, hyaline membrane disease (respiratory distress syndrome), pulmonary interstitial emphysema, and phenobarbital sedation of low-birth-weight infants. [99] A second contralateral pneumothorax may occur in neonates on positive-pressure ventilation. [100] In the older child, trauma is a common cause of pneumothorax. Spontaneous pneumothorax also occurs and may be associated with asthma, bronchiolitis, cystic fibrosis, staphylococcal pneumonia, empyema, metastatic carcinoma,

dermatomyositis, Ehlers-Danlos disease, and Marfan syndrome. [101] An increased incidence of tension pneumothorax, which may occasionally occur bilaterally, accompanies cystic fibrosis. [102] In children at any age, pneumothorax can progress to tension pneumothorax, particularly in those being mechanically ventilated and those on PEEP.

Pleural effusions may be encountered in the pediatric age group for most of the same reasons as they occur in adults. Juvenile rheumatoid arthritis and systemic lupus erythematosus may also be causes. [103] Parapneumonic effusions are common causes of pleural effusions and are most often due to pneumococcal infection. Empyema usually occurs because of a staphylococcal infection. [101] [104] In the neonate, spontaneous hemothorax may be due to hemorrhagic disease of the newborn, disseminated intravascular coagulation, arteriovenous malformations, and pleural vascular rupture. [75]

### Diagnosis of Tension Pneumothorax

The newborn child with a tension pneumothorax may present with increasing respiratory difficulty marked by irritability, grunting, tachypnea, intercostal and supraclavicular retractions, and nasal flaring. Cervical crepitus may be present. Cyanosis and cardiovascular collapse and decreased mental status may occur. The findings of a tracheal shift and ipsilateral tympany to percussion may be found but are often not

appreciated in the newborn. A hyperinflated, relatively immobile hemithorax and decreased breath sounds may be seen occasionally but are frequently absent. This absence may be due in part to the ease with which breath sounds are transmitted from the uninvolved side in the neonate. A shift in the apical pulse may be a more reliable sign. [8]

In the older child, a pneumothorax usually causes dyspnea and pleuritic chest pain. A tension pneumothorax may reveal decreased breath sounds, tympany to percussion, and tracheal shift. Tachycardia, tachypnea, agitation, use of accessory muscles, nasal flaring, and cyanosis may be seen. Subcutaneous emphysema and cardiovascular collapse should suggest the progression of a tension pneumothorax.

A pneumothorax is best seen on an upright chest film. In the neonate, a cross-table lateral radiograph may be more useful than a supine chest film. [105] Tension pneumothorax is easily recognized on an upright posteroanterior chest film by the absence of pulmonary markings, shift of the mediastinum, and flattening of the diaphragm. Pneumomediastinum and subcutaneous emphysema may be seen. However, it is appropriate to relieve a tension pneumothorax by needle or catheter aspiration when it is recognized clinically. Awaiting radiographic diagnosis is only indicated when the patient's cardiopulmonary status is relatively stable or when the diagnosis of tension pneumothorax is in doubt. The diagnosis may be confirmed by a rush of air and clinical improvement with decompression.

A bilateral tension pneumothorax may be difficult to diagnose clinically. On a chest film, microcardia, flattened diaphragm, and the bilateral absence of lung markings are seen. In such a case, bilateral chest decompression confirms the diagnosis.

**Diagnosis of Pleural Effusion**

In the pediatric population, pleural effusions may be asymptomatic or may appear with respiratory compromise, pleuritic chest pain, cough, dyspnea, or signs and symptoms of underlying systemic illness, such as fever or weight loss. A pleural friction rub may be heard. Bronchial breath sounds may be heard in the infant. [101] A moderate to large pleural effusion may be recognized on physical examination by the presence of decreased breath sounds, dullness to percussion, and decreased excursions of the ipsilateral hemithorax.

Large pleural effusions may be seen on a standard upright chest film displaying the same findings as in the adult. For small effusions, lateral decubitus films may be necessary. Bilateral decubitus views may help assess the underlying lungs as well as whether the effusion is loculated.

**Treatment**

Treatment is directed toward the underlying cause. Supplemental oxygen may be beneficial. The indications for thoracentesis in children include relief of a tension pneumothorax and diagnostic or therapeutic drainage of a pleural effusion. In cases of

parapneumonic effusion from lobar pneumococcal pneumonia, thoracentesis may be withheld. [101]

**Position**

Immobilization of the child may pose a significant problem because of the child's size as well as the frightened child's inability to comprehend the explanation for the procedure. Gentle and simple explanations are appropriate for older children. Sedation may be helpful when respiratory distress is minimal. Oxygen saturation (see Chapter 6) should be monitored if sedation is used. Children should be held securely by an assistant. It is important to position the child as described for adults to provide the proper access to the insertion site and reliable landmarks. To relieve a tension pneumothorax, a child may be positioned supine on a stretcher. The head may be elevated 30°. For removal of pleural fluid, the child should be seated, leaning against the back of a chair or table in the same manner as indicated for adults. If pillows are used, care must be taken to ensure an adequate airway. For the child who is unable to sit up, the lateral decubitus position may be used. A neonate may be held in the "burping" position by an assistant. [106]

**Insertion Site and Procedure**

The insertion sites for relieving tension pneumothorax and removing pleural fluid are much the same as those recommended for adults. The second or third intercostal space in the midaxillary line is usually recommended for relief of a tension pneumothorax. The fourth or fifth intercostal space in the anterior axillary line is also suggested. [106] A needle or catheter may be inserted using the smaller suggested gauge as recommended for adults. The procedure follows the same technique as that used in adults.

Pleural fluid is best removed from the seventh or eighth intercostal space in the posterior axillary line. In children, the seventh intercostal space, located at the tip of the scapula when the arm is slightly elevated, is a reliable landmark. [107] Confirmation by physical examination and chest film is appropriate. A standard needle, a "butterfly scalp vein" needle, over-the-needle catheter, or a through-the-needle catheter may all be used. Again, the smaller-gauge needle suggested for adults is recommended. The techniques for needle or catheter insertion are the same as those described for adults. Pigtail catheters have been used successfully in children to drain both pneumothoraces and effusions. [33] [34] The Seldinger

technique as described earlier for adults is used for inserting the catheters.

Once a tension pneumothorax is diagnosed, a thoracostomy tube is indicated and should be inserted, even if the patient has experienced temporary relief from needle decompression. A thoracostomy tube should also follow thoracentesis if a hemothorax or empyema is found.

## COMPLICATIONS

The most frequent complication caused by inserting a thoracentesis needle or catheter into the thorax from any approach is the *creation* of a pneumothorax. Although many patients already have a small pneumothorax, it may become larger or even become a tension pneumothorax during the procedure. The mechanisms for this complication are a laceration of the underlying lung, an inadequate coverage of the hub of the needle or catheter after the pleural space is entered, an inadequate drainage system, or an air leak in the drainage system or thoracentesis apparatus. The risk of pneumothorax or tension pneumothorax secondary to lung puncture may be increased with patients who are intubated and on positive-pressure ventilation. Iatrogenic pneumothorax also appears to occur more often when performing therapeutic thoracentesis and when malignancy is present. [86] If a pneumothorax is found on a follow-up chest film, tube thoracostomy may also be indicated, according to the criteria in Chapter 9 . Approximately one third of pneumothoraces induced during thoracentesis require a thoracostomy tube. [86]

Cough is another frequently encountered complication (9% of patients). [86] Although seemingly only a symptomatic problem, it may be associated with the creation of an iatrogenic pneumothorax. It has been suggested that on encountering coughing, one should consider terminating the procedure.

Although rare, unilateral pulmonary edema may occur following thoracentesis. It was initially reported following the use of excessive negative pressures in the evacuation of a pneumothorax. [108] [109] Unilateral pulmonary edema has, likewise, been seen following evacuation of a pleural effusion. [110] The pulmonary dysfunction may be the consequence of local hypoxia in the atelectatic lung, with resultant changes in the basement membrane or loss of surfactant, as well as from excessive pleural negative suction pressure. When performing thoracentesis for evacuation of air, one can minimize these changes by applying a passive underwater seal. Any negative suction pressure should be applied gently and may best be accomplished via controlled pressure drainage systems and tube thoracostomy. When removing large pleural effusions, the incidence of postexpansion pulmonary edema may also be decreased by applying gentle evacuation pressures and by frequent measuring of pleural pressures using a column manometer held below the entry site. It is recommended to cease the evacuation if pressures go below -18 cm $H_2O$. [82] Once pulmonary edema develops, administering oxygen is appropriate for relief of hypoxia. Rarely, PEEP may be needed to correct this complication. A case has been reported in which the hypoxia associated with reexpansion pulmonary edema actually improved following the inadvertent redevelopment of the pneumothorax. [111]

Reexpansion hypotension has also been reported following rapid evacuation of persistant unilateral pneumothoraces of at least 1 week's duration. The hypotension occurred in association with unilateral reexpansion pulmonary edema, a rising hematocrit level, and anuria despite relatively normal pulmonary capillary wedge pressures. Intravascular volume depletion and myocardial depression are the suggested mechanisms. [112] [113]

Transient hypoxia has been noted as a consistent finding after thoracentesis. [114] There

may be a ventilation-perfusion mismatch with perfusion of atelectatic lung or areas of localized pulmonary edema. Although the hypoxia usually is not significant clinically, oxygen administration may be indicated, particularly in the patient with minimal respiratory reserve. Oxygen saturation monitoring (see Chapter 6) may be useful in such patients.

Although hemothorax and hemoperitoneum are uncommon, they represent significant potential complications of thoracentesis. Hemothorax may be due to laceration of the lung and diaphragmatic, intercostal, or internal mammary vessels. Cardiac perforation has been reported following thoracentesis in a pneumonectomized patient. [115] Careful attention to technique, such as avoiding the superior portion of the intercostal space, never puncturing medial to the midclavicular line, and not penetrating too deeply into the thorax during needle insertion, should be practiced. Hemothorax may be diagnosed by the rapid accumulation or reaccumulation of fluid as noted on a post-thoracentesis upright chest film. If this occurs, evacuation through a thoracostomy tube is usually indicated according to the criteria indicated in Chapter 9 . Hemoperitoneum may result from puncture of the spleen or liver through the diaphragm. This may occur when thoracentesis is performed with a low posterior approach during expiration. If laceration of intra-abdominal contents is suspected, close observation is essential. A surgical consultation is usually in order, and an exploratory laparotomy may be indicated. Computed tomography may be helpful as an alternative diagnostic approach for suspected intra-abdominal solid organ injury.

As with all surgical procedures, there is a potential for infection. The risk of infection is estimated at 2%. [8] The risk is kept low with proper attention to patient preparation and sterile technique. In the case of rapid relief of a tension pneumothorax, preparation is minimal; however, any risk of infection is greatly outweighed by the risk of cardiopulmonary collapse from a tension pneumothorax.

Inadvertent shearing of the plastic catheter may occur when a through-the-needle catheter technique is used. This complication is prevented by securing the needle with a needle guard after it has been withdrawn from the chest. In addition, the catheter should not be withdrawn back through the needle at any time. Should a catheter tip be left in the chest, a method of percutaneous retrieval using a bent needle tip under fluoroscopy has been described. [116] Some other technical problems of thoracentesis may be encountered. The most common are "traumatic" taps, which are contaminated by blood (11%), and "dry" taps, in which no fluid or inadequate fluid is obtained (9%). [86] Grogan and associates found fewer pneumothoraces and dry taps when thoracentesis for pleural effusion was guided by ultrasonography. [117] Air embolus can occur if the thoracentesis device is left open to air and if the needle or catheter is inadvertently inserted into a pulmonary or intrathoracic blood vessel. Hypoproteinemia may occur after removal of a large pleural effusion. A thick pleural "peel" may accumulate secondary to inadequate

drainage of a hemothorax or an empyema. This complication is avoided by using tube thoracostomy, rather than thoracentesis, to drain blood or empyema more completely. Pleural "peels" may occasionally require thoracotomy and chest decortication.

# Chapter 9 - Tube Thoracostomy

**Seth W. Wright**

Tube thoracostomy is a commonly performed procedure that is used to evacuate an abnormal collection of air or fluid from the pleural space. Normally, the visceral and parietal pleurae are closely approximated. This potential space between the two layers is occupied by only a thin layer of fluid that acts as a lubricant. The pleural space normally has a subatmospheric pressure secondary to the elastic recoil forces of the lung and chest wall. [1] The addition of air, blood, or other fluids to this space disrupts the normal ventilatory mechanism, producing subjective dyspnea and interference with normal gas exchange. The amount of pulmonary and cardiovascular dysfunction is generally proportional to the volume of the abnormal collection and the rate at which it accumulates. Experimental studies of pneumothorax in animals have demonstrated that respiratory and cardiovascular embarrassment result from multiple mechanisms, including respiratory muscle dysfunction, mechanical interference with ventilation, shunting of pulmonary blood flow, ventilation-perfusion mismatch, and increased resistance to blood flow through the pulmonary vascular bed. The end result is a reduction in stroke volume and systemic oxygen delivery. [2] [3] Data from monitored humans with tension pneumothorax further reveal an elevation of pulmonary arterial, central venous, and pulmonary artery occlusive pressures, along with the development of hypoxemia and mild acidemia. [4]

Tube thoracostomy is indicated for a wide variety of medical and surgical conditions (Table 9-1) . The procedure

| **TABLE 9-1** -- Indications for Tube Thoracostomy |
| --- |
| Traumatic conditions |
|   Pneumothorax |
|   Hemothorax |
|   Hemopneumothorax |

| |
|---|
| Open pneumothorax |
| Conditions requiring "prophylactic" surgical management |
| "Occult" pneumothorax |
| Iatrogenic complications |
| Central venous line placement |
| Thoracentesis |
| Pleural biopsy |
| Intercostal block |
| Bronchoscopy |
| Bronchoalveolar lavage |
| Feeding tube placement |
| Positive pressure ventilation |

| |
|---|
| Nontraumatic conditions |
| Primary spontaneous pneumothorax |
| Secondary spontaneous pneumothorax |
| IV drug use-associated pneumothorax |
| Drainage of recurrent pleural effusion |
| Empyema |
| Chylothorax |

is widely used in the emergency department for individuals who have sustained penetrating chest trauma during violent crimes or blunt trauma following motor vehicle accidents or falls. Tube thoracostomy also is used frequently in cases of spontaneous and iatrogenic pneumothorax. In the setting of a tension pneumothorax, the procedure may be immediately life-saving.

## BACKGROUND

The treatment of empyema using incision, cautery, and metal tubes was first described by Hippocrates. However, it was not until the 19th century that modern methods for the evacuation of pleural contents were developed. Continuous intercostal drainage with a water seal for the treatment of empyema was described in the English literature in 1872 [5] and a closed system for empyema drainage, shortly thereafter. [6] However, these techniques were not widely performed until 1917, when they were utilized for the treatment of postinfluenzal empyema. [7] In World War II, closed tube drainage was routinely used following chest injury. It is believed that the increased use of these tubes as both initial and definitive treatment for wartime thoracic trauma led to much of the decrease in the mortality rate. [8] During the Civil War, the mortality rate following thoracic trauma was 62.5%; in World War I, 24.6% of chest wounds were fatal; and in

World War II, the mortality rate following chest trauma dropped to 12%. [9]

# INDICATIONS

## Traumatic Pneumothorax, Hemothorax, or Hemopneumothorax

**Pathophysiology**

Pneumothorax and hemopneumothorax are common after blunt or penetrating thoracic trauma. The North American Major Trauma Outcome Study looked at the prevalence of pneumothorax and hemothorax in patients sustaining major trauma. Among 15,047 trauma patients with thoracic injuries (70% from blunt trauma), a pneumothorax was present in 20%, and 25% had a hemothorax. [10] Pneumothorax following blunt chest trauma is usually attributed to lung puncture from a rib fracture. After trauma, a definite rib fracture may not always be evident on radiographs. In the absence of fractures, pneumothorax is believed to result from rupture of an alveolus secondary to abrupt increases in intrathoracic and intra-alveolar pressures against a closed glottis. The air leak may be self-limited, or a ball valve mechanism may lead to a tension pneumothorax (see later discussion).

Air in the pleural space following blunt trauma is not always secondary to primary lung injury. Esophageal rupture and injuries to the tracheobronchial tree also may be responsible for pneumothorax and are often manifested by persistent air leaks; occasionally food particles in the thoracostomy tube drainage may be noted. Penetrating injuries result in simple pneumothorax by allowing air to enter the chest, either through a persistent chest wall defect or by direct injury to underlying lung.

Hemothorax may result from bleeding from the heart,

lungs, great vessels or their branches, intercostal arteries or veins, mediastinal veins, diaphragm, chest wall vessels, fractured ribs, or torn pulmonary adhesions. Bleeding from the lung parenchyma is usually self-limited because of the relatively low-pressure vascular supply of the lung and the high concentration of tissue thromboplastins. In addition, reexpansion of the collapsed lung generally tamponades the low-pressure bleeding sites. Partially severed intercostal arteries bleed particularly briskly, because all but the first two come directly from the aorta. In addition, a partially lacerated internal mammary artery rarely ceases bleeding without surgical intervention.

**Diagnosis**

**Physical examination.**

Alert patients with a pneumothorax, hemothorax, or hemopneumothorax may complain of chest pain, usually pleuritic in character, and shortness of breath. Pneumothorax alone may be manifested by increased resonance to percussion, decreased tactile fremitus, decreased or absent breath sounds, or subcutaneous (SQ) emphysema. As a general rule, air in the SQ tissues (SQ emphysema) originates from an internal

structure, such as lung, bronchi, or esophagus, although it may represent air introduced from the ambient environment following a penetrating thoracic wound. On occasion, SQ emphysema following blunt trauma may not be associated with a pneumothorax. With an isolated hemothorax, breath sounds will be decreased as well, but the percussion note is dull. A succussion splash has been described when air and fluid are both present. It is important to note that *physical examination is often misleading, and many pneumothoraces may go undetected by physical examination alone*.

Hemothorax alone also may be difficult to detect. Patients can rapidly lose 30 to 40% of their blood volume into the pleural space, with little resistance from the compliant lung. They may then present primarily with signs of shock. The presence of shock in a patient with a chest injury should also raise the question of pericardial tamponade, which may not be manifested by distended neck veins in the hypovolemic patient. Evidence of mediastinal shift or compression (distended neck veins, shifted trachea) may represent massive blood or fluid accumulation or, more likely, tension pneumothorax. In contrast, a small hemothorax (<400 mL) may produce few clinical findings. Physical findings of a hemothorax include dullness to percussion or loss of breath sounds at the lung base.

**Radiographic diagnosis.**

A standard, 6-foot posteroanterior (PA) upright chest radiograph is the diagnostic procedure of choice for hemothorax or pneumothorax. However, radiographic studies may be limited to a portable supine anteroposterior (AP) view in the critically ill patient. A pneumothorax is diagnosed more readily on the *expiratory* radiograph, because the lung collapses further. A pneumothorax may also be accentuated with a lateral decubitus film (unaffected side down, injured side up). One can often obtain a sitting (portable) 6-foot PA film in patients (who may require some assistance) by having them sit up and "hug" the x-ray plate to their chest. The machine is placed at the standard distance behind the patient. This view more closely approximates standard technique and allows a better evaluation of mediastinal size. Otherwise, AP films should be taken with the patient sitting in as erect a position as possible.

Computed tomography (CT) scans of the thorax and upper abdomen may be superior to plain radiographs for the detection of a small hemothorax or pneumothorax. Garramone and coworkers [11] reported the unexpected finding of a pneumothorax in 26 of 457 patients undergoing abdominal CT scanning for the evaluation of trauma. None were apparent on prior x-rays of the chest. Poole and colleagues [12] compared plain films with chest CT scans in blunt chest trauma patients. The CT scan detected significantly more pneumothoraces (23% vs 14%) and more pleural effusions (34% vs 14%) than did plain films. However, the findings seen only on CT scans were invariably small and unassociated with complications.

When one is interpreting a supine chest film, it is important to compare the relative densities of both lung fields. On a supine chest radiograph, as much as 1000 mL of blood may be manifested only as a *slight homogeneous increase in the density of one hemithorax*. Up to 500 mL of blood may be required to produce blunting of the costophrenic angle on an upright film. Subpulmonic collections can resemble an elevated hemidiaphragm. Lateral decubitus films may be required to demonstrate either

of these findings. *If an air-fluid level is seen* (straight line to the top of the fluid collection), as opposed to a fluid meniscus, *a pneumothorax must also be present* (Fig. 9-1) .

The size of the pneumothorax can be estimated by the method outlined in Figure 9-2 . [13] The area of the collapsed lung is subtracted from the area of the involved hemithorax. The area of the collapsed lung can be calculated in the figure using the area of a rectangle drawn to include the extreme superior ( *a*), lateral ( *b*), and inferior ( *c*) margins of the collapsed lung and the center of the mediastinum ( *d*). This number is then subtracted from the area of a rectangle encompassing the entire affected hemithorax, measured from the inferior border of the first rib ( *A*), the inner border of the midlateral chest wall ( *B*), the tip of the costophrenic angle ( *C*), and the center of the mediastinum ( *D*). The difference between the area of the hemithorax and the area of the collapsed lung is divided by the area of the hemithorax to yield the percentage of the pneumothorax.

Alternatively, the size of a pneumothorax can be estimated by calculating the average interpleural distance as described in Figure 9-3 (Figure Not Available) . [14] The average interpleural distance is the mean of the maximal apical interpleural distance and the interpleural distances of the upper and lower lung fields. The average interpleural distance is then correlated with the percentage of the pneumothorax size by means of the nomogram in Figure 9-4 (Figure Not Available) .

A simpler way of monitoring the size of the pneumothorax involves measuring from the lateral lung margin to the lateral chest wall on full inspiration. It is important to have some easily reproducible means of repeatedly recording the size of a pneumothorax, particularly if the patient is to be examined by different physicians and management is to be based on serial measurements of pneumothorax size.

**Figure 9-3** (Figure Not Available) Calculation of percent pneumothorax using the average interpleural distance method. The base of the lung is not seen in the posteroanterior (PA) view. The total height of the PA view is assumed to be the same as that shown on the lateral view. *(From Rhea JT, DeLuca SA, Greene RE: Determining the size of pneumothorax in the upright patient. Radiology 144:733, 1982. Reproduced with permission.)*

**Treatment**

In a patient in extremis with evidence of major thoracic trauma (SQ emphysema, palpable rib fractures, flail chest), no further diagnostic studies need be undertaken. Needle aspiration may confirm the diagnosis but can be misleading or may even cause a pneumothorax. If a tension pneumothorax is suspected, needle aspiration may be a temporary solution, but subsequent tube thoracostomy is indicated on the suspected side of injury.

Tube thoracostomy allows egress of the fluid or air and provides a means of continuous drainage and monitoring of the pleural space. Further drainage can be quantified, and the need for other intervention can thus be assessed. Observation of the drainage and collection devices allows for the diagnosis of persistent air leak or other problems that

may require additional treatment.

Not all patients with a pneumothorax require tube thoracostomy. Small pneumothoraces and hemothoraces (<400 mL) have been treated successfully with observation alone if the patient is stable, relatively asymptomatic, otherwise healthy, and not likely to require positive-pressure ventilation. [15] Resolution occurs over 2 to 3 weeks. [16] [17] Northfield has suggested that supplemental oxygen will increase the rate of pleural air absorption several-fold, [18] and such additional

**Figure 9-4** (Figure Not Available) Nomogram for the prediction of pneumothorax size from an average interpleural distance (see text). The intrapleural distance is measured in centimeters from the outer edge of the collapsed lung to the inner aspect of the chest wall (i.e., parietal pleura to visceral pleura). *(From Rhea JT, DeLuca SA, Greene RE: Determining the size of pneumothorax in the upright patient. Radiology 144:733, 1982. Reproduced with permission.)*

therapy should be considered. The placement of a chest tube in stable patients with a small pneumothorax is based on clinical assessment of the potential benefits and risks associated with the individual case. Patients with larger or more symptomatic traumatic hemothorax, pneumothorax, or hemopneumothoraces should have a thoracostomy tube placed.

A tube may be placed "prophylactically" in a patient with evidence of a penetrating injury to the chest, even without demonstrable intrathoracic injury, if anesthesia and positive-pressure ventilation are required or if the patient will be transported a long distance for definitive care of other injuries. Such patients are at high risk for developing a tension pneumothorax when subjected to positive airway pressures. They may also develop a simple or tension pneumothorax during transport when diagnostic adjuncts are suboptimal or definitive treatment may be difficult or impossible.

With the increased use of CT scans for evaluation of thoracic and abdominal trauma, it is clear that many patients with previously unrecognized small pneumothoraces have safely undergone general anesthesia with positive-pressure ventilation without developing a clinically evident pneumothorax. [11] [12] Therefore, whether all patients with a small pneumothorax (i.e., evident only on CT scan) secondary to trauma require a "prophylactic" chest tube when undergoing positive-pressure ventilation is currently controversial. Garramone and coworkers suggest that routine chest tube placement can be avoided if the pneumothorax is small and there are no associated rib fractures. [11] If a chest tube is not placed in a patient with a small, CT-evident pneumothorax and positive-pressure ventilation is administered, close observation for signs of a tension pneumothorax is mandatory.

A recent animal study disputes an old assumption that blood in the chest may tamponade a briskly bleeding vessel and that pleural drainage should be delayed until after volume resuscitation. [19] Indeed, hypoxia and hypercapnia may result if pleural drainage is not instituted quickly. However, early institution of blood replacement is recommended in patients with massive hemothorax (>2000 mL), since massive hemothoraces are often associated with ongoing hemorrhage. Autotransfusion using a commercial device for the collection, filtration, anticoagulation, and autotransfusion of blood obtained by tube thoracostomy (see Chapter 28) is indicated in instances of

massive hemothorax.

Many patients (72 to 82%) with traumatic hemothorax can have their injuries managed successfully by tube thoracostomy and volume replacement alone. [20] [21] [22] In the remaining patients, immediate or delayed elective thoracotomy may be required. There is disagreement among different researchers concerning the indications for surgery. Table 9-2 provides a summary of surgical indications.

**Open Pneumothorax**

Open pneumothorax ("sucking chest wound"; Fig. 9-5) most commonly results from shotgun or combat injuries that result in a loss of chest wall integrity. Such wounds can produce markedly deficient gas exchange and cardiovascular function when the negative intrapleural pressure is replaced with atmospheric pressure. If the chest wall defect is larger in cross-sectional area than the trachea, air will move preferentially through the chest wall with diaphragmatic excursions, and no ventilation will occur. With smaller defects,

| **TABLE 9-2** -- Indications for Surgery after Tube Thoracostomy |
| --- |
| Massive hemothorax (i.e., >1000-1500 mL initial drainage) |
| Bleeding |
|   Rapid (i.e., >300-500 mL in first hour) |
|   Continued (i.e., >200 mL/hour for first 3 or more hours) |
|   Increasing size of hemothorax on chest film |
| Persistent hemothorax |

| |
|---|
| After 2 functioning tubes placed |
| Clotted hemothorax |
| Large air leak preventing effective ventilation |
| Persistent air leak after placement of second tube or inability to expand lung fully |
| Documented bronchial injury |
| Ruptured esophagus |
| Ruptured diaphragm |
| Upper mediastinal entrance wound |
| Pericardial tamponade |
| Cardiac arrest secondary to penetrating trauma |
| Great vessel injuy |
| Cardiac injury |

| |
|---|
| Open pneumothorax |
| Gross intrapleural contamination from a foreign body |

a tension pneumothorax (see subsequent discussion) may develop. Clinically, a chest wall defect and SQ emphysema are seen in a patient with marked respiratory distress.

Prehospital treatment involves the application of a (preferably sterile) dressing to act as a one-way (flap) valve, allowing air to exit the pleural space while blocking reentry. In the field, anything (e.g., a palm, plastic wrap, or gauze) can be used. The patient is instructed to perform the Valsalva maneuver after deep inspiration or to cough just as the dressing is placed. Ideally, a sterile dressing of petrolatum-impregnated gauze extending 6 to 8 cm beyond the wound in all directions is used. This underlying dressing should be covered by gauze dressings and secured on three sides only. An airtight dressing could predispose to tension pneumothorax in the presence of a continued intrapleural air leak. This dressing may be sealed after tube thoracostomy through a separate site is performed to allow continued evacuation of air or fluid. The presence of the open wound is an indication for operative debridement and closure of the chest wall defect with continued tube drainage of the pleural space.

## Spontaneous Pneumothorax

### Pathophysiology

Spontaneous pneumothorax (Fig. 9-6) can be divided into primary and secondary spontaneous pneumothorax. [23] Primary spontaneous pneumothorax results from the rupture of a subpleural bleb in a patient who is otherwise without underlying intrathoracic disease. The male-to-female ratio is 6:1, and the typical patient is 20 to 40 years of age and is tall and thin. Less than 10% of primary pneumothoraces occur with exercise. [17] Large case series have documented that spontaneous pneumothorax is primarily a disease of cigarette smokers. [16] [17] A Swedish study [24] has demonstrated that smoking increases the risk of spontaneous pneumothorax 9-fold in men and 22-fold in women. They determined that the life-span risk of developing a spontaneous pneumothorax for heavy smokers is approximately 12%, compared

with a risk of only 1 in 1000 in people who have never smoked. In the United States, the age-adjusted yearly incidence of primary spontaneous pneumothorax is 7.4 per 100,000 males and 1.2 per 100,000 females. [25]

Patients with secondary spontaneous pneumothorax have underlying lung or pleural disease as the cause of the pneumothorax. The most common predisposing factor is emphysema. [23] Other associated potential etiologic factors include chronic bronchitis,

asthma, tuberculosis, pneumonia, bronchiectasis, atelectasis, pulmonary fibrosis, lymphoma, and various connective tissue diseases such as scleroderma or eosinophilic granuloma. Tuberous sclerosis, rupture of a hydatid cyst, pulmonary infarction, foreign body, and alpha1 -antitrypsin deficiency have all been implicated. Neoplasm is a consideration in older patients. Recurrent pneumothoraces attributed to pleural diaphragmatic endometriosis (i.e., catamenial pneumothoraces) occur in some women at the time of menstruation. [26] Patients with acquired immunodeficiency syndrome and *Pneumocystis carinii* pneumonia also have been found to be at increased risk for spontaneous pneumothorax. [27]

The rupture of an emphysematous bleb, an alveolar septum, or the bronchial wall causes air to flow freely into the pleural space. The majority of cases occur when the patients are at rest, and 70% of affected individuals seek medical attention within 24 hours of onset. Case series have reported that 0 to 11% of cases are bilateral. [28] [29] The symptoms correlate with the amount of lung collapse, the mobility of the mediastinum, the amount of respiratory reserve, the presence of underlying disease, and the degree of compression of the rest of the lung. A spontaneous pneumothorax may progress to a tension pneumothorax, although such progression is unusual.

**Diagnosis**

Almost all patients with a spontaneous pneumothorax (95%) complain of chest or shoulder pain (or, rarely, back or abdominal pain) that is usually sudden in onset, sharp, pleuritic, and cutting. Tightness may be described. Sixty percent have dyspnea in addition to these symptoms, and 12% have a mild cough. [16] [30] Breathlessness and anxiety are more common in older patients, and morbidity and mortality are increased in patients with underlying disease. [17] Various degrees of respiratory distress may be manifested. SQ emphysema rarely occurs. Decreased breath sounds, decreased tactile fremitus, and hyperresonance to percussion may be noted in an uncomplicated spontaneous pneumothorax, although the physical examination may reveal no abnormalities if the pneumothorax is small.

The diagnosis is made on a standard upright PA chest film. Small pneumothoraces are best seen by radiography with the patient in full expiration. During expiration, the volume of air in the pleural space remains the same, but the expiratory decrease in the volume of the collapsed lung on the affected side increases the apparent relative size of the pneumothorax. A lateral film helps rule out complications and may help define the etiology when the pneumothorax is secondary to some other intrathoracic pathology. In pediatric

patients or in those who are uncooperative for other reasons, radiographs in both decubitus positions may successfully demonstrate a small pneumothorax. If the patient is placed in the lateral decubitus position with the affected side up, air will collect in the uppermost portion of the pleural space, and the pneumothorax may be more perceptible.

In the patient with chest pain, pneumothorax must be differentiated from myocardial infarction, dissecting aneurysm, pericarditis, pneumonia, pneumomediastinum,

pulmonary embolus, spontaneous esophageal rupture, perforated peptic ulcer, and biliary or renal colic. Radiographically, giant bullae or lung cysts may mimic a pneumothorax, and these must be carefully differentiated, usually by obtaining a decubitus view.

**Treatment**

The treatment varies with the age of the patient, the symptoms, the degree of respiratory compromise, the bilaterality, the need for general anesthesia, the size of the pneumothorax, and whether the current episode represents a recurrence. *From a purely physiologic viewpoint, all patients with a pneumothorax do not automatically require pleural space aspiration.* However, exactly when to treat becomes a clinical judgment issue, and compliance, the need for close follow-up, or other mitigating circumstances may be such that placement of a chest tube is the safest and most expedient course. Otherwise healthy, asymptomatic, reliable patients with small pneumothoraces (<10% or <1 cm collapse laterally) may be treated by observation alone. As previously noted, supplemental oxygen may hasten the resolution of a pneumothorax, and its use should be considered. [18] A period of observation (with the length depending on the amount of time the pneumothorax has been present) is recommended to ensure that the pneumothorax is not expanding. Reexpansion is estimated to occur at 1.25% of lung volume daily. [13] Affected patients who are released home following a period of emergency department observation must be instructed to return immediately if symptoms increase, to minimize their activities, and to have follow-up chest films to document resolution.

Needle or catheter aspiration of a pneumothorax has been advocated by some and has had a variable degree of success (see later discussion). Traditional therapy for a spontaneous pneumothorax consists of tube thoracostomy and water seal drainage with or without suction. The tube evacuates the intrapleural air, prevents further accumulation, and allows monitoring for persistent air leaks. The local irritation produced by the tube is believed to aid in scar formation and in preventing recurrence. The tube is left in place for 24 hours after all evidence of continued air leak has disappeared.

A number of investigators have reported successful outpatient management of chest tubes in about 75% of their patients with spontaneous pneumothorax. [31] [32] [33] Stable patients are selected; these individuals should be free of significant underlying disease or persistent air leaks and should have satisfactory lung reexpansion after 1 to 12 hours of observation in the emergency department (ED). They are sent home with a flutter (Heimlich) valve attached and are seen 3 to 4 days later for tube removal if complete reexpansion is maintained.

Surgical treatment (usually thoracotomy with abrasion of pleural surfaces) is advocated at the time of the first or second recurrence. [33] [34] Patients who have had one pneumothorax have a 30 to 50% chance of ipsilateral recurrence within 1 to 2 years. [33] After a second spontaneous pneumothorax, the probability of a third rises to 50 to 80%. [33] [35] Surgery may be recommended on the occasion of a patient's first pneumothorax in a number of situations: life-threatening tension pneumothorax, massive air leaks with

incomplete reexpansion, an air leak persisting 4 to 5 days after a second intercostal tube has been placed, associated hemothorax with complications, cases of identifiable bullous disease, and failure of easy reexpansion in patients with cystic fibrosis.

## Tension Pneumothorax

### Etiology

Tension pneumothorax (Fig. 9-7) may be a complication of both spontaneous pneumothorax and traumatic hemopneumothorax. Rarely, a pneumothorax that has been stable for a number of days will rapidly develop tension. Fractures of the trachea or the bronchi, the presence of an occlusive dressing over an open pneumothorax, or a ruptured esophagus also may result in tension pneumothorax. The risk is markedly increased in patients with chest trauma who are undergoing positive-pressure ventilation. Because of this, a patient with penetrating thoracic injury (even without immediate evidence of intrathoracic bleeding or air) may be a candidate for a "prophylactic" chest tube placed before surgery. A "tension hemothorax" does not occur in the absence of massive fluid replacement, because the volume of blood required to produce a shift of mediastinal structures requires nearly total exsanguination into the chest. Rarely, a malignant pleural effusion will cause a tension hydrothorax. [36]

### Pathophysiology

Classically, a pulmonary parenchymal or bronchial tear creates a ball valve mechanism. The increased endobronchial diameter and intrathoracic negative pressure during inspiration allow air to pass into the pleural space. The decrease in bronchial diameter and the relatively elevated intrathoracic pressure during expiration cause the leak to close. This mechanism traps increasing amounts of air in the pleural space with each respiratory cycle. As intrapleural pressure rises, venous return to the right heart declines, and cardiac output drops. The mediastinum shifts toward the uninvolved side, mechanically interfering with right atrial filling. Ventilation of both the involved lung and the opposite lung is compromised, and hypoxemia, acidosis, or both result from ventilation-perfusion inequalities. Tension pneumothorax may develop at any time after injury, during resuscitation, or with cardiopulmonary resuscitation (CPR). It should be considered as a possible cause for deterioration in any susceptible patient, particularly if positive-pressure ventilation is being used.

### Diagnosis

Clinically, patients with tension pneumothorax may present with or rapidly develop restlessness, dyspnea, agitation, or cyanosis. Hypotension, tachypnea, tachycardia, nasal flaring, and retractions may occur. Pulsus paradoxus may be evident. There is hyperresonance to percussion and decreased breath sounds on the affected side. Obvious chest trauma, rib fractures, or SQ emphysema should alert one to the possibility of tension pneumothorax. The trachea and the cardiac apex are displaced toward the uninvolved side. Neck veins may be distended but can be flat in the hypovolemic patient. The prominent, fixed, and overinflated hemithorax may be obvious

when the semi-upright patient is inspected from the head or the foot of the bed. Increased airway resistance in an intubated patient (one of the first signs of a tension pneumothorax) may be manifested by increased difficulty in manual ventilation or increased ventilatory pressures when a volume-cycled respirator is used. Although the clinical scenario generally does not permit the luxury of a chest radiograph, the findings of a depressed hemidiaphragm and lung collapse on the affected side with a shift of the mediastinum to the opposite side is classic.

Pericardial tamponade figures strongly in the differential diagnosis and should be considered in the trauma patient when mediastinal shift, hyperresonance, and decreased breath sounds are not prominent clinical features or when needle aspiration of the hemithorax fails to bring prompt relief.

**Treatment**

If the patient is in extremis and the diagnosis is suspected clinically, needle aspiration of the involved side should be undertaken without further delay (see Chapter 8) . Even if no facilities are available for chest tube placement, a large needle inserted into the chest to convert a tension pneumothorax to an open pneumothorax can be life-saving. A 14-ga needle is commonly placed in the second anterior intercostal space but is effective anywhere in the pleural space. A temporary flutter valve may be fashioned from the fingers of a rubber surgical glove until definitive treatment is available. The definitive treatment of a tension pneumothorax is tube thoracostomy.

## Other Indications

**Occult Traumatic Pneumothorax**

In many institutions CT of the abdomen is the procedure of choice for the diagnosis of intra-abdominal injuries following blunt trauma. Abdominal CT routinely includes the lower aspect of the thorax, and small pneumothoraces are easily visualized. Occult traumatic pneumothorax is defined as a pneumothorax that is not evident on a conventional chest radiograph, but is visible on CT. The management of these small pneumothoraces has been controversial, with some advocating immediate tube thoracostomy [37] and others in favor of expectant management in selected patients. [11] [38] One prospective randomized study found that patients with an occult pneumothorax who require positive-pressure ventilation are at risk for progression of the pneumothorax and development of tension pneumothorax. [39] In patients not requiring positive-pressure ventilation, expectant management may be appropriate if the patient is hemodynamically stable. [11] [12] In these patients, close observation and interval radiography is mandatory, and a qualified physician must be immediately available to place a chest tube, if needed. [38]

**Pneumothorax Associated with IV Drug Use**

Pneumothorax has been described following the inhalation of drugs, usually marijuana or cocaine. The barotrauma that follows recreational drug inhalation is likely caused by

the prolonged Valsalva maneuver used to increase the effect of the drug. [40] A common complication seen in IV drug users is a pneumothorax resulting from injecting into the central venous circulation. These "pocket shooters" typically have no peripheral venous access because of sclerosis of their veins. The "pocket shot" involves the central or middle approach to the internal jugular vein and a pneumothorax is a common complication due to the proximity of the apex of the lung. [41] The patient will present with the typical signs and symptoms of a spontaneous pneumothorax. Treatment of these patients is discussed below.

**Drainage of Recurrent Pleural Effusions**

Initially, most pleural effusions can be managed by thoracentesis (see Chapter 8) , but recurrent effusions may require tube thoracostomy.

**Empyema**

Empyema was one of the first recorded indications for continuous intercostal drainage in adults [6] and children [5] and remains a prominent one today.

**Chylothorax**

Chylothorax can be a rare complication of thoracic trauma or central line placement with resultant thoracic duct injury. It may be seen with penetrating injury or a fall from a height. Chyle collects extrapleurally, and 2 to 10 days may elapse before it enters the pleural cavity. Initially the few clinical manifestations may be masked by other injuries. As fluid accumulates in large amounts, dyspnea and the physical findings of a pleural effusion become prominent. Thoracentesis reveals a milky white liquid with a high lymphocyte count, 4 to 5 gm/dL of protein, and a high fat content. Repeated thoracentesis or tube thoracostomy is combined with bedrest and parenteral alimentation until the volume of chyle declines. [42]

**Postoperative Thoracotomy**

Chest tubes are nearly always placed under direct vision when open thoracotomy is performed.

**Iatrogenic Pneumothorax**

The leading causes of iatrogenic pneumothorax are transthoracic needle aspiration, subclavian vein catheterization, and thoracentesis. [43] Patients with a minimal pneumothorax have been managed successfully with observation and supplemental oxygen. Those with a small pneumothorax have been treated variably (e.g., catheter placement, pneumothorax aspiration and Heimlich valve use vs small chest tube placement). Those with large or recurrent pneumothoraces or those undergoing positive-pressure ventilation generally receive formal tube thoracostomy. Despars and coworkers [44] found that 66% of patients with an iatrogenic pneumothorax were treated with a chest tube for a mean duration of 4.7 days. Nine of 65 (14%) patients required a

second chest tube, and 2 deaths were attributed to an iatrogenic pneumothorax in their series.

## CONTRAINDICATIONS

A list of contraindications is provided in Table 9-3 . There are probably no absolute contraindications in the compromised patient who requires the procedure, although some relative contraindications exist. Multiple pleural adhesions, emphysematous blebs, and scarring should mandate caution in a stable patient. It is important to note that a giant emphysematous bleb or bulla in adults and congenital lobar emphysema in infants may be extremely difficult to differentiate from a pneumothorax on chest films. A second or third spontaneous pneumothorax in a stable patient may be an

| **TABLE 9-3** -- Relative Containdications to Tube Thoracostomy |
| --- |
| Multiple adhesions, blebs |
| Recurrent pneumothorax mandating surgical treatment |
| Need for immediate open thoracotomy |
| Bleeding dyscrasia |

| **TABLE 9-4** -- Instrument Tray for Tube Thoracostomy |
| --- |
| Preparation razor |

| |
|---|
| Sterile towels--4 |
| Basin for preparation solution |
| Gauze pads |
| Towel clips (optional)--4 |
| 10- to 20-mL syringe and assorted needles for infiltration of local anesthetic |
| Large, straight (suture) scissors |
| Large, curved (Mayo) scissors |
| Large clamps (Kelly)--2 |
| Needle holder |
| No. 0 or 1-0 silk on large cutting needles--several |
| Knife handle No. 4--1 |
| No. 10 scalpel blades |

| |
|---|
| Forceps |

indication to proceed directly to surgery instead of attempting another tube thoracostomy. A patient requiring immediate open thoracotomy (e.g., in the case of cardiac arrest after penetrating trauma) may not benefit from chest tube placement. The presence of a massive hemothorax usually requires rapid blood or crystalloid replacement with or without immediate surgery. The setting of bleeding dyscrasias before clotting factor replacement may be a relative contraindication.

## EQUIPMENT

### Instruments

The instruments required for performing tube thoracostomy by the method detailed below are listed in Table 9-4 . Prepared "trays" are available in hospitals and often contain many more instruments than are required or described here. In addition to the instruments, a number of other materials are needed; these are listed in Table 9-5 . All the necessary items should be assembled and tested before the start of the procedure. If the tape is torn as desired, the solutions are poured, the packages are opened, and a checklist (mental or written) is reviewed before beginning, the procedure will go much more smoothly.

| **TABLE 9-5** -- Other Materials Required for Tube Thoracostomy |
|---|
| Local anesthetic |
| Antiseptic solution |
| Arm restraints (padded) |
| Petrolatum-impregnated gauze |

| |
|---|
| Tincture of benzoin |
| Adhesive tape--cloth-backed |
| Chest tubes |
| No. 28 to 36 Fr for adults |
| No. 16, 20, 24 Fr for children |
| Clear, sterile plastic tubing in 6-foot lengths, 1/2-inch diameter |
| Hard plastic serrated connectors |
| Drainage apparatus with sterile water for water seal |
| Y connectors |

**Chest Tubes**

The size, shape, and characteristics of tubes used for thoracostomy vary considerably. The most commonly used devices are clear plastic, straight tubes with a series of holes at one end. They have a radiopaque strip that is commonly interrupted by the last fenestration. Sizes used for adults vary from No. 12 to 42 Fr. Most authorities believe that No. 20 to 32 Fr tubes are adequate for a pneumothorax alone, although small catheters (8 Fr) have been suggested, especially if the patient is to be treated and released. Larger tubes (a minimum of 36 Fr) are best when blood or pus is to be drained. It is a mistake to attempt to drain a hemothorax with a small chest tube. For

pediatric patients, No. 16, 20, and 24 Fr tubes are adequate.

# PROCEDURE

The ideal procedure performed under ideal circumstances is described. The degree of urgency, as determined by the patient's condition and the available resources, will dictate how closely to the ideal a particular chest tube insertion will come. Under the best circumstances, the diagnosis should be established before the procedure and the appropriate radiographs taken. The preferred films are upright PA and lateral chest radiographs. The nature and necessity of the procedure should be explained to the patient as completely as possible, and it is preferable to obtain and document informed consent.

**Tube Location**

The classic approach has been to place tubes anteriorly in the second intercostal space, midclavicular line (usually 2 inches from the lateral border of the sternum) for pneumothorax alone, and dependently in the midaxillary or posterior axillary line, and then direct them posteriorly for fluid removal (Fig. 9-8) . *In an emergency, a tube placed anywhere in the pleural space should be adequate.* The anterior second intercostal space is nearly always mentioned as a location for tube placement but in practice is less often used. Disadvantages include the need to dissect through several inches of muscle mass and the resulting unsightly and highly visible scar.

It is probably reasonable to avoid the second intercostal space entirely and use only midaxillary line placement for all clinical indications. It is cosmetically preferable and better tolerated and is believed to result in increased pleural involvement and scarring. If the tube is placed slightly anterior to the midaxillary line, the patients may lie on their backs more comfortably. Hegarty randomly placed tubes in the second intercostal space, midclavicular line, or the fifth intercostal space, midaxillary line, in 131 cases of pneumothorax or combined hemopneumothorax. He found that the time to removal of the tube was not influenced by location, regardless of whether air, blood, or both, was being drained. [45] Another study looked at 156 randomly placed chest tubes and found no unsatisfactory results with pneumothorax, regardless of tube position. [46] Logically, as the collapsed lung expands and the parietal and visceral pleurae become more tightly opposed, either air or fluid will follow the path of least resistance and enter a functioning drainage

**Figure 9-8** Standard sites for tube thoracostomy. *A,* The second intercostal space, midclavicular line. *B,* The fourth or fifth intercostal space, midaxillary line. Most clinicians prefer midaxillary line placement for all chest tubes, regardless of pathology. Note that placing the tube too far posteriorly will not allow the patient to lie down comfortably.

tube, regardless of the tube's location. Although some clots may remain in the pleural space, blood that is drained from the chest cavity has no demonstrable fibrinogen. [47] Therefore, a tube anywhere in the pleural space should adequately drain a hemothorax

of unclotted blood, as long as there are no adhesions.

Specific recommendations for lateral placement have varied from anterior to posterior axillary lines and from the fourth to the eighth intercostal space. If time permits and the physician who will subsequently be caring for the patient can be consulted, the physician's preference should be followed. For all clinical indications for routine placement in the emergency department, the fourth or fifth intercostal space slightly anterior to the midaxillary line is suggested for chest tube placement. This is roughly at the level of the nipple or the inferior scapular border in most patients. Placement of the tube posterior to the midaxillary line may result in significant patient discomfort when the patient reclines. The tube is directed posteriorly and toward the apex of the lung. This has proved satisfactory for drainage of either fluid or air. If fluid continues to accumulate, a second tube may be placed in the posterior axillary line at the same interspace or one interspace lower.

The location of the diaphragm varies with the position of the patient and can rise quite high when the patient is supine. The phase of respiration and other associated injuries (diaphragmatic hernia, abdominal distention) also can alter its position. The most reliable means of preventing inadvertent damage to lung or abdominal viscera is a thorough digital exploration of the pleura before tube insertion. If the patient breathes during this procedure, the location of the diaphragm can often be verified, and intrapleural placement can be ensured.

## Tube Insertion

### Patient Preparation

For axillary insertions, the patient is ideally placed with the head of the bed elevated 30° to 60° (Fig. 9-9) . Inserting a

**Figure 9-9** To insert a chest tube, the patient is placed semi-erect with the ipsilateral shoulder abducted. Supplemental oxygen is suggested, as is the liberal use of appropriate parenteral analgesia and/or sedation.

chest tube while the patient is lying flat increases the chances of injury to the diaphragm, the spleen, or the liver and presents a more difficult operating position for the practitioner. When the patient is lying down, the diaphragm may rise as high as the third intercostal space. The arm on the affected side is placed over the patient's head and restrained in that position with wide strips of adhesive tape or by other means. The other arm can be restrained comfortably at the patient's side. (Even in the conscious and seemingly cooperative patient, judicious use of comfortable restraints before painful or frightening procedures often allows more rapid and efficient procedural completion.) For anterior midclavicular line placements, the patient's arms may be placed at the sides.

After identifying the area of tube placement, the surrounding skin should be shaved if necessary. The area is sterilized with a povidone-iodine solution or another suitable

antiseptic and draped with sterile towels. Nasal oxygen may be helpful for the patient with subjective dyspnea.

**Anesthesia**

*A local anesthetic should be used generously*; careful anesthesia can render the procedure nearly painless (Fig. 9-10) (Figure Not Available) . Often 1% buffered lidocaine (Xylocaine) with epinephrine (1:100,000) is used. The maximum lidocaine dose of 5 mg/kg should not be exceeded. A skin wheal should be raised with a 25- to 27-ga short (½- to 5/8-inch) needle in the area of the skin incision. Many physicians advocate locating the skin wound one intercostal space below the one through which the tube will pass. The "tunneling" up and over the next rib that is required if this is to be done is believed to provide a better seal against air leaks both while the tube is in place and during and after its removal (Fig. 9-11) (Figure Not Available) . A larger (23-ga, 1½ inch) needle is used to infiltrate the SQ tissues, the muscle, the periosteum, and the parietal pleura in the areas through which the tube will pass. The sterile syringe should be kept readily available, as additional anesthesia is often required. A common error is inadequate local anesthesia. Intercostal nerve (rib) blocks above and below the incision and insertion rib spaces are also helpful. The anesthetic needle and syringe should be used to aspirate the pleural cavity in the area of insertion (Fig. 9-12) (Figure Not Available) . If air or fluid is not obtained and the patient's condition is stable, the diagnostic evaluation may need to be repeated or the insertion site changed. This simple and extremely useful technique to verify the location and the character of the intrapleural accumulation is often forgotten.

Appropriate IV analgesia and sedation should be administered to the hemodynamically stable patient. The careful use of conscious sedation during the procedure is strongly advocated when appropriate (see Chapter 35) . Supplemental oxygen should be considered, if only for patient reassurance. Once the tube is in place, anesthetic may be administered through the chest tube. [48] One approach for stable patients is to administer 10 mL of 0.5% bupivacaine through the chest tube while the patient is lying on the contralateral side. After 5 minutes without drainage of the thorax, standard gravity or vacuum drainage is reinitiated. Additional parenteral analgesic agents may be administered as the patient's condition warrants.

**Insertion**

One measures the length of the tube to be inserted by holding it near the chest wall. *It is a common mistake to advance chest tubes too far or not far enough.* Estimation of the desired placement depth will minimize tube adjustments and repeated radiographs. The distance from the incision site to the apex of the lung is estimated, and a clamp is placed on the tube at the point at which it should enter the chest wall. This position must be proximal enough from the last drainage hole to ensure that all holes are within the pleural space. The beveled (extrathoracic) end of the tube is often cut

**Figure 9-10** (Figure Not Available) Insertion of a chest tube can be relatively painless with proper infiltration of the skin and pleura with local anesthetic. The liberal use of *buffered* 1% lidocaine with epinephrine (maximum lidocaine dose, 5 mg/kg) is recommended. *(Redrawn from Hughes WT, Buescher*

*ES: Pediatric Procedures. 2nd ed. Philadelphia, WB Saunders, 1980, p 234.)*

**Figure 9-11** (Figure Not Available) The skin wound is made one intercostal space below the space through which the tube will pass *(A)*. By spreading tissue with scissors or a Kelly clamp *(B)*, blunt dissection is carried subcutaneously and into the pleural space *(C)*. A common error in technique is to attempt to insert a large chest tube through a skin incision that is too small. *(Redrawn from Hughes WT, Buescher ES: Pediatric Procedures. 2nd ed. Philadelphia, WB Saunders, 1980, p 237.)*

squarely at this time so that it better fits the commonly available connectors.

A generous 2- to 4-cm transverse skin incision is made through the skin and the SQ tissues directly over the rib located one interspace beneath the rib the tube will pass over. It is a common error to attempt to place a chest tube through an incision of inadequate size. This incision is extended by blunt dissection with a Kelly clamp to the fascia overlying the intercostal muscles. *A scalpel is needed only to make the skin incision.* Care must be taken to avoid the intercostal vessels and the nerve located on the inferior margin of each rib (Fig. 9-13) (Figure Not Available) . A large Kelly clamp is used to tunnel superiorly through the SQ tissues over the rib above, pushing forward with the closed points and then spreading and pulling back with the points spread (see Fig. 9-11) (Figure Not Available) . The closed points of the heavy clamp are then pushed with some force through the muscles, and the parietal pleura immediately overlying the rib and the pleural cavity is entered.

**Figure 9-12** (Figure Not Available) Use of the anesthetic needle to puncture the parietal pleura and establish the presence of blood or air in the pleural space. This procedure not only is diagnostic, but also may be a temporary therapeutic maneuver in a patient with tension pneumothorax. *(Redrawn from Richards V: Tube thoracostomy. J Fam Pract 6:631, 1978.)*

A twisting or drilling motion will enhance pleural penetration. A rush of air or fluid should occur at this point, and the patient may feel discomfort. If pleural fluid is under enough pressure, it may be expelled with considerable force and cover the unwary (and subsequently embarrassed) practitioner with blood or pus. Therefore, appropriate gown, glove, mask, and goggle precautions should be observed by the practitioner. The tips of the clamp, still within the pleural cavity, are spread widely and withdrawn (Fig. 9-14) (Figure Not Available) . The clinician must be certain to make an adequate opening in the pleura.

Next is one of the most important parts of the procedure: *the insertion of a gloved finger into the chest to verify that*

**Figure 9-13** (Figure Not Available) Location of the intercostal neurovascular bundle, running inferiorly and slightly medial to the rib. *(From Millikan JS, Moore EE, Steiner E: Complications of tube thoracostomy for acute trauma. Am J Surg 140:739, 1980.)*

**Figure 9-14** (Figure Not Available) One accomplishes blunt dissection by forcing the closed points of the clamp forward and then spreading the tips and pulling back with the points spread. A rush of air or fluid signifies penetration into the pleural space. *(From Bricker DL: Safe, effective tube thoracostomy. ER Reports 2:49, 1981.)*

*the pleura has been entered and that pleural adhesions are absent.* The finger should

sweep completely around the hole in the chest wall. Dense adhesions may mandate an alternative site for tube placement. The finger is left in the pleural space, and the tube is then grasped with the curved clamp, with the tube tip protruding from the jaws (Fig. 9-15) . (Another technique is to pass one jaw of the clamp inside the tube through a distal fenestration.) *With the finger in the chest cavity as a guide*, the tip is placed into the pleural space (Fig. 9-16) (Figure Not Available) . The finger is used to verify intrapleural

**Figure 9-15** The tube is grasped with the curved clamp, with the tube tip protruding from the jaws.

**Figure 9-16** (Figure Not Available) Using the finger as a guide to ensure entry into the pleural cavity, one places the tip of the tube into the pleural cavity. It is surprisingly easy to advance a chest tube subcutaneously, entirely missing the pleural space. *(From Millikan JS, Moore EE, Steiner E: Complications of tube thoracostomy for acute trauma. Am J Surg 140:739, 1980.)*

placement of the chest tube. The curve in the clamp is used to guide the tip superiorly and posteriorly. The clamp is released, and the tube is pushed superiorly, medially, and posteriorly until the marker clamp that was previously attached to measure the insertion distance touches the chest wall. All the holes in the chest tube must be within the pleural space. A common error, especially in obese patients, is to fail to advance the chest tube far enough into the pleural space, leaving the drainage holes either in the SQ tissues or outside the skin.

Alternatively, the tube may be advanced until pain is felt or resistance is met, and then pulled back 2 to 3 cm. It is surprisingly easy to misdirect the chest tube, even though the pleural space has been opened. Subcutaneous placement is a frequent complication, and dissection of tissue planes in the chest wall by an advancing chest tube can simulate entry into the chest cavity (Fig. 9-17) . Therefore, after placement, a finger is passed along the tube to verify entry into the thorax. Entry into the thoracic cavity also is suggested by the appearance of condensation on the inside of the tube coincident with respiratory movements, the audible movement of air through the tube during respiration, the free flow of blood or fluid, and the ability of the operator to rotate the tube freely after insertion. The tube is attached to the previously assembled water seal or suction setup by means of a sterile serrated connector before the clamp is released. Asking the patient to cough and thereafter observing bubbles in the water seal device is a good way to check system patency.

**Securing the Tube**

Before elaborate steps are taken to secure the tube, the clinician should be certain that the chest tube position is

**Figure 9-17** Subcutaneous (SQ) placement of a chest tube ( *arrows*) is surprisingly easy, as the tube can dissect through tissue planes with relative ease. The incorrect placement was not appreciated until the radiograph was taken. If the tube is directed posteriorly, an anteroposterior radiograph of the chest would erroneously "confirm" intrapleural placement despite the tube being SQ throughout its entire course.

verified on a chest film. There are as many ways of fastening a chest tube in place as there are physicians who place the device. More important than the individual technique chosen is the need to communicate the method used effectively to the person who will be caring for the patient, particularly the one who will be removing the tube. Some clinicians prefer wire, but I advocate sturdy silk sutures. Nylon sutures are difficult to tie tightly and are not recommended.

A reasonable approach is as follows: An 0 or 1-0 silk suture on a cutting needle is used. (Flimsy suture material, such as size 3-0 or finer, should be avoided.) The first suture is placed next to the tube to close the lateral margin of the skin incision and is tied firmly (Fig. 9-18) . The ends of this "stay" suture are left long and are then tied repeatedly around the chest tube and knotted securely to hold it in place. The sutures must be tied tightly enough to indent the chest tube slightly and thereby avoid slippage. A horizontal mattress suture is then placed around the tube approximately 1 cm across the incision on either side of the tube (i.e., cephalad and caudad to the tube) (Fig. 9-19) . This will be used to close the incision after the tube is removed. No knots are tied initially, but the skin should be pulled snugly together and held with a surgeon's knot (double-throw knot). The loose ends are repeatedly wound tightly around the chest tube as it enters the skin, with occasional repeated surgeon's knots tied tightly enough to indent the tube gently. The final knot is a bow, which should clearly identify the suture as *one not to be cut* when removing the chest tube.

An occlusive dressing of petrolatum-impregnated gauze should be placed where the tube enters the skin. Overlying this should be two or more gauze pads with a Y-shaped cut from the middle of one side to the center. These are oriented at 90° to each other (Fig. 9-20) . The shaved skin and the

**Figure 9-18** A "stay" suture is first placed next to the tube to close the skin incision. *A,* The knot is tied securely and the ends, which will subsequently be wrapped around the chest tube, are left long. *B,* The ends of the suture are wound twice about the tube tightly enough to indent the tube slightly and are tied securely.

tube may be coated with tincture of benzoin and wide (8- to 9-cm) cloth adhesive tape used to hold the tube more securely in place. Two strips of tape applied with an "elephant-ear" technique at 90° to each other provide an excellent method of securing the tube in place (Fig. 9-21) . The tape is torn so that one end is split into three pieces extending halfway to the center. The two outside pieces are placed on the skin on either side of the tube site, and the center piece is wrapped tightly around the tube. This is repeated with a second piece of tape, which is torn similarly and placed at 90° to the first. A third simple piece of tape may be used elsewhere on the chest to prevent the tube from being pulled loose accidentally. The connections are then securely taped.

**Confirmation of Tube Placement**

Repeat PA radiographs (and occasionally a lateral radiograph) must be taken to confirm tube placement and to document the degree of resolution. In a stable patient, it is often

better to obtain the postprocedure films with a less elaborate temporary dressing in place, in case tube repositioning is required. It is important to note that a simple pneumothorax should be completely reexpanded within a few minutes of continual suction. If the film taken after chest tube insertion shows that the lung is still collapsed, the clinician should consider three possibilities: (1) The tube may be in the wrong place, or the most proximal hole is outside the chest cavity. (This is the most easily corrected problem.) (2) A persistent air leak, usually from a large bronchus or the trachea, may be delaying expansion. (3) Plugging of a main bronchus with blood, mucus, or aspirated material may be delaying resolution of the pneumothorax.

## Drainage System

Physicians who place chest tubes infrequently or who do not participate in the ongoing care of patients who have undergone this procedure may have little familiarity with the physiology and mechanics of chest tube drainage systems. Life-threatening complications may arise from improper use of these devices, however, and the physician must be knowledgeable about the salient features of the system.

With the availability of modern closed drainage systems, the classic glass bottle collection system is rather cumbersome and largely antiquated. Nevertheless, the principles of the various drainage systems are discussed here. The simplest drainage device is a flutter (Heimlich) valve (commonly used only for pneumothorax) attached to the end of the chest tube itself (Fig. 9-22) . Such valves allow one-way flow of air from the chest but collapse to prevent air from passing back into the chest. Normal respiration, assisted by coughing, gradually removes the excess air from the pleural space, and the lung reexpands to fill the thoracic cage. The one-way Heimlich valve does not require a suction source when its purpose is the drainage of a pneumothorax. Therefore, it can be used in the treatment of outpatients. This valve is not generally used in conjunction with a closed suction-driven collection system to drain a hemothorax, because backflow is not a problem during continuous suction.

The simplest device to remove small amounts of either fluid or air is the underwater seal (single-bottle) device (Fig. 9-23) . The chest tube is connected to a second plastic tube,be extends 2 to 4 cm below the surface of the sterile water placed in the drainage bottle. The water provides a seal against the entrance of further air into the chest. The water also acts as a one-way valve. The intrathoracic pressure need only be greater than the depth of immersion of the tip of the drainage tube in the collection bottle to cause the intrathoracic air or fluid to exit into the bottle. This is easily accomplished with simple coughing.

For air to enter back into the pleural space through the chest tube, the patient must generate enough negative intrathoracic pressure to pull the water in the collection bottle up to the height of the chest. Normal inspiration is not forceful enough to do this if the bottle is kept on the floor. Normal fluctuation in the height of the fluid level in the long tube during respiration provides proof that free communication exists with the pleural space and that the tube is functioning normally. An absence of respiratory fluctuation or a decrease in the drainage implies blockage or, if the tube has been in for a sufficiently long period, full expansion of the lung and obliteration of the pleural space. The two

situations (blockage and full expansion) should be distinguishable by clinical and radiographic means.

On the other hand, an increase in respiratory fluctuation may imply an increased inspiratory effort owing to airway obstruction or atelectasis. If blocked, the tube and collecting tubing may be changed, "milked," or "stripped" to dislodge clots. *Milking* refers to forcing air or fluid back into the chest by pinching or clamping the tube distally and, with the other hand, compressing the tube and forcing the contents proximally. This can dislodge a blocking intrathoracic clot and can obviate the need for tube replacement when radiographic or clinical examination suggests incomplete expansion or drainage. *Stripping* involves proximal pinching or clamping and progressive distal compression followed by release of the proximal aspect. This allows the tube to spring open. The sudden increase in negative pressure may extract clots and fluid from a more proximal location. These procedures are more effective with soft latex tubing than with clear plastic tubes. However, routine use of milking and stripping should be avoided because of the high pressures generated during these procedures. [49]

Persistent bubbling in the tube during both expiration and inspiration implies an air leak, the most common source of which is the drainage system connections. These should be taped thoroughly and rechecked frequently. Another source of leakage may be failure to get the last opening in the chest tube inside the chest wall so that ambient air is sucked into the exposed hole. When the tube is appropriately placed into the thorax, air leaks through the chest wall are uncommon. One can best prevent such leakage at the skin incision site by initially tunneling up one interspace and by using effective suture technique and a petrolatum-impregnated gauze dressing.

The drainage bottle must always remain dependent, because gravity contributes a great deal to the proper drainage of the pleural space. Elevating the bottle above the chest can cause fluid to reenter the chest and can increase the probability of infection. The length of the tubing must be carefully controlled so that dependent loops of fluid do not form. Such loops of accumulated liquid must be displaced before more air or fluid can pass. The amount of positive intrapleural pressure required for air to pass through a dependent loop of fluid is greater than the vertical elevation of the fluid in the loop (Fig. 9-24) (Figure Not Available) . If the fluid loop becomes high enough (15 to 25 cm $H_2O$), egress of air may be blocked to a degree sufficient to cause a tension pneumothorax. [50] Similarly, as fluid accumulates in the water seal, the immersed tip of the tube must be raised so that it stays 2 to 4 cm below the water surface. Otherwise, a similarly progressive increase in intrathoracic pressure will be required to continue emptying the pleural space.

Sometimes a second trap, or collecting bottle, is placed proximal to the water seal (Fig. 9-25) . This has the advantage of keeping the level of fluid in the water seal bottle constant and allows for better measurement of collected drainage. A disadvantage is that air can enter the chest tube from the first bottle with accidental disconnection of any of the tubes or with a significant increase in negative intrapleural pressure. The dead space provided by the dry trap can produce an air lock effect and can lead to to-and-fro pressure changes with ventilation without effective drainage. More commonly, when a 2-bottle system is used, the second bottle is connected to suction. The amount of

suction in tube 1 is regulated not by the pressure reading on the wall suction valve, but by the depth of water in the second bottle above tube 3 and by the depth of tube 1 (Fig. 9-26) . When suction exceeds the depth of the water in bottle 2, air enters from the top of the third tube to prevent further increases. The internal diameter of the various tubes (especially tube 3) also contributes to the amount of suction that can be created.

The more complex the system, the more problems that can occur. Evaporation occurs quickly, and fluid levels must be maintained to keep pressures from steadily decreasing. Vigorous bubbling in the first bottle may cause foam to rise and to be suctioned into the second, either breaking the water seal or changing the pressure regulation. A few drops of a chemical agent (caprylic alcohol) can be used in bottle 1 to prevent this. As the first bottle fills with fluid, the effective suction decreases because of an increase in hydrostatic pressure at the bottom of tube 1. Although a 2-bottle system (water seal plus suction regulator) is adequate for nearly all emergency department applications, [51] a number of

**Figure 9-26** A 2-bottle system for applying regulated suction to the pleural space. The height of the column of water in bottle 2 regulates the amount of suction applied, independent of the pressure on the suction valve.

self-contained setups that avoid the bulky bottle system are commercially available.

Some add a third bottle for fluid collection (Fig. 9-27) (Figure Not Available) . Commercially available chest drainage systems that mimic the 3-bottle system are currently used by most hospitals. These units are plastic, lightweight, compact, easily transported, and easily assembled. Currently available models (Fig. 9-28) (Figure Not Available) collect up to 2500 mL of fluid, allow easy regulation of suction, and can be used for autotransfusion. Because glass bottles are cumbersome and time consuming to assemble, EDs are strongly urged to use the convenient, disposable chest drainage collection systems.

Occlusive clamping of chest tubes should be performed only with great trepidation and physician supervision, particularly in the first 24 hours after the tubes are placed. Clamping with a persistent intrathoracic air leak or fluid accumulation may lead rapidly to a tension pneumo- or hemothorax. Clamping is appropriate only to change the underwater seal bottle rapidly. Patients with chest tubes in place are best transported without clamping--on water seal only, with the bottle placed well below chest level.

**Role of Suction**

Suction is recommended at least initially in all patients with chest tubes placed for either pneumothorax or hemothorax. [35] [52] Ideally, a suction machine must have high flow (up to 20 L/min) and a regulated constant suction (0 to 60 cm $H_2O$). The continuous bubbling and the lack of respiratory variation with suction can mask the presence of an air leak. With extensive bleeding, excessive suction may actually increase the rate of blood loss, particularly when the bleeding is from a relatively low-pressure pulmonary vessel. Intermittent clamping or water seal use may be preferred. With a massive air

leak, excess suction may cause respiratory distress by removing inspired air before alveolar gas exchange can occur. Suction may be useful for rapid initial expansion and drainage. Because of the added complexity and complications, suction should be replaced by simple underwater seal drainage as long as expansion and drainage are satisfactory and no persistent air leak exists. When suction is applied, a pressure of 20 cm $H_2O$ is normally used.

**Prophylactic Antibiotics**

The routine use of prophylactic antibiotics in patients requiring tube thoracostomy is controversial. However, patients with associated injuries who are at high risk for infection (e.g., those who have undergone a nonsterile procedure or those with open fractures, a perforated viscus, or an esophageal injury) are always given antibiotics. Whether patients with isolated chest trauma or spontaneous pneumothorax require antibiotic prophylaxis has yet to be definitively determined. In one prospective, randomized, double-blind study of 75 patients with penetrating chest injuries, 2.6% of those treated with clindamycin developed empyema, compared with 16.2% of patients given placebo. [53] In another study of the use of cefoxitin in patients with either blunt or penetrating chest trauma, there was 1 infectious complication among 30 patients (3.3%); the chest infection rate in patients who did not receive antibiotics was 29%. [54] Another randomized double-blind study comparing cefazolin with placebo demonstrated a lower rate of sepsis and positive sputum cultures. [55]

**Figure 9-27** (Figure Not Available) A 3-bottle system with fluid trap bottle, water seal, and adjustable vent tube. *(From Miller KS, Sahn SA: Chest tubes: Indications, technique, management and complications. Chest 91:258, 1987.)*

However, in a series of 80 patients with penetrating chest injuries, 0 of 40 patients treated with doxycycline and only 1 of 40 who were not given antibiotics demonstrated infections. [56] A meta-analysis concluded that there was a statistically significant effect favoring antibiotic prophylaxis

**Figure 9-28** (Figure Not Available) Diagram of a modern commercially available chest drainage system. Included is a 2500-mL fluid collection chamber, a suction control device, an air leak indicator, and a fluid sampling port. A separate attachment is available for autotransfusion. *(Courtesy of Davol, Inc, a division of CR Bard, Cranston, RI.)*

after tube thoracostomy. [57] Probably more important than the use of antibiotic prophylaxis is ensuring, when time permits, that strict aseptic technique is followed when the tube is initially placed.

If antibiotics are used, the first dose is given as soon as possible, preferably before the tube is inserted, and continued until the tube is removed. The chosen antibiotic should cover *Staphylococcus aureus,* as this is the most common organism causing empyema, but gram-negative bacteria coverage is also desirable. A first- or second-generation cephalosporin is probably a good choice, because these agents have a broad spectrum of activity and easily pass into the pleural space. Nichols and colleagues [58] noted a reduction in infectious complications from 10.7 to 1.6% in patients requiring chest tube insertion predominantly for penetrating chest trauma with the prophylactic use of the

once-daily cephalosporin cefonicid. Others have recommended clindamycin, cefoxitin, cefazolin, cefamandole, or doxycycline. It appears reasonable to administer prophylactic antibiotics when the chest tube is placed in an emergency situation and the potential for infection is greater because of possible contamination, especially in cases of penetrating chest trauma. Stable patients who receive a chest tube in controlled and elective situations (especially in the setting of a spontaneous or iatrogenic pneumothorax) probably do not require antibiotic prophylaxis.

## Tube Removal

Recommendations vary, but chest tubes should generally be removed when there has been no drainage of fluid or air for a minimum of 24 hours, when respiratory variations in the water seal have ceased, and when high-quality radiographs reveal satisfactory resolution of the initial pathology. Because of pleural irritation, small amounts of serous fluid (<200 mL/day) may continue to drain without contraindicating removal.

To remove a chest tube, the patient should be placed in a semi-upright position, and the dressings should be removed. Sedation or restraints may be helpful. The area should be sterilized and draped, and sterile technique should be followed. The only instruments required are sterile basins, heavy scissors to cut the suture, dressing materials, and instruments to tie the previously placed pursestring suture or to place a new one. Facilities and equipment should be

available to reinsert a new chest tube promptly if it should become necessary. The suture holding the tube to the skin should be removed from the tube, and the pursestring suture that was previously placed should be loosened and readied for tying. A second (gloved) assistant is helpful. The tube should be clamped, and the connecting tubing should be removed. A petrolatum- or antibiotic-impregnated gauze dressing should be prepared. The patient should inhale fully and perform a mild Valsalva maneuver. The tube is removed in one swift motion while the patient holds the breath. Two fingers hold the skin edges shut, and the pursestring suture is tied. The occlusive dressing is placed and taped securely. A period of observation (minimum, 2 to 6 hours) is recommended if the patient is to be sent home, with a chest film obtained at the end of that time. Any increase in symptoms requires prompt reevaluation. After 48 hours, the dressing may be removed and the wound managed as any sutured skin wound would be.

## Other Techniques

### Trocar Insertion

Percutaneous introduction of a chest tube with a trocar was a common practice in the past. This technique has been largely abandoned because of the risk of major complications, such as damage to lung and solid organs. [59] Most authorities currently condemn the use of a trocar for the placement of a large-bore chest tube. [52] [60] The above described blunt technique is the favored method. Although use of the trocar to forcibly enter the chest is condemned, some clinicians still use it to carefully guide (not force) the tube through an existing thoracic wall opening made by the blunt dissection

technique.

Whereas the use of trocars for initial entry of the chest during insertion of large-bore tubes is fraught with danger, new minicatheter systems that combine the trocar approach with a guide wire dilator system appear promising for management of pneumothoraces. These catheter systems are used in the fashion described below.

**Minicatheter Insertion**

**Indications and contraindications.**

A less invasive alternative to traditional tube thoracostomy for patients with a simple uncomplicated pneumothorax is treatment with minicatheter placement for pneumothorax aspiration. Advantages of this technique include the ease of catheter insertion, decreased patient discomfort, less scarring, increased patient mobility, and decreased cost. In addition, after successful reexpansion of the lung, selected patients may be treated as outpatients. Patients with iatrogenic pneumothorax (e.g., that which occurs after central line placement, lung biopsy, or bronchoscopy), those who have IV drug use-induced pneumothoraces, victims of minor chest trauma, and patients with spontaneous pneumothoraces are potential candidates for catheter aspiration of the pneumothorax. Patients with multiple rib fractures, tension pneumothorax, hemothorax, hemodynamic instability, or serious associated injuries requiring surgery should receive conventional tube thoracostomy. Patients with underlying lung pathology, such as pneumonia, congestive heart failure, asthma, or emphysema, are generally not candidates for minicatheter use.

Minicatheter aspiration is particularly effective for patients with a needle-induced (i.e., iatrogenic or IV drug "pocket shooters") pneumothorax. In these patients, success rates ranging from 75 to 93% have been reported. [41] [61] [62] [63] This technique is somewhat less effective in patients with a spontaneous pneumothorax [63] [64] because of the continued air leak from ruptured blebs. A recent randomized study compared aspiration with traditional tube thoracostomy in patients with a spontaneous pneumothorax. [65] Aspiration was effective 80% of the time, and the patients had decreased pain and a shorter hospital stay. The authors concluded that aspiration is a simple and safe procedure and is the treatment of choice in patients with normal lungs and a spontaneous pneumothorax, irrespective of size.

**Equipment.**

A No. 14 through-the-needle Intracath, a 14-ga IV catheter, or an 8.5 Fr Arrow trauma catheter inserted using the Seldinger (guide wire) technique may suffice for simple aspiration. However, these catheters are designed for fluid administration and have only a single distal hole that can easily become obstructed or adhere to the pleura when suction is applied. Therefore, it is suggested that a catheter designed specifically for the purpose be used, particularly if it is to be left in place for a prolonged period of time. The commercially available catheters have multiple distal side ports and are made of a soft,

flexible, thrombosis-resistant, radiopaque material such as polyurethane.

Available commercial systems include the 8 Fr guide wire system by Cook (CCASP-FORD-110485, Cook Catheter, Bloomington, Ind), and 8 Fr over-the-needle systems by Arrow (AK-01500, Arrow International, Reading, Pa) and Argyle (8888-567032, Argyle, Sherwood Medical, St Louis). A 14 Fr introducer (Introstat, Hart Medical, Clearwater, Fla) and 12 Fr polyvinyl chloride chest tube system (Argyle, Sherwood Medical) has been used as a compromise between the smaller catheters and a full-sized chest tube. [32]

**Procedure.**

Placement of a catheter into the pleural space is relatively simple. The patient is placed in a semi-upright position and sterile preparation is performed with povidone-iodine solution at either the fourth or fifth intercostal space at the anterior axillary line or the second or third intercostal space at the midclavicular line; either site is acceptable. Anesthesia with generous local infiltration of lidocaine, as described for standard tube thoracostomy, is administered before insertion of the catheter. The exact technique depends on the type of equipment to be used.

**Guide wire (Seldinger) technique.**

A thin-walled 16-ga needle is advanced cephalad over the top of the rib at a 60° angle (Fig. 9-29) . When the pleural space is entered (identified by the aspiration of air into the syringe), a guide wire is inserted and the needle removed. A small incision is made in the skin with a No. 11 blade, and the minicatheter with an introducer is threaded over the guide wire into the pleural space. The wire is then removed, and a three-way stopcock is attached along with a 60-mL syringe. Air is then aspirated until resistance is felt. Often a surprising amount of air can be removed. Occasionally air cannot be aspirated because the catheter is kinked or blocked with soft tissue. If no air is obtained, the clinician should place the patient in the full upright position, have the patient cough or take a deep breath, or withdraw the catheter slightly. This often results in successful aspiration. When no additional air can be aspirated, a chest radiograph is obtained to document full expansion of the lung. If residual pneumothorax is present, an attempt at further aspiration is made.

**Over-the-needle technique.**

First, a small incision is made in the skin with a No. 11 blade (Fig. 9-30) . Then the needle tip, with constant suction on the attached syringe, is advanced into the pleural space. Proper placement is confirmed by the free flow of air into the syringe. The catheter is then advanced over the rib into the pleural space, ensuring that all of the side ports are within the pleural space. The catheter is then connected to a three-way stopcock, and air is aspirated with a syringe as described above.

A protocol using catheter aspiration as the first step in treating simple pneumothoraces (Fig. 9-31) (Figure Not Available) has been described. [66] Patients with successful

aspiration are observed in the emergency department for 4 hours, and if a repeat radiograph shows no reaccumulation of air, the catheter is removed. After 2 more hours of observation, another chest radiograph is obtained, and the patient is released if there is no recurrent pneumothorax. A Heimlich valve is attached to the catheter if a patient has more than a 10% residual apical pneumothorax. The valve is placed on suction only if this does not result in full expansion. Patients with continued residual pneumothorax then receive a conventional tube thoracostomy.

Using this protocol, Vallee and coworkers [66] reported a 59% success rate with simple aspiration in 35 patients with a total of 37 pneumothoraces. An additional 27% responded to the use of a Heimlich valve with or without suction. Only five patients (14%) required a tube thoracostomy; two of these were patients who initially responded to catheter aspiration but returned with a recurrent pneumothorax on follow-up visits. This protocol appears to be useful in patients with selected simple pneumothoraces, but further study is warranted. In particular, outpatient therapy consisting of a small catheter with an attached Heimlich valve may be the best strategy in selected patients with a spontaneous pneumothorax. [67]

### Simple Thoracostomy

When an emergent condition exists with hemodynamic decompensation, the clinician may consider simple thoracostomy without immediate chest tube placement. Deakin and colleagues reported using this technique in out-of-hospital patients needing decompression of a pneumothorax or hemothorax while under positive-pressure ventilation. [68] They used the standard approach for chest tube placement (i.e., incision, curved forceps penetration of the pleura, and digital exploration of the thorax to rule out adhesions and confirm intrathoracic location). Incisions were covered with plain sterile gauze. A chest tube was placed at a later time if radiographs revealed incomplete expansion of the pneumothorax or incomplete drainage of a hemothorax. They observed no obstruction of the incision by a thrombus. *It is imperative that patients treated in this manner receive positive-pressure ventilation.* Otherwise, the thoracostomy incision may give rise to an increasing pneumothorax as negative inspiratory pleural pressures develop.

## PNEUMOTHORAX IN PEDIATRIC PATIENTS

Pneumothorax occurs more commonly during the neonatal period than at any other time of life. When chest radiographs are taken of all newborns in large series, the incidence of pneumothorax is about 1 to 2%. The incidence does not seem to change when normal (term) vaginally delivered newborns are compared with premature newborns or those delivered by cesarean section. The incidence of symptomatic pneumothorax in newborn infants, however, is consistently only 0.05 to 0.07%. Among newborns with pneumothorax, several studies report twice as many males as females. Some studies report more instances of right-sided collapse. Ten to 20% of cases are bilateral. [69]

It should be noted that lobar emphysema in a newborn may cause severe respiratory symptoms shortly after birth (Fig. 9-32) (Figure Not Available) . The physical examination may detect decreased breath sounds in a hemithorax and even evidence of

mediastinal shift. Lobar emphysema often looks like a tension pneumothorax radiographically, and the unwary physician may rush to insert a chest tube. The treatment in this case is surgical removal of the diseased lobe, and a chest tube may worsen the clinical condition.

## Pathophysiology

There seem to be two groups of newborns who develop pneumothorax. The first are term or post-term neonates with a history of fetal distress; difficult delivery; need for resuscitation; or aspiration of meconium, amniotic fluid, or blood. These infants tend to become symptomatic within the first 2 hours of life and generally fare quite well. The mechanism in this group is believed to be an excess intra-alveolar pressure generated at birth. With the first breath, the transpulmonary pressure increases from 40 to as much as 100 cm $H_2O$. Compression of the chest during vaginal delivery places the diaphragm and the muscles of respiration at a marked mechanical advantage. With mechanical obstruction of some alveoli or bronchioles, as can occur with aspiration, the intense transpulmonary pressure is transmitted to the normally aerated alveoli, which can overdistend and rupture. Mechanical ventilation, end-expiratory pressure, and resuscitative efforts can also precipitate alveolar rupture.

The second group of newborns with pneumothorax are those who have underlying pulmonary disease (most notably, respiratory distress syndrome) or congenital abnormalities. These infants commonly develop a pneumothorax in the second day of life, often while being treated with positive airway pressures. The prognosis in these cases is much worse. [69] [70]

## Presentation and Diagnosis

The physical examination of the newborn with pneumothorax can yield findings ranging from no abnormalities whatsoever to complete cardiovascular collapse. Grunting respirations and tachypnea (to a respiratory rate as high as 120 breaths/min) are often seen. Retractions or nasal flaring can be seen, and crepitus in the neck may be present. Cyanosis may be present or may occur only with crying or feeding. Irritability, restlessness, apneic periods, bradycardia, or tachycardia may be the only manifestation. Distention and tympany of the affected side may be found. A decrease in breath sounds is difficult to appreciate in the newborn. With tension pneumothorax, the cardiac impulse and the trachea may be shifted away from the affected side.

The definitive diagnosis is made with high-quality radiographs taken in both the AP and the horizontal beam (cross-table) lateral projections. Small pneumothoraces may be seen only on the lateral view, as the air collects at the top of the thoracic cavity. Bilateral tension pneumothorax can appear as microcardia, without any mediastinal shift. Further radiologic studies may be needed to differentiate this condition from lung cysts, lobar emphysema, and skin folds.

Transillumination of the chest with a high-intensity fiberoptic light source has been used with great success to detect and follow pneumothorax and pneumomediastinum in newborns. [71] It has been noted that abrupt changes in transthoracic impedance in

infants on respiratory monitors have been related to the appearance of pneumothorax. [72] Such changes should initiate prompt reevaluation of the patient's respiratory status.

**Treatment and Indications for Thoracostomy**

In general, tube thoracostomy is the treatment of choice once a symptomatic pneumothorax is detected in infants. When signs of tension pneumothorax are present, immediate aspiration with a plastic catheter over-the-needle device is recommended (see Chapter 8) . Small pneumothoraces (<20% of the hemithorax) in relatively asymptomatic infants (i.e., those who are without other problems and who do not require positive airway pressures) can be monitored merely with close observation.

Repeated films or transillumination and frequent monitoring of vital signs and arterial blood gases are indicated. Breathing 100% oxygen is believed to hasten reabsorption by as much as six-fold. [70] However, the risks of retrolental fibroplasia and pulmonary oxygen toxicity must be carefully assessed when considering oxygen therapy.

When evacuation of the pleural space is elected, needle aspiration using a 50-mL syringe, an 18-ga catheter over-theneedle device, and a three-way stopcock may be attempted once. This may suffice in patients without a continued air leak, although the risk of lung puncture is considerable.

**Thoracostomy Technique**

The technique of tube thoracostomy in pediatric patients varies little from that already described. Small, commercially available thoracostomy tubes can be used. No. 8 to 10 Fr catheters are used in premature infants, and No. 10 to 12 Fr catheters are used in larger newborns. Blunt dissection minimizes the complications of lung puncture, hemorrhage, and traumatic fistula formation, which are seen more often with trocar insertion.

Various tube locations have been proposed. One study compared lateral placement (fourth to fifth intercostal space, anterior axillary line) and superior placement (first to third intercostal space, midclavicular line) of 149 chest tubes for their effectiveness in evacuating pneumothorax. The most important factor was the eventual location of the tube rather than the site of insertion. Anterior tubes were effective 96% of the time, whereas only 42% of the tubes directed posteriorly functioned satisfactorily. [73]

Placement in the third intercostal space, midaxillary line, with the tip directed under the sternum appears to be a good compromise. [72] Care must be taken to avoid the nipple, which can be difficult to identify in the premature infant. Water seal with 10 to 20 cm $H_2O$ for suction is usually recommended until reexpansion occurs and the absence of continued air leakage is verified.

Smaller collecting bottles are recommended to measure drainage more accurately. A miniature water seal apparatus using a 50-mL multiple-use saline bottle, standard IV tubing, and one long and one short needle has been described (Fig. 9-33) (Figure Not

Available) .

## COMPLICATIONS

As with any surgical procedure, complications can and will occur (Table 9-6) . Local infection at the site of insertion is common and may reflect the often hurried performance of this procedure in the emergency setting. Osteomyelitis has been reported in settings in which tubes have been kept in place for a long time. Empyema is uncommon, because tube thoracostomy remains a useful treatment for this problem. Documented cases of empyema developed in only 1.2 to 3.0% of patients after tube thoracostomy. [75] [76] Empyema tends to occur in patients with a loculated effusion and in those who are inadequately drained. Pneumonia and atelectasis are attributed to a decrease in coughing and failure to clear secretions because of pain. As for any postoperative patient, early ambulation and vigorous pulmonary toilet are indicated. The easiest way to provide analgesia for patients with postprocedural pain is to administer parenteral analgesia, but intrapleural bupivacaine after chest trauma or spontaneous pneumothorax is effective in reducing pain and may be useful for reducing pain-related complications. [48] [77] Pain relief extending to several hours has been observed if 20 mL of 0.25% bupivacaine is inserted into the pleural cavity via the chest tube, with pleural drainage interrupted for 5 to 10 minutes and the patient lying supine. [48] Up to 40 mL of 0.25% bupivacaine administered intrapleurally will result in blood levels well below the toxic range. [78]

A local hematoma may occasionally develop at the incision site but may be prevented by careful dissection. Intercostal arteries or veins may be lacerated at the time of tube insertion. These lacerations can be minimized if sharp dissection is carried only to the fascia and the tube is carefully placed just above the rib. The tube may adequately tamponade such bleeding, but if tamponade is insufficient, the incision may have to be extended to expose or ligate the bleeding vessel. If a lacerated intercostal artery does not stop bleeding, the clinician may attempt to insert a Foley catheter into the incision, inflate the balloon, and withdraw the catheter to tamponade the vessel. Anterior chest wall tube placement carried to the midline may result in internal mammary artery laceration. This is notoriously difficult to control and may require thoracotomy. Great vessel injury is uncommon.

A 1% incidence of technical complications (visceral perforation) was documented in a series of 447 patients who

| TABLE 9-6 -- Complications of Tube Thoracostomy |
| --- |
| Infection |

| |
|---|
| Pneumonia |
| Empyema |
| Local incision infection |
| Osteomyelitis |
| Necrotizing fasciitis |
| Bleeding |
| Local incision hematoma |
| Intercostal artery or vein laceration |
| Internal mammary artery laceration (with midclavicular line placement) |
| Pulmonary vein or artery injury |
| Great vessel injury (rarely) |
| Laceration or puncture of nerves or solid organs |

| |
|---|
| Lung |
| Liver |
| Spleen |
| Diaphragm |
| Stomach |
| Colon |
| Long thoracic nerve |
| Intercostal nerve (may result in intercostal neuritis /neuralgia) |
| Intercostal muscles (intercostal myalgia) |
| Mechanical problems |
| Chest tube dislodgment from chest wall |
| Incorrect tube position |

| |
|---|
| Subcutaneous placement |
| Intra-abdominal placement |
| Air leaks |
| Leaks from tubing or drainage bottles |
| Last tube fenestration not entirely within pleural space |
| Leaks from skin site |
| Flow of drainage bottle contents into chest from inadvertent elevation of drainage bottles |
| Blocked drainage |
| Kinked chest tube or drainage tubes |
| Clots |
| Miscellaneous |
| Allergic reactions to surgical preparation or anesthesia |

| |
|---|
| Pulmonary atelectasis |
| Persistent pneumothorax |
| Retained hemothorax |
| Clotted hemothorax or fibrothorax |
| Subcutaneous or mediastinal emphysema |
| Reexpansion pulmonary edema |
| Reexpansion hypotension |
| Recurrence of pneumothorax after chest tube removal |

underwent tube thoracostomy for trauma. [59] Another case series reported a 1.8% rate of errors related to placement of the chest tube in 164 patients in their series. [75] Damage to the lung parenchyma or the intrapulmonary vessels can occur readily. This is more common with the trocar insertion technique, which currently has been largely abandoned in favor of blunt dissection. The use of digital exploration of the pleura to exclude or free adhesions is mandatory before tube placement. Careful attention to anatomy, with recognition of exactly how high the diaphragm may rise (especially if the abdomen is filled with blood), can minimize injuries to the diaphragm or the intra-abdominal organs.

Reexpansion pulmonary edema is a potentially fatal complication of thoracentesis or tube thoracostomy. [79] Pulmonary edema was reported in 14% of patients with spontaneous pneumothorax in one series, [80] although the prevalence reported in other series is much lower. [28] Pulmonary edema, usually ipsilateral, can appear as soon as 1 to 2 hours after lung reexpansion. Edema commonly occurs when collapse has been

present for longer than 72 hours and when 1000 mL or more of pleural fluid is removed. Suction seems to worsen the edema. The exact pathogenesis of reexpansion pulmonary edema has yet to be adequately determined but is likely multifactorial. Increased pulmonary vascular permeability appears to be a major factor and is probably related to hypoxic injury to the capillary beds and alveolar membranes. Increased pulmonary capillary pressure and blood flow, decreased surfactant levels, and mechanical damage to the lung also play a role. Rapid removal of air or fluid may result in an abrupt return of circulation and transudation of protein and fluid into the alveolar space. Occasionally an associated shock-like state occurs, with resulting hypotension and evidence of decreased organ perfusion. [81] Treatment includes supportive care, oxygenation, and positive-pressure ventilation when indicated. Patients with hypotension are given volume replacement and inotropic agents as needed. Invasive monitoring is frequently required. The best course is to prevent this complication with slow, staged removal of large effusions or collections of air (especially if they have been present for several days) and avoidance of suction, if possible.

Mechanical problems commonly result in air leaks with failed reexpansion or inadequate drainage. Tension pneumothorax can occur if a blockage in the drainage system at any point is associated with an air leak. Failure of reexpansion or incomplete reexpansion of a pneumothorax may be due to a mechanical air leak, but it may also indicate a bronchopleural fistula, a continued parenchymal lung leak, or a bronchial injury. Retained hemothorax may result from clotting or poor tube function. The largest possible tube should be used for drainage of a hemothorax. Reinsertion or placement of a second tube may be indicated if the first tube is not functioning properly. Often, an angled tube in the posterior diaphragmatic sulcus will promote drainage of a dependent fluid collection. Clotted hemothorax or fibrothorax is an indication for elective decortication and pleurodesis. Subcutaneous emphysema can occur if the chest tube is partially extruded or plugged.

If a chest tube is not functioning properly or has not reversed the pathologic condition as expected, the clinician must carefully reevaluate the position of the chest tube. In an emergency situation, the tube may have been placed subcutaneously, and the incorrect placement may not be obvious to the physician. If the tube dissects in a fascial plane posteriorly along a rib, even the postinsertion film may appear to confirm proper placement. Such a condition may be lethal. As a general rule, *if a chest tube is not functioning properly and the patient is deteriorating, the tube should be removed and inserted again, or another tube should be inserted.*

# Chapter 10 - Carotid Sinus Massage and Cardioversion

**William Burdick**

## Carotid Sinus Massage

The effects of pressure on the carotid sinus have been known for centuries. They were first described in the medical literature in 1799 when Parry wrote a treatise about the "symptoms and causes of syncope anginosa, commonly called angina pectoris," [1] in which he noted that pressure on the bifurcation of the carotid artery produced dizziness and slowing of the heart. The term carotid is derived from the Greek *karos*, meaning heavy sleep.

Carotid sinus massage (CSM) is a technique involving digital pressure on the richly innervated carotid sinus that takes advantage of the externally accessible position of this baroreceptor for diagnostic and therapeutic purposes. Its main diagnostic utility is now in the differential diagnosis of syncope, particularly in the elderly; in the assessment of tachydysrhythmias and rate-related bundle branch block; and as a clue to latent digoxin toxicity. Its main therapeutic application is for termination of supraventricular tachycardias (SVTs) due to paroxysmal atrial tachycardia (PAT).

The use of CSM has been eclipsed in recent years by the use of adenosine, an ultrashort-acting vagal stimulating nucleotide. The diagnostic and therapeutic effects of adenosine on tachydysrhythmias are similar to those elicited by CSM, and its safety is derived from its short duration of action--usually about 10 seconds. Because adenosine may not always be readily available, and because adenosine cannot be used to assess the sensitivity of the carotid sinus, CSM remains a useful tool.

A number of the other therapeutic uses of CSM have also been made nearly obsolete by current medical therapy. CSM was noted in the 1920s to relieve acute pulmonary edema in many patients with hypertensive and coronary artery disease. Lown and Levine describe the dramatic effect: "Relief is immediate and coincides with the onset of bradycardia. In the majority it is associated with a drop in blood pressure. The patient is promptly able to lie flat. Fear, dyspnea, and chest oppression disappear..." [2] CSM also can relieve anginal pain; this technique may be useful when the diagnosis of angina is uncertain. However, the advantage of this technique over the use of nitroglycerin is unknown. Hence, although CSM is no longer the first approach to either of these common clinical problems, it may remain a therapeutic or diagnostic tool in some difficult cases or when modern pharmacologic agents are unavailable.

### ANATOMY AND PHYSIOLOGY

The bifurcation of the common carotid artery possesses an abundant supply of sensory nerve endings located within the adventitia of the vessel wall (Fig. 10-1) (Figure Not Available) . These nerves have a characteristic spiral configuration, continually

intertwining along their course and eventually uniting to form the carotid sinus nerve. The afferent impulses travel to the vasomotor center of the medulla. Efferent impulses travel along the vagus nerve to the sinus node and the atrioventricular (AV) node, as well as along the sympathetic chain to the heart and the peripheral vasculature.

The afferent nerve endings in the carotid sinus are sensitive to mean arterial pressure and to the rate of change of pressure, with pulsatile stimuli found to be more effective than sustained pressure in evoking a response. Elevated blood pressure stretches the baroreceptors, leading to increased firing of the afferent nerve endings. The carotid sinus is even more sensitive to low blood pressure, which causes a drop in afferent firing.

The parasympathetic and the sympathetic nervous systems both play a role in the carotid sinus reflex. Increased firing of the carotid sinus results in reflex stimulation of vagal activity as well as in reflex inhibition of sympathetic output. The parasympathetic effect is almost immediate; it occurs within the first second and causes a drop in heart rate. The sympathetic effect, which causes a drop in blood pressure through vasodilatation, becomes manifest only after several seconds, and it may not take full effect until a minute has elapsed. [3] These are independent phenomena--the fall in heart rate is blocked by the administration of atropine, whereas epinephrine blocks the reduction in blood pressure. A cerebral effect, characterized by a loss of consciousness, was once thought to be due to stimulation of the carotid sinus. However, it is seen only when sufficient pressure is exerted to occlude the more distal temporal artery pulsation and when contralateral carotid disease is present. This cerebral effect is therefore believed to be a result of decreased bilateral cortical perfusion.

The parasympathetic branch of the carotid sinus reflex supplies the sinus node and the AV node. The sinoatrial (SA)

**Figure 10-1** (Figure Not Available) Anatomy of the carotid sinus reflex. Carotid receptors send impulses to the medulla by way of the sinus nerve of Hering and cranial nerve IX. Efferent nerves are shown on the right. *(Adapted from Scher AM: Control of arterial blood pressure. In Ruch TC, Patton HD: Physiology and Biophysics. 20th ed. Vol 2. Philadelphia, WB Saunders, 1974.)*

pacemaker is more likely to be affected than is the AV node, except when digitalis has been administered. In order of decreasing frequency, the changes seen clinically with CSM include (1) SA slowing, occurring in approximately 75% of cases and leading to sinus arrest approximately 3% of the time; (2) atrial conduction defects, manifested by an increase in width of the P wave on the electrocardiogram; (3) prolongation of the PR interval and higher degrees of AV block, seen in approximately 10% of cases; (4) nodal escape rhythms; (5) complete asystole, defined as sinus arrest without ventricular escape lasting greater than 3 seconds, occurring in 4% of cases; and (6) premature ventricular contractions.

## INDICATIONS AND CONTRAINDICATIONS

CSM may be a useful aid to the diagnosis of syncope in the elderly. Some 14 to 45% of elderly patients referred for syncope are thought to have carotid sinus syndrome, [4] [5]

defined as an asystolic pause greater than 3 seconds or a reduction of systolic blood pressure greater than 50 mm Hg in response to CSM. It shares many characteristics with sick sinus syndrome, suggesting that both are manifestations of the same disease. The carotid sinus syndrome causes cerebral hypoperfusion leading to dizziness and syncope. Analysis of patients with the syndrome indicates that it results from baroreflex-mediated bradycardia in 29% of patients, hypotension in 37%, or both in 34%. [6] Syncope, near-syncope, or a fall of unclear etiology in the elderly are therefore important indications for diagnostic CSM.

Another diagnostic indication for CSM is possible digoxin toxicity. Toxicity from digoxin depends more on the response of the host than on the actual digoxin level. In cases of suspected digoxin toxicity when the digoxin level is in the "normal range," or before the level is available, CSM may be a useful diagnostic adjunct. Significant inhibition of AV node conduction associated with ventricular ectopy, and especially ventricular bigeminy, should lead to suspicion of digoxin toxicity. [2]

The clinician can take advantage, however, of the similarity of the vagal effects of digoxin and CSM. Before starting digoxin administration in a patient, the practitioner can gauge the cardioinhibitory effect that will be achieved with the drug by first performing CSM. Significant slowing or block with CSM suggests a similar sensitivity to digoxin, and a smaller loading dose should be considered.

CSM also is indicated in settings in which slowing conduction in the SA or AV node could provide useful information. These settings include patients with wide complex tachycardia in whom CSM aids in the distinction between SVT and ventricular tachycardia (VT), elucidates narrow complex tachycardia in which the P waves are not visible, or aids in detection of suspected rate-related bundle branch block or suspected pacemaker malfunction. After CSM, a wide complex SVT may be converted to normal sinus rhythm, P waves may be revealed after increased AV node inhibition, or ventricular complexes may narrow as the ventricular rate slows. Because CSM slows atrial and not ventricular activity, AV dissociation may be more easily seen, indicating VT (Fig. 10-2) (Figure Not Available) . In rapid atrial fibrillation, or atrial flutter with 2:1 block, either P waves or irregular ventricular activity with absent P waves may be revealed. Sinus tachycardia may also be more apparent once P waves are unmasked by slowing the SA node (Figs. 10-3 (Figure Not Available) to 10-9) (Figure Not Available) . Adenosine may be used for the same diagnostic purpose in these situations as well.

CSM is *contraindicated* in patients likely to suffer neurologic or cardiovascular complications from the procedure. Patients with a carotid bruit should not have CSM because of the risk of carotid embolization or occlusion. A recent cerebral infarction is another contraindication, because even marginal reduction of cerebral blood flow may produce further infarction.

The presence of diffuse, advanced coronary atherosclerosis is associated with increased sensitivity of the carotid sinus reflex. This hypersensitivity is further augmented during an anginal attack or an acute myocardial infarction. Brown and coworkers found that the degree of carotid sinus hypersensitivity was directly

proportional to the severity of coronary artery disease documented by cardiac catheterization. [7] Patients with acute myocardial ischemia or with recent myocardial infarction are already at higher risk of VT or ventricular fibrillation (VF), and a prolonged asystole may further predispose them to these dysrhythmias. CSM should therefore also be avoided in these patients.

Both digoxin and CSM act through a vagal mechanism to inhibit the AV node. Patients on digoxin may experience a greater inhibition of the AV node with longer AV block as a result. Patients with apparent manifestations of digoxin toxicity or known digoxin toxicity should not have CSM, as AV inhibition may be profound.

## EQUIPMENT AND SETUP

Generally, as a precaution against hypotension, an intravenous (IV) line with normal saline should be started before attempting CSM, and the patient should be placed on a cardiac monitor. Atropine and lidocaine, as well as a transvenous or transcutaneous pacemaker and defibrillator, should be readily available at the bedside in case a life-threatening dysrhythmia develops. The clinician should first auscultate for carotid bruits on both sides of the neck before attempting CSM--the presence of a bruit is a contraindication to massage.

The patient should be in the supine or slight reverse Trendelenburg position if it can be tolerated. Occasionally PAT will convert merely by lowering the back of the bed, presumably because the supine position results in a stretching of the carotid bulb, giving maximum baroreceptor sensitivity. The supine position may also prevent syncope in the event of a significant drop in heart rate or blood pressure.

Berk and colleagues have demonstrated in healthy volunteers that cold-water face immersion and the Valsalva maneuver can produce a greater vagal response than CSM. [8] Mehta and colleagues also found that the Valsalva maneuver was more effective than CSM for conversion of induced SVT. [9] The pneumatic antishock garment has also been used to similarly increase vagal tone by stretching the carotid bulb (see Chapter 30) . [10]

## TECHNIQUE

The clinician should begin CSM on the patient's right carotid bulb as some investigators have found a greater cardioinhibitory effect on this side, [8] [9] although no difference was found in one study. [11] Simultaneous bilateral CSM is absolutely contraindicated, because cerebral circulation may be severely compromised.

Keeping the patient relaxed is helpful for two reasons: a tense platysma muscle makes palpation of the carotid sinus difficult, and an anxious patient will be less sensitive to CSM as a result of heightened sympathetic tone.

With the head tilted backward and slightly to the opposite side, palpate the carotid artery just below the angle of the mandible at the upper level of the thyroid cartilage and

anterior to the sternocleidomastoid muscle. Once the pulsation is identified, use the tips of the fingers to administer CSM for 5 seconds in a posteromedial direction, aiming toward the vertebral column. Although earlier practitioners used longer duration of massage, a shorter period of massage minimizes the risk of complications and is adequate for diagnostic purposes in the majority of patients. [12] Pressure on the carotid sinus may be steady or undulating in intensity; the force, however, must not occlude the carotid artery. The temporal artery may be simultaneously palpated to ensure that the carotid remains patent throughout the procedure.

If unsuccessful, CSM may be repeated after 1 minute. If the procedure is still unsuccessful, the opposite carotid sinus may be massaged in a similar fashion. Simultaneous Valsalva maneuver may also enhance carotid sinus sensitivity.

## COMPLICATIONS

Neurologic complications of CSM are rare and are usually transient. In a recent review of neurologic complications in elderly patients undergoing this procedure, Munro and others found 7 complications from a total of 5000 massage episodes, for an incidence of 0.14%. [13] Reported deficits included weakness in 5 cases and visual field loss in 2 others. In 1 case the visual field loss was permanent. Patients in this study were excluded from CSM if they had a carotid bruit, recent cerebral infarction, recent myocardial infarction, or a history of VT or VF. The duration of massage was 5 seconds. Lown and Levine described 1 patient with brief facial weakness during several thousand tests. [2] Carotid emboli and hypotension have both been implicated as possible causes of the neurologic deficits. Unintentional occlusion of the carotid artery may also be responsible for some neurologic complications.

Cardiac complications include asystole, VT, or VF (Fig. 10-10) . A normal pause of <3 seconds is part of the physiologic response to CSM; a longer pause may be diagnostic of carotid sinus syndrome. In a review of reported cases of ventricular tachydysrhythmias, 5 cases were described. [14] All 5 patients were receiving digoxin, and in several cases, VT or VF followed AV block. Digoxin is associated with more prolonged AV block resulting from CSM, perhaps leaving these patients more vulnerable.

## INTERPRETATION

A pause >3 seconds, or a drop in systolic blood pressure >50 mm Hg in patients to whom CSM is administered while they are in a supine position, is diagnostic of the carotid sinus syndrome (Fig. 10-11) (Figure Not Available) . Patients should be supine during testing to reduce the risk of cerebral hypoperfusion.

When approaching patients with a tachydysrhythmia, the following principles can guide the interpretation of results (Table 10-1) :

1. CSM may slow the atrial rate in VT or complete heart block and may therefore demonstrate previously hidden P waves or obvious AV dissociation.

2. Abrupt changes in the heart rate without conversion are a result of increasing AV block.
3. Gradual slowing of the ventricular rate suggests the presence of a sinus rhythm. Only rarely does CSM decrease AV conduction in the presence of a sinus mechanism.
4. The dysrhythmias most likely to convert to sinus rhythm are PAT and paroxysmal nodal tachycardia.
5. Dysrhythmias that are associated with AV conduction defects (PAT with block, atrial flutter, atrial fibrillation) infrequently convert to a sinus rhythm, but the ventricular rate slows. Rarely, atrial slowing will be sufficient to allow 1:1 AV conduction, which may actually increase the ventricular rate (Fig. 10-12) (Figure Not Available) .

## CONCLUSIONS

The advent of adenosine and effective medications to treat congestive heart failure has diminished the therapeutic use of CSM. It still remains an important diagnostic tool, however, to determine the presence of the carotid sinus syndrome in patients with syncope. CSM should not be performed in patients with a carotid bruit, those who have had a recent stroke, or those with digoxin toxicity. Patients with coronary artery disease will have an enhanced response to CSM, and the technique should be used with care in these patients.

# Cardioversion

Cardioversion is the application of direct electrical current across the chest or directly across the ventricle to normalize the conduction pattern of a rapidly beating heart. Defibrillation refers to application of electrical energy to restore a fibrillating ventricle to normal sinus rhythm (see Chapter 11) .

The patient with a significant tachycardia may be asymptomatic or may complain of chest pain or discomfort, light-headedness, or shortness of breath. These symptoms are the result of altered cardiovascular physiology. Rapid cardiac rhythms allow less time for ventricular filling, resulting in reduced preload and hypotension. The reduced preload as well as the increased ventricular work caused by the rapid heart rate may also result in ventricular ischemia. Pulmonary capillary wedge pressures may also rise despite shortened filling time, due to reduced ventricular compliance secondary to ventricular ischemia. Elevated pulmonary capillary wedge pressures can then lead to pulmonary edema.

Termination of rapid rhythms to alleviate or prevent these symptoms must occur quickly to prevent further deterioration. Persistently poor cardiac output due to rapid heart rate results in development of a lactic acidosis that further compromises cardiac function and makes cessation of the dysrhythmia even more difficult. Unchecked myocardial ischemia may lead to infarction with its attendant sequelae. Drug therapy, rapid cardiac pacing, and cardioversion are the methods available to terminate tachydysrhythmias.

In many cases, direct-current cardioversion has specific advantages over drug therapy. The speed and simplicity of electrical cardioversion enhance its usefulness in the emergency department setting. Cardioversion is effective almost immediately, has few side effects, and is often more successful than drug therapy in terminating dysrhythmias. In addition, the effective dose of many antidysrhythmic medications is variable, and there is often a small margin between therapeutic and toxic dosages. Although they can often suppress an undesirable rhythm, drugs may also suppress a normal sinus mechanism or may create toxic manifestations that are more severe than the dysrhythmia being treated.

In the clinical setting of hypotension or acute cardiopulmonary collapse, cardioversion may be life saving. The key

**Figure 10-11** (Figure Not Available) Hyperreactive carotid sinus reflex. Gentle pressure was applied to the carotid sinus for 3 seconds, resulting in a pause in sinus rhythm of approximately 7 seconds. This syndrome may be the cause of syncope. *(From Bigger JT Jr: Mechanisms and diagnosis of arrhythmias. In Braunwald E (ed): Heart Disease. Vol. 1. Philadelphia, WB Saunders, 1980. Reproduced by permission.)*

| **TABLE 10-1** -- Effects of Carotid Sinus Massage on Various Cardiac Rhythms | |
| --- | --- |
| **Cardiac Rhythm** | **Usual Response to Carotid Sinus Massage (CSM)** |
| Sinus rhythm | 1. Smooth and gradual slowing of ventricular rate with return to original rate with termination of CSM. (The procedure may bring out diagnostic P waves.)<br>2. Occasionally produces varying degrees of heart block.<br>3. *Caution:* Possible prolonged asystole with hypersensitive carotid sinus syndrome. |
| Atrial flutter or atrial fibrillation | 1. Irregular slowing of ventricular rate by increasing atrioventricular (AV) block.<br>2. An effect is rarely absent.<br>3. CSM does not terminate the rhythm but may bring out diagnostic flutter or fibrillation waves.<br>4. *Caution:* Ventricular standstill may occur if CSM is prolonged. |

| | |
|---|---|
| Paroxysmal atrial tachycardia (PAT) | 1. No effect or abrupt termination of dysrhythmia. |
| PAT (Wolff-Parkinson-White syndrome) | 1. Varying results (slowing, no effect, termination). 2. CSM may unmask Wolff-Parkinson-White syndrome by increasing anomalous AV conduction. |
| Paroxysmal AV junctional tachycardia | 1. No effect or termination of dysrhythmia. |
| Nonparoxysmal junctional tachycardia | 1. No response. 2. *Caution:* CSM may be dangerous if rhythm results from digitalis toxicity. |
| Ventricular tachycardia | 1. No response in ventricular rate. 2. CSM may uncover AV dissociation by demonstrating P waves or a decrease in atrial rate. 3. If rhythm is ventricular parasystole, response may be variable. |
| Digitalis-toxic rhythms | 1. CSM should not be attempted. |

concepts in the use of this procedure include understanding the indications for its use, the equipment involved, the importance of adequate sedation, and the concerns for health-worker safety.

## BACKGROUND

The first successful defibrillation of the human heart was performed in 1947 by Beck. By the 1960s, electrical energy was being used to treat dysrhythmias other than VF. Alternating current remained in vogue until 1962, when Lown and colleagues advocated direct current countershock as the method of choice for terminating atrial fibrillation. [15] The use of direct current significantly decreased the incidence of VF following countershock (Fig. 10-13) (Figure Not Available) .

A brief burst of electrical current momentarily causes depolarization of the majority of cardiac cells and allows the sinus node to resume normal pacemaker function. In reentrant dysrhythmias, such as paroxysmal SVT and VT, cardioversion restores sinus rhythm by interrupting a self-perpetuating circuit. Cardioversion is much less effective in terminating tachycardias resulting from augmented automaticity, such as digitalis-induced dysrhythmias.

**Figure 10-12** (Figure Not Available) Acceleration of ventricular rate by carotid sinus stimulation (CSS). Continuous tracing. Upper strip shows 2:1 atrioventricular block: atrial rate = 102/min; ventricular rate = 51/min. The second and third strips were recorded during and after CSS, when the atrial rate was reduced to 68/min; a 1:1 response occurs. *(From Lown B, Levine SA: Carotid sinus--clinical value of its stimulation. Circulation 23:766, 1961. Reproduced by permission.)*

**Figure 10-13** (Figure Not Available) Typical converter-defibrillator monitor with display screen and readout. *(Courtesy of Datascope Corporation, Paramus, NJ.)*

## INDICATIONS AND CONTRAINDICATIONS

Cardioversion is indicated whenever there is a reentrant tachycardia causing chest pain, pulmonary edema, lightheadedness, or hypotension. This excludes those tachydysrhythmias that are known to be caused by digitalis toxicity as well as a known sinus tachycardia. It also is indicated in less urgent circumstances when medical therapy has failed. In elderly patients, in whom a prolonged rapid heartbeat can be anticipated to cause complications related to cardiac ischemia or dysfunction, early intervention with cardioversion may also be beneficial.

A reentrant tachydysrhythmia should be suspected when a sudden change in the heart rate occurs within a few beats. Unless the dysrhythmia is noted while the patient is being monitored, it can only be inferred from the patient's history of sudden onset of symptoms. In the unusual case of sinus-node reentrant tachycardia, rapid onset and offset may be the only clue. [16] Other clues to the presence of a reentrant dysrhythmia are a history of Wolff-Parkinson-White syndrome or another known accessory pathway syndrome. Ventricular rates in excess of those predicted for age strongly suggest an accessory pathway.

Dysrhythmias due to enhanced automaticity will not be terminated by uniformly depolarizing myocardial tissue because a homogeneous depolarization state already exists.

Enhanced automaticity is the cause of most cases of digitalis toxicity-induced dysrhythmia, sinus tachycardia, and, probably, multifocal atrial tachycardia. [17] Enhanced automaticity means that the threshold for phase 4 depolarization has been lowered or that the rate of ion leak during phase 4 has been accelerated. This effect on phase 4 depolarization is caused by alterations in the metabolic or chemical environment or on the cell membrane, causing pacemaker cells to fire more rapidly. Although cardioversion will not work in these cases, medications that suppress

automaticity, including potassium and magnesium, may be useful.

In digoxin toxicity, cardioversion is not only ineffective; it is also associated with a higher incidence of postshock VT and VF. [18] However, for a patient with a therapeutic digoxin level, the risk of cardioversion is now thought to be no different from that of other patients. [19] Digoxin is still generally withheld for 24 hours prior to cardioversion as a precaution against inadvertently elevated levels. Pregnancy at any stage is not a contraindication to cardioversion. [20] [21]

## EQUIPMENT AND SETUP

The critical components of preparation for cardioversion are IV access, airway management equipment, drugs for sedation, and monitoring and direct current delivery equipment (cardioverter).

Secure IV access is essential for delivery of sedatives, antidysrhythmics, fluids, and, possibly, paralytic agents. Although many of these drugs are not used routinely, if they are needed, timing is likely to be critical. A saline lock catheter of at least 20 ga should be inserted and firmly taped to the patient's skin.

A significant and preventable complication of procedures involving sedation is hypoventilation leading to hypoxia. Airway management equipment includes the secure IV catheter discussed above, working suction with tonsil-tipped device attached, bag-valve mask apparatus, oxygen, and appropriately sized laryngoscope and endotracheal tube (see Chapters 1 and 2) . A pulse oximeter is generally recommended for patients undergoing conscious sedation (see Chapters 6 and 35) . Another adjunct is continuous $pCO_2$ monitoring (see Chapter 6) . A rising $pCO_2$ level will be an earlier clue to hypoventilation due to sedation, because the $O_2$ saturation may remain normal for several minutes, especially if the patient has been preoxygenated.

Sedative medications (see Chapter 35) should be ready for use in labeled syringes, with a prefilled saline syringe available for flushing the catheter. Antidysrhythmic medications for ventricular dysrhythmias (e.g., lidocaine and bretylium) and for unexpected bradycardia (e.g., atropine) should be readily accessible.

The cardioverter device consists of 5 components (see Fig. 10-13) (Figure Not Available) : (1) a direct current depolarizer, which provides varying amounts of electrical current; (2) an oscilloscope screen for monitoring heart rate and rhythm; (3) access to a continuous electrocardiogram (ECG) readout to document the patient's course and response to treatment; (4) 2 removable electrode paddles, which can be applied easily to the patient's chest wall; and (5) a synchronizer, permitting discharge of energy outside the vulnerable period of the cardiac cycle. The synchronizer permits triggering of the electrical discharge by the R or S wave of the ECG (Fig. 10-14) (Figure Not Available) .

**Figure 10-14** (Figure Not Available) Phases of vulnerability for atrium and ventricle. Note that an alternating current shock of 0.20 second may end at the T wave even when synchronized with the R wave of the electrocardiogram. *(From Resnekov L: Theory and practice of electroversion in cardiac*

Paddles must be large enough to depolarize the majority of heart fibers simultaneously; therefore, most conventional paddles have an electrode diameter of at least 4 inches. Larger paddles also limit the risk of myocardial injury by decreasing the density of current passing through the myocardium.

## TECHNIQUE

If time permits, metabolic abnormalities such as hypokalemia and hypomagnesemia should be corrected before attempting cardioversion. At a minimum, hypoxia should be corrected with supplemental $O_2$ . If a patient has metabolic acidosis, compensatory hyperventilation after endotracheal intubation may be indicated prior to cardioversion. Respiratory acidosis should always be treated prior to the use of sedative drugs.

### Sedation

Cardioversion may be extremely painful or terrifying, and *patients must be adequately sedated prior to its use*. Patients who are not adequately sedated may experience extreme anxieties and fear. [22] Several IV medications are available for sedation of patients prior to cardioversion. These include etomidate (0.15 mg/kg), midazolam (0.15 mg/kg), methohexital (1 mg/kg), propofol (1.5 mg/kg), and thiopental (3 mg/kg). [23] [24] In addition, fentanyl (1.5 mug/kg), a synthetic opioid analgesic, is sometimes administered 3 minutes prior to induction.

Midazolam (Versed) is probably the most commonly used agent, with induction occurring about 2 minutes after a dose of about 0.15 mg/kg, or at least 5 mg for an average-sized adult. Although induction with midazolam takes slightly longer than the other medications, it has the advantage that a commercial antagonist, flumazenil, is available for reversal if necessary. Small additional doses of fentanyl (1 to 1.5 mug/kg) may be added for more profound sedation. Fentanyl can cause respiratory depression, but it is reversed with naloxone. Methohexital has the advantage of quick onset and somewhat shorter duration than midazolam, but it has a rare association with laryngospasm. All the drugs except etomidate cause a small drop in blood pressure, and infusion of propofol and etomidate is painful.

In elderly patients the pharmacodynamics and kinetics are altered by coexisting illness and polypharmacy, rather than by any intrinsic effect of old age. [25] Older patients with medical conditions such as congestive heart failure, renal failure, cancer, or malnutrition will therefore experience deeper, prolonged sedation with increased respiratory depression. Drug dose should be reduced in these patients.

Administer the anesthetic agent(s) IV over about 30 seconds and wait until the patient is unable to follow simple commands and loss of the eyelash reflex is noted. Pushing the agent too quickly may result in hypotension; pushing the agent too slowly may not allow blood levels to reach a therapeutic range, if the agent has a rapid rate of metabolism.

**Cardioverter Use**

Selection of synchronized or nonsynchronized mode is the next critical step. In "synchronized" mode, the cardioverter searches for a large positive or negative deflection, which it interprets as the R or S wave. It then automatically discharges an electric current that lasts <4 msec, avoiding the "vulnerable" period during repolarization when VF can be easily induced. When the cardioverter is set to "synchronize," a brief delay will occur after the buttons are pushed for discharge, as the machine searches for an R wave. This delay may be disconcerting to the unaware operator.

If concern exists about whether the R wave is large enough to trigger the electrical discharge, the clinician can place the lubricated paddles together and press the discharge button. Firing should occur after a brief delay. When the R- or S-wave deflection is too small to trigger firing, change the lead that the monitor is reading or move the arm leads closer to the chest. If there is no R or S wave to sense, as in VF, then the cardioverter will not fire. *Always turn off "synchronization" if VF is noted.*

**Electrode Contact**

A number of substances can be used to ensure good contact between the paddle and the skin (see Chapter 11) ; it is necessary that they be nonflammable and have a low electrical resistance. Conductive gel or paste is most commonly used, but waxy conductive pads are also available. Generous use of conductive gel on the underside and especially along the edges of the electrode paddles is essential, both to reduce transthoracic impedance and to prevent skin burns. Paste should be applied liberally but must not run onto the skin between the paddles, because the paste may divert current over the skin surface and away from the heart. Even under ideal circumstances only 10 to 30% of the total current passes through the heart, so diversion over the skin may significantly reduce the effectiveness of an electric discharge. Saline-soaked pads are therefore generally not desirable. Pre-gelled adhesive electrode pads are useful if available.

**Electrode Position**

Electrode paddles may be positioned in 2 ways on the chest wall: (1) the anterolateral (or base and apex) position, with one paddle placed in the left fourth to fifth intercostal space, midaxillary line, and the other just to the right of the sternal margin in the second to third intercostal space (Fig. 10-15) (Figure Not Available) ; or (2) the anteroposterior position, with one paddle placed anteriorly over the sternum and the other on the back between the scapulae (Fig. 10-16) (Figure Not Available) . The anterolateral position is used for emergent cardioversion, when placement of an electrode on the patient's back may not be feasible. Paddles should be pressed firmly against the skin to avoid arcing or skin burns.

Safety is a key concern in the performance of cardioversion. Any staff member acting as a ground for the electrical discharge can be seriously injured. The operator must

announce "all clear" and give staff a chance to move away from the bed before discharging the paddles. Care must be taken to clean up spills of saline or water, because they may create a conductive path to a staff person at the bedside.

**Energy Requirements**

The amount of energy required for cardioversion varies with the type of dysrhythmia, the degree of metabolic derangement, and the configuration and thickness of the chest wall (Table 10-2) . Obese patients may require a higher energy level for cardioversion; the anteroposterior paddle position is sometimes more effective in these patients. If patients are shocked while in the expiratory phase of their respiratory cycle, energy requirements may also be lower.

Ventricular tachycardia in a hemodynamically stable patient should be treated with lidocaine followed by procainamide, and bretylium should be given if necessary. If these drugs are unsuccessful, cardioversion is then used. Cardioversion

*Figure 10-15* (Figure Not Available) Anterolateral paddle electrode position. *(From Suratt PM, Gibson RS: Manual of Medical Procedures. St Louis, CV Mosby, 1982. Reproduced by permission.)*

with 10 to 20 J is successful in converting VT in more than 80% of cases. Cardioversion will be accomplished with 50 J in 90% of cases, and conversion should be initially attempted at this energy level. [26] Cardioversion should be synchronized unless the T wave is large and could be misread as the R wave by the cardioverter. If this is unsuccessful, the energy level should be doubled, and doubled again if necessary, until a perfusing rhythm is restored. Immediately following conversion of VT, antidysrhythmic medication should be given to prevent recurrence.

Patients with pulseless VT should be initially shocked with 200 J, followed by 300 J if the first shock is not successful.

Reentrant SVTs generally respond to low energy levels. Atrial flutter, for example, usually requires <50 J for conversion. [27] Cardioversion of atrial flutter in the emergency department is indicated when the ventricular rate is not slowing in response to pharmacologically enhanced AV-node blockade, or if the patient is unable to tolerate the aberrant rhythm.

The majority of patients with PAT respond to adenosine. If they do not, or if urgent conversion is needed due to high ventricular rate, electric countershock should be administered in the synchronized mode at 50 J, and doubled if necessary.

In atrial fibrillation, the response to cardioversion is dependent on the duration of atrial fibrillation and the underlying cause. Cardioversion is successful in 90% of cases

*Figure 10-16* (Figure Not Available) Anteroposterior paddle electrode position. *(From Suratt PM Gibson RS: Manual of Medical Procedures. St Louis, CV Mosby, 1982. Reproduced by permission.)*

| | Energy Setting (J or Watt-sec) |
|---|---|
| **TABLE 10-2** -- Energy Settings for Cardioversion [*] | |
| Atrial fibrillation | |
|   Initial | 100 |
|   Subsequent | 200 |
| Atrial flutter | |
|   Initial | 20 |
|   Subsequent | 50<br>100 |
| Atrial tachycardia | |
|   Initial | 50 |
|   Subsequent | 100<br>200 (maximum) |

| Ventricular tachycardia | |
|---|---|
| Initial | 50-100 |
| Subsequent | 200 |

* Note: "Synchronization" always used.
For elective cardioversion of atrial fibrillation, 3 weeks of prior warfarin therapy is suggested. Embolic rates are highest following cardioversion in patients with mitral stenosis, enlarged left atrium, and history of embolic disorders. Quinidine is usually given before or after cardioversion to maintain sinus rhythm. Attempts to cardiovert patients with digitalis toxicity can lead to VT/VF. This is not seen with therapeutic digoxin levels.

secondary to hyperthyroidism but in only 25% of cases secondary to severe mitral regurgitation. [28] However, 50% of cases revert within 6 months, especially those with long-standing atrial fibrillation. [29] [30]

Most patients with atrial fibrillation do not require cardioversion in the emergency department unless their ventricular response is high due to a bypass tract, as in Wolff-Parkinson-White syndrome. They may also require cardioversion when sequelae of rapid ventricular contraction are present or anticipated and the ventricular rate is not responding to drug therapy aimed at slowing AV node conduction. Conversion of atrial fibrillation generally requires more energy than the reentrant SVTs (about 100 J in most cases). [31]

**COMPLICATIONS**

Complications of cardioversion may affect the patient, particularly the patient with a cardiac pacemaker, as well as health care personnel at the bedside. Patient complications are dose related and may involve the airway, heart, or chest wall, or they may be psychological.

Injuries to health care personnel with cardioversion/defibrillation include mild shock and burns, and there is a reported incidence of 1 injury per 1700 shocks for paramedics in the field. [32] Of the 13 injuries reported in this study, 15% were due to equipment failures, 23% were due to injuries during testing or demonstrations, and 23% were due to arcing of the electric shock from the paddle to an electrode on the patient's chest, whereas the remainder were due to direct contact with the patient or with the stretcher.

Hypoxia may result if sedation is excessive or if the airway becomes compromised. With proper preparations and precautions, airway complications can be minimized.

Respirations may also be depressed by any of the anesthetic agents,

and adequacy of tidal volume must be continually assessed by either direct observation or end-tidal $CO_2$ monitoring. If another physician is available, he or she should be placed in charge of monitoring the patient's airway. Routine supplemental $O_2$ is suggested for all patients undergoing sedation.

Chest wall burns resulting from electrical arcing are generally superficial partial-thickness burns, although deep partial-thickness burns have occurred. [33] These are preventable by adequate application of conductive gel and firm pressure on the paddles. Paddles should not be placed over medication patches or ointments, especially those containing nitroglycerin, because electrical discharge may cause ignition resulting in chest burns. [34]

Cardiac complications following cardioversion are proportionate to the energy dose delivered. In the moderate energy levels used most commonly, the hemodynamic effects are small. At higher energy levels, however, complications include dysrhythmias, hypotension, and, rarely, pulmonary edema, which may occur several hours after the countershock. [35] A transient failure of myocardial $O_2$ extraction due to a direct effect on cellular mitochondria has been proposed as an explanation for some of these cardiac complications. [36]

The dysrhythmias following high-dose (approximately 200 J) direct-current shocks include VT and VF, bradycardia, and AV block, in addition to transient and sustained asystole. Sustained VT or VF was reported following 7 of 99 shocks in a study of patients undergoing electrophysiologic study and requiring cardioversion for VT, VF, or atrial fibrillation. [37] These episodes occurred only in the patients with prior VT or VF. Patients with ischemia or known coronary artery disease appear to be at much higher risk for significant postshock bradycardia, with rate support pacing required after 13 of 99 shocks in the study. Asystole requiring pacing occurred only once in 99 countershocks.

Two types of VF following cardioversion have been described. The first variety occurs immediately after countershock and is easily reversed by a second, nonsynchronized shock. This type of VF results from improper synchronization, with discharge of current occurring during the vulnerable period. The second variety, which is more ominous, occurs approximately 30 seconds to a few minutes following attempted cardioversion. This dysrhythmia is characteristically preceded by the development of PAT with block or a junctional rhythm. In affected patients, it may be very difficult to convert the dysrhythmia to a sinus rhythm. This phenomenon occurs in patients who have been taking digitalis glycosides and is presumably a manifestation of digitalis toxicity (Fig. 10-17) (Figure Not Available) . [38] [39]

In the event of VF following cardioversion, the equipment and manpower are present for immediate defibrillation. If postcardioversion VF occurs, switch the cardioverter to "nonsynchronized" before attempting defibrillation. Electrical discharge will not occur in the "synchronized" mode, because the machine will be searching for a nonexistent R

wave.

VF is much more likely to occur if depolarization occurs on the T wave. If a patient has large T waves in the lead selected for cardioverter sensing, the electric shock may discharge during the vulnerable period of the cardiac cycle, resulting in VF. [40] Always examine the complexes on the cardioverter monitor carefully for large T waves and, if necessary, change the sensing lead. A randomly firing pacemaker can also be sensed by the cardioverter, resulting in countershock during the vulnerable period. [41]

Transient and intermittent ST-segment elevation has also been reported to occur (though rarely) after cardioversion, with myocardial injury or coronary vasospasm offered as possible explanations. [42]

An increase in serum enzyme levels (creatine kinase, lactate dehydrogenase, aspartate aminotransferase) may also occur following cardioversion, and the incidence has been reported to be between 10 and 70%. The enzyme rise is usually a consequence of skeletal muscle injury rather than myocardial damage. Cardioversion does not alter the enzyme profile of patients with myocardial infarction. [43]

## CONCLUSIONS

Cardioversion is a safe and effective method of quickly terminating reentrant tachycardia. Complications related to psychological trauma, respiratory depression, and unintentional health-worker shock can be avoided with proper precautions. Adequate sedation is essential. Synchronized shock should be administered after close scrutiny of the lead used for sensing, to be sure that the R or S wave is significantly larger than the T wave. Be prepared for postshock VT or VF, and if VF occurs, switch the cardioverter to "nonsynchronized" and defibrillate. Atropine and temporary pacing equipment should be available to treat postshock bradycardia, especially in patients with myocardial ischemia or infarction.

# Chapter 11 - Defibrillation

**Jerris R. Hedges, Michael I. Greenberg**

Ventricular fibrillation (VF) can be associated with myocardial ischemia or infarction, marked electrolyte disturbances, electrical injuries, pronounced hypothermia, or drug toxicity (e.g., that caused by cyclic antidepressants, quinidine, and digitalis). Because this rhythm is clearly incompatible with life, it must be treated rapidly and effectively. Electrical defibrillation is defined as the conversion of VF to an alternative (preferably supraventricular) rhythm. Although brief periods of VF with spontaneous reversion to a sinus rhythm have been recorded, VF is usually irreversible without such electrical countershock. [1]

Ventricular fibrillation is the primary cause of most cases of sudden cardiac death. [2] Contrary to popular belief, the majority of victims of sudden death have not suffered a myocardial infarction. Rather, VF is the culprit. However, many victims of sudden death have evidence of advanced coronary disease, although it may have been previously asymptomatic. In addition, survivors of sudden death attributable to VF are at increased risk of suffering a recurrence, although this chance may be lessened with the use of long-term beta-blocker therapy. [3] [4]

With the advent of portable defibrillation units and out-of-hospital and in-hospital cardiac resuscitation teams, the challenge of providing early defibrillation treatment to the potential victim of sudden death in VF is being addressed aggressively. It has been estimated that 10-35% of out-of-hospital VF cardiac arrest victims could be saved if closed chest cardiac massage and artificial ventilation were provided in <4 minutes and followed by defibrillation in <8 minutes of collapse. [5] [6] However, optimal survival rates depend on prompt recognition and treatment of VF; several recent urban studies report an overall survival to discharge rate of only 2-4% in patients experiencing out-of-hospital VF cardiac arrest. [7] Although survival from VF is highly dependent on many variables, the timeliness of defibrillation may be the most important intervention determining the prognosis in out-of-hospital cardiac arrest. [8] Because prompt, effective defibrillation is equally important for in-hospital VF cardiac arrests, all physicians and nurses should ideally be thoroughly familiar with this procedure.

## BACKGROUND

The concept of electrical shock therapy in resuscitation can be traced to the experiments of Abildgaard in the 18th century. Abildgaard described chickens as being "lifeless" following electrical shocks and noted successful resuscitation after the use of additional shocks. Subsequent animal studies were reported by Preust and Batelli. [9] In 1947 the first successful human defibrillation using the direct application of electrical current to the heart was reported by Beck and coworkers. [10] Nine years later, Zoll and colleagues reported the first successful cardiac defibrillation in a clinical setting using an alternating current (AC) electrical shock applied externally to the thorax. [11]

Portable direct current (DC) defibrillators were introduced by Lown and colleagues [12]

and Edmark and coworkers [13] during the 1960s. DC defibrillators opened the way for out-of-hospital defibrillation modalities. Although the basic design of modern defibrillators has not changed much since the 1960s, modifications in paddle size, energy delivered, energy waveform, conducting materials, and pharmacologic enhancement of defibrillation have been topics of active investigation since the mid-1970s.

## INDICATIONS AND CONTRAINDICATIONS

Electrical defibrillation of the heart in the presence of VF is indicated whenever immediate spontaneous conversion to an effectively perfusing rhythm does not occur. "Quick-look" paddles permit immediate monitoring of the arrested patient's rhythm before electrical defibrillation. These can be quite helpful in the emergent situation to determine if defibrillation is indicated. Patients who are unresponsive and who have regular tachydysrhythmias as diagnosed by the "quick-look" method are best treated with synchronized cardioversion (see Chapter 10) , although it is important to remember that if monitoring is not immediately available, an initial unsynchronized countershock may be life saving.

The precise timing for defibrillation during resuscitation has been the subject of debate, but most authorities are in agreement that an initial electrical shock should be applied to the victim of sudden death before drug administration. In most resuscitations, closed chest cardiac massage and concomitant ventilation support are initiated while the defibrillator is being readied. If the patient is unconscious, apneic, and pulseless, it is reasonable to assume that an episode of VF is occurring if cardiac monitoring is not available with the device. In such instances, an immediate attempt at defibrillation is warranted. Although asystole and, more rarely, ventricular tachycardia (VT) may appear clinically similar, an immediate countershock is unlikely to worsen either clinical situation.

Because "fine" VF can occasionally masquerade as ventricular standstill or asystole, monitoring paddle electrodes should be rotated 90° from their original position or the monitor lead changed before the decision to withhold defibrillation in the victim of sudden death is made (Fig. 11-1) . [14] Low-voltage VF likewise is not a contraindication to defibrillation, because it may reflect low monitor gain or a problem with monitor lead or paddle placement.

## CHARACTERISTICS OF VENTRICULAR FIBRILLATION

VF is characterized on the electrocardiogram (ECG) by the presence of rather low-amplitude baseline undulations that are variable in both amplitude and periodicity. Although many consider VF to represent an electrically random process, electrical directionality to depolarization (i.e., wavefronts) can exist. [14] Mechanically, VF represents an uncoordinated and distinctly disorderly and mechanically ineffective contractile process. Resulting from the lack of effective contractile cardiac function, tissue perfusion is markedly compromised, and if it is not promptly corrected, death

becomes inevitable.

At the tissue level, VF represents a disorganization of the orderly depolarization sequence that usually occurs in the ventricles. Normally, the refractory period of depolarized muscle prevents the development of reentrant ventricular rhythms. When ischemia, electrolyte disorders, cardiac drug toxicities, rapid ventricular rates, hypothermia, and certain other disorders exist, refractory periods may shorten or conduction velocities may tend to increase in certain areas of the ventricle. Wandering depolarization wavefronts that become self-perpetuating can develop. A combination of disorders of impulse formation (automaticity) and impulse conduction (reentry) contribute to the development of VF. [15] [16] The tendency for VF to occur is enhanced by, but is not entirely dependent on, premature ventricular impulses that occur during the "vulnerable" period of the cardiac cycle represented by early ventricular repolarization (T wave). [17]

Asynchronous ventricular depolarization may be confined to a small area of the ventricle if the remaining ventricle is refractory to further stimulation. Several studies have shown that a critical muscle mass is required for VF to be self-sustaining, possibly explaining why VF is so uncommon in infants undergoing resuscitation (who usually die from respiratory arrest). A large mass of muscle involved in asynchronous depolarization having a brief refractory period and a slow conduction velocity increases the tendency for the ventricles to fibrillate.

Cummins and coworkers [18] [19] have classified VF on the basis of average peak-to-trough wave amplitude (Table 11-1) (Table Not Available) . They note that the amplitude of the VF waveform is associated positively with the probability of resuming a perfusing rhythm. However, the clinical importance of coarse vs. fine VF in relation to ultimate survival is unclear. Signals that have an amplitude of <1 mm (when the monitor is calibrated at 10 mm/mV) should be considered to indicate asystole, [18] [19] because countershock of such low-amplitude rhythms is only rarely associated with conversion to a perfusing rhythm. [20] [21]

Electrical defibrillation represents the simultaneous depolarization of sufficient ventricular tissue to render the tissue that is ahead of the VF wavefronts refractory to further electrical conduction. Following generalized depolarization, the sinus node or other pacemaker region of the heart

| TABLE 11-1 -- Classification of Ventricular Fibrillation (VF) Based on Mean Waveform (Peak to Trough Averaged Over a 3- to 6-Second Interval) Amplitude |
| --- |
| (Not Available) |

with the highest degree of automaticity can then acquire dominance of a well-ordered

depolarization-repolarization sequence.

## DEFIBRILLATOR CHARACTERISTICS

General standards and product evaluations for cardiac defibrillation devices are beyond the scope of this chapter. Rather, this section discusses the major components and design configurations of a prototypical defibrillator and how they relate to effective defibrillation.

### Waveforms

Despite the fact that the first successful human defibrillation was performed using alternating current (AC), [10] Lown and others demonstrated that direct current (DC) is more effective than AC in accomplishing defibrillation and results in a significantly reduced incidence of postcardioversion dysrhythmias. [12] Only DC defibrillators are in clinical use today. Most of the modern defibrillators use a damped half-sinusoidal waveform or a trapezoidal (truncated exponential decay) waveform. The trapezoidal waveform can be modified to resemble a square waveform. The more square the waveform, the more effective it is for experimental defibrillation. [22] Furthermore, in a comparison of square waveforms and damped half-sinusoidal waveforms for animal defibrillation, it was found that less peak current per kilogram was needed with the square waveforms, although the average current levels were equivalent. [23] Furthermore, the delivered waveform is dependent on thoracic resistance. In clinical practice, however, there is little clinical difference in the effectiveness of the currently available waveforms.

### Stored Energy

Because the ability to defibrillate is dependent primarily on current delivered to the myocardium, [24] the stored energy is one factor that, coupled with transthoracic impedance and internal defibrillator energy loss, contributes to successful defibrillation. Each defibrillator is calibrated by measuring the current delivered as a function of time across a 50-ohm impedance. It is important to note that defibrillators do not always deliver the energy indicated on the device. With a "stored" energy of 400 J (1 J = 1 watt-sec), from 155 to 410 J may be delivered. It stands to reason that the current (for a square waveform) delivered to the myocardium is related to the energy delivered by the defibrillator, the electrical impedance in the device and chest, and the duration of current flow:

Energy (J) = current (amperes)$^2$ × impedance (ohms) × duration (seconds)

Obviously, any increase in transthoracic impedance will further reduce delivered current.

### Device Switches

Many portable (capable of battery operation) defibrillators have separate power switches for the accompanying monitor and recorder and for the defibrillator. The same

device can be used for cardioversion as well as defibrillation. Before attempting defibrillation, the physician must be completely familiar with the location and operation of the controls. This knowledge will assure that, for example, the operator will avoid attempting to defibrillate a patient when the defibrillator portion is not yet turned on.

Defibrillators also have a control permitting *synchronous cardioversion*. The physician or other operator must be certain that the control is correctly set to the *asynchronous* mode to permit defibrillation; otherwise, the device in the synchronous mode would "wait" indefinitely for a nonexistent repetitive series of R waves prior to discharging.

Energy settings may be determined by a switch or a dial setting or may be read off of a meter permitting a continuous range of settings. In each case the operator must be aware of the need to charge the device initially and to recharge after each discharge. The mechanism of charging the device may be intrinsic to the setting of the dial or meter but more commonly requires the use of a separate charge button on the device control panel or paddle handle. Full charge accumulation usually takes from 2 to 5 seconds following activation of the charging mechanism.

Discharge controls are generally present on the paddle handles, allowing the operator to position and hold the paddles in place while delivering the charge. Alternatively, there may be a separate control on the panel. The simultaneous activation of the control on both paddles is usually required for energy discharge.

Monitor controls permit alteration of lead-monitored image size and often allow the selection of chest lead electrode vs. paddle electrode monitoring. The latter is desirable when an initial "quick-look" rhythm evaluation is desired before placement of the chest lead electrodes. A hard copy paper recorder for documenting rhythms may operate in a real-time, delay, or standby mode.

**Paddles or Electrode Pads**

Most commercial defibrillator devices come equipped with adult-sized paddle electrodes with diameters between 8 and 9 cm. Canine studies have shown that slightly larger (12.8 cm diameter) paddles are more effective for defibrillation, [25] and produce less myocardial injury. [26] Paddles that are slightly smaller (4.5 cm diameter) produce greater damage at the same energies. The larger paddles may permit a greater amount of muscle to be depolarized while simultaneously decreasing the potentially damaging current density. If the paddles are too large with respect to the heart (e.g., if adult paddles are used during infant resuscitation), the current density may be less and defibrillation may be rendered less effective. [27] The recommended minimum diameter for infant transthoracic paddles is 2.2 cm. Atkins and colleagues suggest that because of their reduction in transthoracic impedance, the larger adult electrodes should be used whenever a child's thorax is large enough to permit electrode-to-chest contact over the entire paddle surface. [28] This transition in paddle size occurs at 1 year of age or at an approximate weight of 10 kg. Because the time delay required to change from adult to pediatric paddles may be excessive, the routine use of adult-size paddles on any child who weighs >10 kg is recommended. [29]

Some defibrillators have a flat posterior ground shield rather than a second paddle for lateral chest wall placement.

With the two-paddle system, charge and discharge controls are generally present on the paddles.

The metal composition of the paddle electrode will affect transthoracic impedance to the defibrillation discharge. Most modern defibrillators use stainless steel because of its durability, although copper alloys and several other metals provide a lower transthoracic impedance.

Defibrillation can also be performed with self-adhesive electrode pads applied to the skin. The self-adhesive monitor/defibrillator pads appear to perform as well as or better than hand-held paddles. [30] Stults and colleagues, in a controlled out-of-hospital study, found that the use of self-adhesive pads shortened the time to successful defibrillation, reduced the number of countershocks, reduced the amount of rhythm artifact, and improved survival until hospital admission when compared with the use of standard hand-held paddles. [27] However, the expense of self-adhesive defibrillator pads has limited their general acceptance. If future studies support their superiority in other clinical settings, it is likely that the pads will gain wider acceptance.

**Conductive Materials**

Transthoracic impedance varies with the type of conductive material applied between the paddles and the chest wall. [31] For paddles that are 8.0 cm in diameter, the transthoracic impedance is $91 \pm 20$ ohms for bare contact, $71 \pm 11$ ohms for saline-soaked gauze, and $64 \pm 15$ ohms for Redux Paste. Clearly, electrode-skin agents will reduce impedance and allow more current to be delivered to the heart, but the ideal agent is the subject of some debate. Ewy and Taren recommend that Corgel, Redux Paste, American Writer, GE Gel, Electrode Jelly, or Trucon Electrode Paste be used to minimize impedance. [31] Saline-soaked gauze pads may be used, although one must be careful not to allow the saline, coupling gels, or paste to flow into a "bridge" on the skin between the electrodes, creating a potential fire hazard. Although some form of coupling medium (e.g., paste, cream, gel, pad) should be used to reduce impedance, data conflict regarding which product is optimal. Although Redux Paste (Hewlett-Packard) has been associated with significantly lower transthoracic impedance, a statistically significant increase in the success of defibrillation attempts has not been demonstrated with any specific product.

Hummel and coworkers investigated conductive materials with regard to their potential to overheat and to spark. [32] They found that the products that offer lower impedance (e.g., Redux Paste, Signagel) remained stable and did not spark after 4 or 5 defibrillation discharges, which was seen with the higher impedance products (e.g., Redux Cream, Aquasonic 100, EKG Sol, Spectra 360, and Derma-Jel). [32]

# PROCEDURE

Sudden death victims suspected to be in VF should be defibrillated as quickly as possible (Fig. 11-2) . Current recommendations are that defibrillation be attempted up to 3 times prior to initiating cardiopulmonary resuscitation (CPR), [33] unless additional personnel are on the scene and can perform CPR while the defibrillator device is being readied for use.

After applying conductive material to the entirety of the

**Figure 11-2** Algorithm for management of unwitnessed ventricular fibrillation (VF) or witnessed VF not responsive to precordial thump. After each therapy, the rhythm is reassessed, and the rescuer must evaluate the need for continued treatment for VF. If VF persists, the rescuer continues through the protocol. CPR, cardiopulmonary resuscitation; IV, intravenously.

paddle's conductive surface, the paddles should be firmly applied to the chest wall and held there by the operator. Ideally, the current should pass through the heart. One paddle should be positioned to the right of the upper sternum below the clavicle; the second paddle is placed just to the left of the nipple in the midaxillary line and is centered in the fifth intercostal space (see Fig. 10-15) (Figure Not Available) . Placement of both paddles close together on the anterior chest wall should be avoided. In some paddle sets, each paddle is labeled as either "sternum" or "apex" so that any rhythm detected on the monitor can be properly aligned. This feature is irrelevant

for defibrillation but is important for cardioversion. Anteroposterior paddle positioning is also acceptable and may deliver more current to the heart. The anterior paddle is placed to the left of the sternum over the precordium and the posterior electrode is placed just to the left of the spine directly posterior to the heart (see Fig. 10-16) (Figure Not Available) .

With the monitor turned on and set to display the "paddle" electrodes, the rhythm is evaluated. If a flatline rhythm is detected, the monitor gain must be increased fully in order to rule out the presence of a "fine VF" tracing. Should the tracing remain flat during a brief pause in closed-chest cardiac massage, the paddles should be rotated 90° from the original position and the rhythm reassessed. The incidence of VF masquerading as asystole was approximately 2.5% in 1 out-of-hospital study of patients with an initial flatline monitor rhythm. [19] Other conditions can cause a flatline rhythm during VF arrest. Cummins [33] recommends that the rescuer also check all monitor cable connections to the patient and defibrillator, check the ECG size control, and check the power supply. If VF is observed during any of these maneuvers, defibrillation should proceed without delay. Should a bradycardiac or asystolic rhythm be detected, standard resuscitation measures, including basic CPR, correction of hypoxia, administration of catecholamines, correction of volume or cardiac filling deficiencies, and emergency cardiac pacing, should be initiated when indicated. Although defibrillation should have no theoretical benefit for asystole, the use of countershock will result in the development of a QRS rhythm in a very small percentage of patients with flatline rhythms who receive

such shocks. Presumably, such cases represent instances of fine VF simulating a flatline rhythm on the EGG.

In cases of "fine VF" in which a patient is wearing an implanted (subcutaneous) pacemaker, the pacer spikes may initially appear to be a paced but nonconducted rhythm; attention to the baseline and lack of ST changes characteristic of capture should reveal the true nature of the dysrhythmia. [34] Because injury to the pacemaker pulse generator [35] and to the myocardium can occur by transmission of current down the pacing electrode, the physician must be careful to situate the defibrillator paddle at least 12 cm away from the pulse generator.

In the presence of VF, the paddles should be immediately charged to a stored energy of 2 J/kg for children and 200 J for adults. Keep in mind that separate power switches may be needed to turn on the defibrillator and to store the charge. The amount of charge is usually set by a button or a dial on the control panel. In most devices, the preset level of charge (energy) can be stored if a button on the "apex" paddle is depressed. The physician should always check to be certain that the defibrillator is not in the synchronous (cardioversion) mode.

Once the paddles are charged, the physician should instruct all personnel to "*STAND BACK*" from the patient and the stretcher to avoid stray discharge. However, there is no need for the individual who is bag-tube ventilating the patient to drop the bag and stand back *if their only contact is a rubber or plastic bag*. That individual is not touching conductive materials and will thus be protected from electrical shock. The defibrillator operator, in particular, must be sure that his or her only direct contact with the patient or stretcher is with dry paddle handles. The patient is allowed to exhale passively to minimize transthoracic impedance, [36] while firm (25 lb) pressure is applied through the paddles to the thorax. [37] To minimize energy decay inside the device, the energy in the paddles is discharged through the chest as soon as possible after charging. Simultaneous depression of both paddle discharge buttons is essential for discharge. Anticipation of patient extremity motion subsequent to discharge of the paddles will minimize operator and patient injury.

Should no skeletal muscle contraction occur following simultaneous depression of the discharge buttons, the physician should ensure that firm chest wall contact has been made (some devices will not discharge without adequate contact), that the device is set in the asynchronous mode, that a charge has been stored and that the defibrillator (not just the monitor) is turned on, and the battery is not depleted (when operating off the storage battery). If there is no muscle contraction even when these factors have been ruled out, a replacement defibrillator should be immediately brought into use.

After the first shock, the paddles should remain in place for 5 to 10 seconds to enable the physician to check for an organized rhythm while ventilation is continued. While waiting to analyze the rhythm, the rescuer should recharge the paddles for an immediate second defibrillation (200 to 300 J for adults, 2 to 3 J/kg for children) should VF persist. Should VF continue after the second shock, an immediate third defibrillation (360 to 400 J for adults; 4 J/kg for children) should be given. If the third defibrillation is unsuccessful, closed-chest cardiac massage should be continued, hypoxia corrected,

and alpha-agonist catecholamines administered to elevate the diastolic pressure and to improve coronary perfusion. [38] [39] Following circulation of the catecholamine agent, an attempt at defibrillation should be repeated. Currently there are no convincing data to suggest that "high-dose" epinephrine use is associated with superior long-term survival compared to standard doses in either children or adults. [40]

Infants who develop VF often have underlying cardiac disease and are often taking digitalis preparations. Because excessive defibrillation energy may produce irreversible VF in the patient suffering toxic effects of digitalis, the lowest available energy level should be used for the initial defibrillation. If the initial energy dose is unsuccessful, the energy level can be increased cautiously for successive countershocks.

Additional therapy (see below) can be undertaken to enhance defibrillation. Adequate ventilation, cardiac massage, and correction of electrolyte disorders are intrinsic to every resuscitation and are not discussed further. In addition, evaluation of the patient for hypothermia and rapid core rewarming when indicated (see Chapter 71) should not be overlooked. When practical, electronic monitoring devices and transvenous pacemakers should be turned off or, preferably, disconnected from the patient to avoid equipment damage. Recently manufactured patient monitoring devices, however, have built-in protective filter circuitry, which makes equipment damage an unlikely occurrence.

## ENHANCING DEFIBRILLATION SUCCESS

### Early Defibrillation

Energy requirements for conversion of VF may increase dramatically shortly after the onset of VF. [37] The rationale for

early defibrillation is that in the absence of adequate coronary perfusion, cellular metabolism continues with the depletion of energy substrates and the accumulation of toxic metabolites. Electrophysiologic changes secondary to cellular ischemia develop rapidly and contribute to continued asynchronous transmission of VF wavefronts.

Clinical investigation of immediate defibrillation rather than drug therapy preceding defibrillation is limited. Martin and coworkers, in a retrospective analysis of out-of-hospital VF resuscitation, found that survival until hospital discharge was increased when CPR followed by immediate defibrillation was used rather than CPR and drug therapy before countershock. [41] The group that received drug therapy first had a longer mean time until defibrillation (12 minutes additional), which was explained in part by the time required for IV line placement, drug administration, and drug circulation. Nonetheless, until further clinical studies are available, immediate defibrillation for VF appears to be the most appropriate course of action.

## Transthoracic Impedance

Delivery of current to the heart is dependent on the energy supplied to the paddles and the impedance to transmission of that energy. The greater the impedance or resistance, the less the delivered current. Reported values for human transthoracic impedance using electrodes 8.0 cm in diameter range from 50 to 100 ohms with a mean of 75 ohms. [42] We have previously discussed the importance of paddle electrode composition and size, conductive materials, applied paddle-to-chest-wall pressure, state of ventilation, and location of the paddle on the chest wall (Table 11-2) (Table Not Available) .

The transthoracic impedance of direct current discharge also decreases with higher energy shocks, with increasing number of previous countershocks delivered, [42] [43] and with decreasing interval between the discharges. Unfortunately, each of the aforementioned maneuvers is also associated with an increased potential for myocardial injury.

Nonetheless, one may be faced with the need to defibrillate a very obese patient who is unresponsive to standard paddle placement and maximum device energies. Should such a situation exist, the patient can be rolled on the side and anteroposterior defibrillation attempted. Should this prove unsuccessful, a second defibrillating device can be

| **TABLE 11-2** -- Determinants of Transthoracic Impedance |
| --- |
| (Not Available) |

simultaneously charged and used to administer countershock immediately following discharge of the first defibrillator. It is interesting to note that a canine study of internal defibrillation indicated that 2 sequential shocks over different pathways reduced both total energy and peak voltage required to terminate VF. [44] Hence, sequential defibrillation using slightly different paddle placements may be beneficial independent of the concurrent reduction in transthoracic impedance.

## Energy Choice

Many investigators have referred to a so-called defibrillation energy threshold for converting VF. Davy and coworkers suggest that no unique defibrillation energy threshold exists for the in vivo heart. [45] Experimentally they found that successful defibrillation was related to delivered energy, which follows a typical "dose-response

curve." However, there is evidence for a defibrillation current threshold. [24]

Successful defibrillation is dependent on the simultaneous depolarization of a sizable mass of the myocardium resulting from the passage of current through the heart. For a given thorax, defibrillation device, and defibrillation technique, more current is passed through the heart and, hence, more tissue is depolarized with larger energies. Once sufficient tissue has been depolarized, however, additional current is not desirable and may, in fact, produce additional tissue injury. Kerber and coworkers have suggested that the use of defibrillators that adjust defibrillation energy for the patient's transthoracic impedance (measured by the device just before charging) may be one means to deliver an adequate current to the myocardium while minimizing potential harm. [46] Such devices are not in routine use at present, but they may be helpful in the future for both the identification of high-impedance situations and adjustment of delivered energy. [47]

Several studies have supported the concept that when current needs cannot be predicted nor current delivery measured, a weight-adjusted dosage of energy is preferred for converting VF. Indeed, a dose based on the patient's weight has been found clinically useful for treating children suffering VF. [48] Other prospective human adult studies have questioned the importance of dose strength to conversion of VF. [49] [50] Weaver and coworkers alternated treatment protocols to determine prospectively the merits of 175 J (200 J of stored energy) vs. 320 J (400 J of stored energy) countershocks for defibrillation. [51] On test days, VF patients were shocked initially with one or two 175 J discharges, and all subsequent shocks needed were 320 J. On alternate days, only 320 J shocks were given. The investigators found that 73% (n = 76) of the patients were defibrillated following the first 2 shocks in the low-energy group, whereas 81% (n = 77) of the patients were initially defibrillated in the high-energy group (difference not statistically significant). Asystole occurred in 19% of patients receiving high energy and in 12% receiving low energy. Transient or persistent heart block occurred in 25% of patients shocked with high energy vs. 11% of patients shocked with low energy. Survival until hospital discharge was inversely related to the number of shocks required; no patients who required >8 shocks survived.

Weaver and associates concluded that low-energy (175 J delivered) countershocks were safe, effective, and less cardiotoxic. Obviously, many factors besides discharged energy

play a role in successful defibrillation. Kerber and colleagues have found that defibrillation success rate is a unimodal function of transthoracic current. [52] The maximum defibrillation rate for their patients with a brief duration of VF (<1 minute) occurred at a peak transthoracic current of 38 to 41 amperes (A). Whether a similar optimum transthoracic current is found in patients with prolonged VF remains to be determined. Until energy levels based on measured transthoracic impedance are shown to be predictive of defibrillation success in a variety of settings, most authorities recommend using an initial 200 J energy setting in adults and a 2 J/kg energy setting in children. When 2 sequential defibrillations at this setting are unsuccessful, countershock at 360 J (or maximum output, if needed) is recommended.

## Postdefibrillation Rhythms

Following defibrillation a number of cardiac rhythms may occur. Ultimate prognosis is linked to the rhythm that evolves during the first few minutes following defibrillation. In an evaluation of the prognostic significance of postdefibrillation rhythms in 94 patients, Hoffman and Stevenson [53] noted survival rates of 20-33% in patients who developed a supraventricular rhythm and 8% in those who developed an organized idioventricular rhythm; there were no survivors if defibrillation was unsuccessful or resulted in asystole or an unorganized rhythm. Survival rates may be influenced by the underlying condition or cause of the arrest or by subsequent drug therapy, but ultimate prognosis is dismal indeed if the first few attempts at defibrillation fail to restore a supraventricular rhythm.

## Drug Therapy

The role of correcting hypoxia and electrolyte disorders in the treatment of VF has been discussed previously. The benefit of alpha-agonist catecholamines to enhance aortic diastolic pressure and to improve coronary perfusion has also been described. Further discussion of the role of sodium bicarbonate, adrenergic agents, and antidysrhythmic agents follows. It should be noted that although adjunctive drug therapy is often used or advocated, there are few data supporting better long-term survival rates when any drug therapy regimen is combined with defibrillation. When evaluating any intervention, *it is paramount to focus on survival to discharge and not to be overly concerned with minor changes in initial resuscitation rates that merely provide ultimately unsalvageable patients with transient cardiac activity.* [54]

### Sodium Bicarbonate

The administration of sodium bicarbonate during CPR is controversial, and previous recommendations are currently undergoing reappraisal. [55] Although acidosis can reduce the responsiveness of the myocardium and vascular system to catecholamines, no clinical studies have supported the use of sodium bicarbonate for treatment of VF. Yakaitis and colleagues found that although acidosis did not alter defibrillation success in a canine model of brief VF (75 seconds), prearrest acidosis or hypoxia (in the absence of exogenous epinephrine) was rarely associated with resumption of perfusion. [56] Guerci and coworkers used a prolonged VF (20 minutes of CPR) canine model with epinephrine administration before defibrillation to assess the potential benefit of sodium bicarbonate administration. [57] These investigators failed to demonstrate an advantage from the use of sodium bicarbonate in either defibrillation success or maintenance of perfusion after defibrillation. The influence of acidosis on catecholamines appears to be most prominent at low doses and may not apply to the large amounts of epinephrine used during CPR. Although sodium bicarbonate therapy should be considered for the patient with suspected or proven extreme acidosis, hyperkalemia, or both, it cannot be recommended for routine use as a standard part of the therapy for VF.

There are a number of theoretical reasons to avoid sodium bicarbonate therapy during cardiac arrest. Aside from the fact that it does not specifically facilitate defibrillation and may not be associated with a better final outcome, bicarbonate therapy will shift the

oxyhemoglobin dissociation curve to inhibit oxygen release, induce hyperosmolar and hypernatremic states, produce paradoxical intracellular acidosis through an acceleration of $CO_2$ production, and exacerbate central venous acidosis.

**Adrenergic Agents**

Drugs that stimulate alpha-adrenergic receptors have been advocated during cardiac arrest to increase myocardial and central nervous system blood flow during CPR. These agents may not enhance the ability to defibrillate VF, but they may enhance the development of a perfusing rhythm following defibrillation. [58] It is important to note that alpha agonists are potent vasopressors and that they increase systemic vascular resistance and elevate aortic diastolic pressure, resulting in increased coronary and carotid blood flow. This enhanced perfusion, and not direct beta-adrenergic stimulation, is credited with increased survival rates. [59] In fact, the beta-adrenergic properties of epinephrine and isoproterenol have been postulated to be harmful because these pharmacologic effects increase myocardial oxygen consumption and reduce subendocardial perfusion. When used alone, isoproterenol, a potent beta agonist without alpha effects, does not favorably influence resuscitation rates. Epinephrine has been the vasopressor of choice during CPR, administered in advanced cardiac life support (ACLS) recommended doses of 0.5 to 1.0 mg every 5 minutes. Large doses of epinephrine (0.2 mg/kg), 10 to 15 times higher than currently recommended, may provide better initial resuscitation rates but have not been shown to enhance ultimate survival rates. [40] [60]

A number of pure alpha agonists have been advocated to overcome the theoretical disadvantages of epinephrine. For example, Silvast noted similar resuscitation rates from out-of-hospital cardiac arrest (e.g., from VF, asystole, and pulseless electrical activity [PEA]) when phenylephrine (one or two 1.0-mg boluses) was used in place of standard doses of epinephrine. Methoxamine (10 to 20 mg) has also been used with success rates similar to those of epinephrine. [61] There is concern, however, that although pure alpha agonists favorably augment peripheral vascular resistance, they do so at the expense of cerebral perfusion and myocardial blood flow. [62] [63] The specific role of these agents on defibrillation is unclear. Like high-dose epinephrine, the use of pure alpha agonists in facilitating defibrillation or enhancing long-term survival is not supported by current data. Although adherence to standard ACLS guidelines is logical until the benefits of alternative therapies are clarified, the role of adrenergic adjunctive drug therapy remains controversial.

**Bretylium Tosylate**

Bretylium tosylate has been used to facilitate ventricular defibrillation. Bretylium (Bretylol) has been shown in one animal model to decrease the threshold shock strength required for defibrillation. [64] Other investigators have found no significant effect on the defibrillation threshold. The drug increases the effective refractory period in normal ventricular muscle and Purkinje fibers [65] and enhances electrical uniformity throughout the myocardium, thus tending to terminate conditions supportive of reentrant rhythms. Bretylium raised the threshold for electrically induced VF in a canine CPR model within 10 minutes after administration. [66] Bretylium appears to elevate the VF

threshold by adrenergic neuronal blockade. One canine study has shown an increased potential for PEA with bretylium following defibrillation. [67]

Spontaneous chemical defibrillation has been reported in myocardial infarction patients who were given bretylium by IV drip rather than by bolus perfusion during cardiopulmonary resuscitation. [68] A retrospective study has suggested that the early use of bretylium tosylate during VF sudden death may enhance survival. [69]

For VF, 5 mg/kg of bretylium is given by rapid intravenous bolus, and cardiac massage is performed for 1 to 2 minutes to permit circulation of the drug before defibrillation attempts. If after 2 minutes the initial therapy is unsuccessful, a repeat dose of 10 mg/kg is given, and defibrillatory efforts are continued. Successful defibrillation may be followed by hypotension and the physician must be prepared to administer volume or pressor agents to support the blood pressure.

**Lidocaine**

Lidocaine has long been used to facilitate defibrillation. The rationale for use of lidocaine in VF is primarily based on anecdotal experience. Two studies of nonischemic canines have demonstrated that lidocaine increases the energy required for electrical defibrillation. [62] [70] Kerber and coworkers suggest that the elevation of the defibrillation threshold noted by others may be a function of the anesthetic used in the animal model and may not be a clinical factor. [71]

Lidocaine has complex effects on membrane responsiveness. Little change in conduction velocity occurs in normal myocardium, whereas conduction in ischemic tissue is decreased following lidocaine administration. Lidocaine increases uniformity of the action-potential duration and refractory period throughout the ventricles and can terminate ventricular reentrant rhythms. Lidocaine also raises the threshold for electrically induced VF in a canine CPR model within 5 minutes after administration, although the antifibrillatory effect is not sustained with a single bolus dose.

One retrospective study of out-of-hospital VF arrests documented a small but statistically insignificant improvement in both defibrillation rate and survival when patients refractory to conventional therapy for VF were given lidocaine during the course of their resuscitation. Unfortunately, strict drug and therapy protocols were not followed, and variations in treatment may have masked a beneficial effect of lidocaine administration. Comparison studies of out-of-hospital lidocaine and bretylium use for refractory VF showed similar conversion and survival rates in the 2 drug treatment groups. [72] [73] Although the role of lidocaine and bretylium in the facilitation of defibrillation remains to be defined more clearly, clinical experience suggests that both drugs have value in aiding defibrillation. Because one drug may be more beneficial than the other in a given patient, broader generalizations cannot be made. Certainly both drugs are useful for preventing degeneration of a supraventricular rhythm once effective defibrillation occurs, and one or the other has been empirically advocated to be used postdefibrillation. Lidocaine is initially given as a bolus of 1 mg/kg to the VF sudden death victim who is refractory to conventional defibrillatory efforts. A second 1 mg/kg bolus can be given in 10 to 15 minutes. It is important to remember to reduce the

maintenance (not the initial loading dose) dose of lidocaine in patients who have a history of hepatic and/or left ventricular dysfunction.

**Other Drug Therapies**

As the search continues for useful pharmacologic adjuncts to facilitate defibrillation or to serve as alternatives to countershock, anecdotal reports of success with newer antiarrhythmics have appeared in the literature. No medications are of proven value, but amiodarone and magnesium sulfate have been evaluated in limited clinical trails. Amiodarone is a class III antiarrhythmic that has recently become available in an IV form to treat life-threatening ventricular arrhythmias and VF. Kowey and coworkers suggest that it may be as effective as bretylium [74] in treating recurrent VT and VF, and others have suggested a possible role for this agent in cardiac arrest. [75] [76] Hypotension and bradycardia are common side effects, and the role of amiodarone in facilitating defibrillation remains unclear.

Miller and colleagues have proposed a possible benefit for one to two 5-g bolus injections of IV magnesium sulfate in refractory cardiac arrest. [77] Magnesium has been touted as an antiarrhythmic for years, but its role in cardiac arrest or for enhancing the success of defibrillation is still unclear.

# COMPLICATIONS

The major complications of direct current defibrillation are (1) injury to skin and other soft tissue, (2) myocardial injury, and (3) cardiac dysrhythmias.

## Soft Tissue Injury

When skin contact is firm and a conductive material is applied between the paddles and the chest wall, contact burns are usually minimal. Nevertheless, repeated countershocks can produce erythema of the skin resembling superficial first-degree burns. The presence of liquids (e.g., blood, IV solutions, vomitus, urine, excessive sweat) may permit the passage of current across the trunk. This electrical arcing will produce thermal burns (third-degree at times) and ineffective defibrillation. Hummel and coworkers have shown that repeated defibrillations using certain high-impedance conducting gels are associated with sparking and represent a fire hazard in an oxygen-enriched environment and an explosion hazard in the presence of nitroglycerin ointment or patches. [32] Intrathoracic injuries (extrinsic to the heart) are likely to occur but are difficult to document during the post-resuscitative period and to separate from cardiac injury (e.g., pulmonary edema). [78]

## Myocardial Injury

The direct application of electrical countershock to the heart has long been known to produce epicardial and myocardial injury. Studies have demonstrated that closed-chest defibrillation is capable of producing cardiac injury. Electrical current rather than direct thermal injury produces injury. [79] [80] Multiple countershocks have been shown to

produce ST segment elevation in animals and gradual cell necrosis (over days) with subsequent fibrosis. [81] The lesions are primarily subepicardial at the points of current entrance and exit. Animals receiving less than twice the defibrillation threshold value do not develop significant necrosis. The degree of cardiac injury correlates with increasing energy exposure.

Jones and coworkers, using an in vitro model, have created transient sarcolemmal microlesions during high-intensity electric field stimulation identical to that of defibrillation. [82] These lesions result in a "short-circuit" depolarization of the cells by loss of the normal sodium-potassium gradient across the sarcolemma. When the lesions are limited, the cell can recover after about 60 seconds. However, with extensive lesions, shock-induced cytosolic calcium overload can occur and can result in postshock contracture.

The ability to document anatomic injury to the human heart is limited by the natural reparative process, concurrent ischemic processes producing similar microscopic changes, and the fact that several days are needed for the injuries to manifest themselves. Cardiac isoenzyme (CK MB) levels were shown to rise in patients undergoing elective cardioversion only if the cumulative delivered energy was >475 J. [83] Therefore, standard defibrillation should not generally interfere with the enzymatic diagnosis of myocardial infarction when defibrillation attempts are not excessive and given that fractionated isoenzymes are measured.

Animal studies have shown that ST segment elevation and pathologic changes are increased with more rapidly delivered discharges (1 or 3 seconds vs 15 seconds between discharges). [80] Furthermore, the cumulative energy correlates with myocardial injury for a given dosing schedule. [84]

**Cardiac Dysrhythmias**

The rhythm that one obtains following defibrillation may be ventricular, supraventricular, or flatline (asystole). Laboratory studies have suggested a correlation between the severity of postdefibrillation dysrhythmias and the degree of myocardial damage produced. [85] Reducing the peak current delivered to the heart by changing the waveform of the discharge was associated with fewer dysrhythmias. Weaver and colleagues noted that asystole and transient heart block occurred more commonly with a higher discharge energy in out-of-hospital VF patients. [51] Gueze and Koster also found that postdefibrillation dysrhythmias were more common following prolonged VF and higher-energy-level countershocks. [86]

**Injuries to Health Care Providers**

Any electrical device, including an improperly grounded or poorly insulated defibrillator, can cause injury to the device operator or to others in attendance. Other participants in a resuscitation who touch the patient or the stretcher can also serve as a ground for the defibrillator charge and can sustain electrical injury. Gibbs and coworkers estimate that the rate of paramedic injury during patient defibrillation is 1 per 1700 defibrillatory shocks. [87] They found only 1 paramedic who required hospital admission for therapy

and monitoring of countershock ectopy. Improper use of the device for cranial countershock has been reported to produce short-term memory loss. [88] However, it must be borne in mind that such inappropriate use of these devices could easily cause much more serious injury. Fires resulting from defibrillator sparks in the presence of nitroglycerin patches or ointment, flammable gases, or an oxygen-enriched environment can also injure health care personnel as well as the patient.

## SPECIAL TOPICS IN DEFIBRILLATION

Management of patients with an automatic implantable cardioverter defibrillator (AICD) is discussed in Chapter 12 .

### Automatic External Defibrillators

Automatic external defibrillators have been developed with sophisticated digital software to take advantage of algorithms that can reliably recognize VF. [18] [33] These small, portable, battery-operated devices permit nonparamedic out-of-hospital providers to defibrillate cardiac arrest patients without human rhythm interpretation. [89]

Because the devices monitor and defibrillate through the same skin electrodes, the user must simply place the adhesive electrode pads at the standard locations (right upper sternal and left anterior axillary line below the nipple), turn on the device, and heed warnings to avoid contact with the patient or with any surface in contact with the patient. Detailed discussion of the operation of these devices is beyond the scope of this chapter. The reader is referred to the instruction manual for the individual devices.

### Thump Defibrillation

The precordial thump consists of a firm, sharp, quickly delivered blow applied to the midsternum with a closed fist from a height of about 30 to 38 cm above the anterior chest wall of a pulseless patient. It is believed that such a blow is capable of generating approximately 5 J of energy.

Although such a precordial thump may be of value in witnessed VT/VF arrests, Miller and associates noted no VF conversion in 23 out-of-hospital VF patients. [90] Despite the fact that the rhythm was improved in 3 of 27 VT patients after a precordial thump, the rhythm deteriorated in 12 of the 27 and did not change in the other 12 patients. Caldwell and colleagues reported the results of a precordial thump in 68 cases of VT and 248 cases of VF. [91] They reported 26 favorable cardioversions, including in 5 patients in VF. Because successful internal defibrillation can occur with as little as 1 J following cardiopulmonary bypass, a vigorous precordial thump may create sufficient current flow to defibrillate the heart when the duration of arrest has been brief (i.e., witnessed cardiac arrest).

### Refractory Ventricular Fibrillation

The major determinants of successful defibrillation are the early use of countershock,

adequate oxygenation, lack of serious metabolic derangements, and general health of the patient. Hargarten and colleagues note that few out-of-hospital patients who remain in VF after the fifth shock are

subsequently successfully converted to an effectively perfusing rhythm. [92] Kerber and colleagues note that patients who never defibrillated despite multiple shocks had a prolonged duration of CPR preceding the first shock (21 ± 14 minutes) and systemic hypoxia and acidosis. [93] These conditions were noted to occur in their patients whose initial cardiac arrest rhythm was asystole, severe bradycardia, or PEA.

A number of clinical conditions may result in the inability to convert VF initially or in the recurrence of VF following the first successful defibrillation. Patients with severe hypothermia are often refractory to initial defibrillation and generally require rapid core rewarming to be successfully defibrillated. Severe bradycardia will predispose to lethal escape rhythms, and emergency cardiac pacing may be required. Severe electrolyte disturbances, such as hypokalemia, hypomagnesemia, and hypocalcemia may precipitate refractory VF and be amenable only to the appropriate, rapid correction of the deficient electrolyte. Such situations may occur especially in fad dieters or abusers of diuretic medications. Uncorrected acidosis or hypoxia, such as that seen with drowning, may be the cause of persistent VF. In addition, excessive adrenergic stimulation, such as that seen with cocaine or amphetamine overdose, may require the use of a propranolol infusion before successful defibrillation. As a final note, following defibrillation, all patients should be treated with prophylactic lidocaine (or other appropriate antidysrhythmic therapy) to minimize the chance of recurrent VF.

## Conclusion

Electrical defibrillation is the preferred treatment for VF-related sudden death. Treatment should be initiated as early as possible with strict attention to selection of the proper energy dose as well as to minimization of transthoracic impedance. Although repetitive high-energy shocks may be associated with myocardial injury, this factor has not been found to be a clinical problem at currently recommended energy levels.

# Chapter 13 - Emergency Transvenous Cardiac Pacing

**Georges C. Benjamin**

The purpose of cardiac pacing is to restore or ensure effective cardiac depolarization. Several approaches to pacing exist, including transvenous, transcutaneous (see Chapter 14) , transthoracic, epicardial, endocardial, and esophageal. The transvenous method of endocardial pacing is commonly used and is both safe and effective. In skilled hands, the semifloating transvenous catheter is successfully placed under electrocardiographic guidance in 80% of patients. [1] The technique can be performed in <20 minutes in 72% of patients and in <5 minutes in 30% of patients. As with other medical procedures, it should not be performed without a thorough understanding of its indications, contraindications, and complications. [2]

## BACKGROUND

The ability of muscle to be artificially depolarized was recognized as early as the 18th century. Over the succeeding years, several scattered experiments were reported, and in 1951, Callaghan and Bigelow first used the transvenous approach to stimulate the asystolic heart in hypothermic dogs. [3] Zoll demonstrated the first clinical use of cardiac pacing in humans in 1952. [4] He reported the successful use of an external transcutaneous electrical stimulator in 2 patients with ventricular standstill.

Furman and Robinson demonstrated the transvenous endocardial approach in humans in 1958. [5] They treated 2 patients with complete heart block and Stokes-Adams seizures, reconfirming that low-voltage pacing could completely control myocardial depolarization. The catheter remained in the second patient for 96 days without complication. Other early clinical studies have proved that transvenous pacing is a valuable procedure in medicine. [6] [7] [8] Fluoroscopic guidance was used for placement of the pacing catheter in all of these studies.

In 1964 Vogel and colleagues demonstrated the use of a flexible catheter passed without fluoroscopic guidance for intracardiac electrocardiography. [9] One year later, this technique was used by Kimball and Killip to insert endocardial pacemakers at the bedside. [10] They noted technical difficulties, including intermittent capture, difficulty passing the catheter, and catheter knotting, in 20% of their patients. During the same year, Harris and associates confirmed the ease and speed with which this procedure could be accomplished. [11]

Before 1965 all intracardiac pacing was done asynchronously, which meant that the pacing catheter could cause electrical stimulation during any phase of the cardiac cycle. Asynchronous pacing frequently resulted in the pacemaker firing during the vulnerable period of an intrinsic depolarization; this occasionally caused ventricular tachycardia or fibrillation. In 1967 a *demand pacemaker generator* that sensed intrinsic depolarizations and inhibited the pacemaker for a predetermined period of time was used successfully by Zuckerman and associates in 6 patients. [12]

A further improvement in the pacing catheter was made by Rosenberg and colleagues when they introduced the Elecath semifloating pacing wire. [1] The Elecath was stiffer than the Flexon steel wire electrode. Rosenberg and coworkers achieved pacing in 72% of patients, with an average procedure time of 18 minutes. They also noted that 30% of their patients were paced in 5 minutes or less. [1] The technique of heart catheterization using a flow-directed balloon-tipped catheter was introduced by Swan and Ganz in 1970. [13] This concept was used successfully by Schnitzler and coworkers for the placement of a right ventricular pacemaker in 15 of 17 patients. [14]

In 1981 Lang and colleagues compared the bedside use of the flow-directed balloon-tipped catheter with insertion of a semirigid electrode catheter in 111 perfusing patients. [15] These researchers found a significantly shorter insertion time (6 minutes and 45 seconds compared with 13 minutes and 30 seconds), a lower incidence of serious arrhythmias (1.5% compared with 20.4%), and a lower incidence of catheter displacement (13.4% compared with 32%) with the balloon-tipped catheter. They concluded that the balloon-tipped catheter was the method of choice for temporary transvenous pacing (Table 13-1) .

Kruger and associates reviewed retrospectively the experience of general internists with transvenous pacemaker placement under electrocardiogram (ECG) guidance. [16] A 4% risk of complications and a 14% incidence of electrode malfunction were reported, and these percentages were noted to be similar to those reported by university cardiologists. They concluded that pacemaker placement by primary care physicians was safe and effective when done under ECG guidance without fluoroscopy. Temporary transvenous dual chamber pacing currently is readily available and is generally safe and effective. [17]

## INDICATIONS

The purpose of cardiac pacing is to resume effective cardiac depolarization. In most cases the specific indications for cardiac pacing are clear; however, some controversial areas remain. The decision to pace on an emergent basis requires knowledge of the presence or absence of hemodynamic compromise, the etiology of the rhythm disturbance, the status of the atrioventricular (AV) conduction system, and the type of dysrhythmia. In general, the indications can be grouped into those that cause either tachycardias or bradycardias (Table 13-2) . Transcutaneous cardiac pacing has become the mainstay of emergent cardiac pacing and is often used pending placement of the transvenous catheter or to determine whether potentially terminal bradyasystolic rhythms will respond to pacing.

| | | |
|---|---|---|
| **TABLE 13-1** -- History of Transvenous Pacing | | |
| **Date** | **Investigator** | **Event** |

| | | |
|---|---|---|
| 1700 | Early investigators | First restimulation studies |
| 1951 | Callaghan & Bigelow | First transvenous approach in dogs |
| 1952 | Zoll | Transcutaneous cardiac stimulator |
| 1958 | Falkmann & Walkins | Implanted pacing wires after surgery |
| 1959 | Furman & Robinson | First transvenous pacer in humans |
| 1964 | Vogel et al | Flexible electrocardiographic catheter without fluoroscopy |
| 1965 | Kimball & Killip | First bedside transvenous pacing |
| 1966 | Goetz et al | Demand pacemaker developed |
| 1967 | Zuckerman et al | Use of demand pacemaker clinically |
| 1969 | Rosenberg et al | Semifloating pacing catheter |
| 1973 | Schnitzler et al | Balloon-tipped pacers |

**TABLE 13-2** -- Indications for Cardiac Pacing

**Bradycardias**

Without myocardial infarction

  Symptomatic sinus node dysfunction (sinus arrest, tachybrady [sick sinus] syndrome, sinus bradycardia)

  Second- and third-degree heart block

  Atrial fibrillation with slow ventricular response

With myocardial infarction

  Symptomatic sinus node dysfunction

  Mobitz II second- and third-degree heart block

  New left bundle-branch block (LBBB), right bundle-branch block (RBBB) with left axis deviation, bifascicular block, or alternating bundle-branch block

Trauma patient with hypotension and unresponsive bradycardia

Prophylaxis--cardiac catheterization, after open heart surgery, threatened bradycardia during drug trials for tachydysrhythmias

| |
|---|
| Malfunction of implanted pacemaker |
| Asystolic arrest patient (not clear) |
| **Tachycardias** |
| Supraventricular dysrhythmias |
| Ventricular dysrhythmias |
| Prophylaxis--cardiac catheterization, after open heart surgery |

## Bradycardias

### Sinus Node Dysfunction

In a review of 200 initial pacemaker implants at Montefiore Hospital during 1975, 36.5% were used for sinus node dysfunction; 11.3%, for sinus arrest; 20.2%, for tachybrady (sick sinus) syndrome; and 5%, for sinus bradycardia. [18] Patients without myocardial infarction who present with symptomatic sinus node dysfunction should be promptly paced if medical therapy fails. Escher and Furman note that pacing is indicated until the etiology of the dysrhythmia is clarified and stability is ensured. [19]

In the asymptomatic patient, a more intensive cardiac evaluation is required to decide whether pacing will be beneficial. This evaluation frequently includes 24-hour Holter monitoring, noting sinus node recovery times, and coronary care unit monitoring.

Sinus bradycardia occurs in an average of 17% of patients with acute myocardial infarction. [20] Sinus bradycardia occurs more frequently in inferior than in anterior infarction and has a relatively good prognosis when accompanied by a hemodynamically tolerable escape rhythm. However, sinus bradycardia is not a benign rhythm in this situation; it has a mortality rate of 2% with inferior infarction and 9% with anterior infarction. [21] Several mechanisms have been suggested to explain sinus node dysfunction with infarction. Among these, ischemia of the node or its neurologic controls [21] and reflex slowing secondary to pain play dominant roles. [22] Sinus node dysfunction

frequently responds to medical therapy but requires prompt pacing if this fails.

**Asystolic Arrest**

Transvenous pacing in the asystolic or bradyasystolic patient has little value. In 1 study of 13 patients who had suffered cardiac arrest, capture of the myocardium was noted in 4 patients, but there were no survivors. [23] Transvenous pacing alone may also not be effective in postcountershock pulseless

bradyarrhythmias. [24] This failure of pacing has also been demonstrated with transcutaneous pacemakers, suggesting that failure of effective pacing is primarily related to the state of the myocardial tissue. [23] Other causes of failure to pace include catheter malposition and dislodgment of the pacing wire during closed-chest massage. [23] Cardiac pacing may be used as a "last ditch" effort in bradyasystolic or asystolic patients but is rarely successful and is not considered standard practice. Early pacing is essential when done for this purpose, if success is to be achieved. [25] A more complete discussion of pacing in bradyasystolic cardiac arrest appears in Chapter 14 .

**Atrioventricular Block**

Atrioventricular block is the classic indication for pacemaker therapy. In symptomatic patients without myocardial infarction and in the asymptomatic patient with a ventricular rate below 40, pacemaker therapy is indicated. [26]

In patients with acute myocardial infarction, 15 to 19% progress to heart block: approximately 8% develop first-degree block, 5% develop second-degree block, and 6% develop third-degree block. [27] First-degree block progresses to second- or third-degree block 33% of the time, and second-degree block progresses to third-degree block about one third of the time. [28]

Atrioventricular block occurring during anterior infarction is believed to occur because of diffuse ischemia to the septum and conduction tissue infranodally. *These patients tend to progress to high-degree block without warning and are often prophylactically paced temporarily, even without hemodynamic compromise.*

During inferior infarction, early septal ischemia is the exception, and block develops serially from first degree to Mobitz type I second degree, then to third degree. These conduction abnormalities frequently result in hemodynamically tolerable escape rhythms because of sparing of the bundle branches. The hemodynamically unstable patient who is unresponsive to medical therapy should be paced promptly. When the stable patient should be paced is unclear, but placing a transcutaneous pacer is one option that can be tried before placing a transvenous pacing catheter.

One study in which the indications for temporary and permanent pacemaker insertion were reviewed in 432 patients with myocardial infarction concluded that patients with second- or third-degree atrioventricular block should be paced, because a higher incidence of sudden death or recurrent high-degree block over the following year was

found in patients who were not continuously paced. [29]

**Trauma**

In the patient with nonpenetrating chest trauma, several rhythm and conduction disturbances have been documented. In these patients, traumatic injury to the specialized conduction system may predispose the patient to life-threatening dysrhythmias and blocks that can be treated by cardiac pacing. [30]

Hypovolemia and hypotension can cause ischemia of conduction tissue and cardiac dysfunction. [31] Continued marked bradyarrhythmias after vigorous volume replacement may respond to cardiac pacing in patients with such trauma. [32]

## Bundle-Branch Block and Ischemia

Bundle-branch block occurring in acute myocardial infarction is associated with a higher mortality rate and a greater incidence of third-degree heart block than uncomplicated infarction. Atkins and associates noted that 18% of patients had bundle-branch block with myocardial infarction. [33] Of these patients, complete heart block developed in 43% who had right bundle-branch block and left axis deviation, in 17% who had left bundle-branch block, in 19% who had left anterior hemiblock, and in 6% who had no conduction block. The investigators concluded that right bundle-branch block with left axis deviation should be prophylactically paced.

A study by Hindman and colleagues confirmed the natural history of bundle-branch block during myocardial infarction. [34] In their study, the presence or absence of first-degree atrioventricular block, the type of bundle-branch block, and the age of the block (new versus old) were used to determine the relative risk of progression to type II second-degree or third-degree block (Table 13-3) (Table Not Available) .

Because of the increased risk, most physicians would pace the following conduction blocks: *new-onset* left bundle-branch block, right bundle-branch block with left axis deviation or other bifascicular block, and alternating bundle-branch block. [34] One authority recommends prophylactic pacing for all new bundle branch blocks when myocardial infarction is evident. [35]

Whether to place a transvenous pacemaker prophylactically in patients with left bundle-branch block before insertion of a flow-directed pulmonary artery catheter remains controversial. Some researchers strongly advocate this procedure because of the risk of transient right bundle-branch block and life-threatening complete heart block. [36] One study notes that this risk is low in patients with prior left bundle-branch block but continues to recommend temporary catheter placement for all cases of *new* left bundle-branch block. [37] One solution to this problem is to place a transcutaneous pacemaker before catheterization as an emergency measure should heart block develop. In these cases, a temporary transvenous pacemaker can be placed in a semi-elective manner when needed. [38]

**Tachycardias**

Hemodynamically compromising tachycardias are usually treated by medical means or electrical cardioversion (see Chapter 10) . Since 1980, there has been an increasing interest in pacing therapy for symptomatic tachycardias. Supraventricular dysrhythmias, with the exception of atrial fibrillation, respond well to atrial pacing. By "overdrive" pacing the atria at rates 10 to 20 beats/min faster than the underlying rhythm, the atria become entrained, and when the rate is slowed, the rhythm frequently returns to normal sinus. A similar procedure is done for ventricular dysrhythmias. [39] Overdrive pacing is especially useful for recurrent prolonged Q-T interval arrhythmias such as those seen with quinidine toxicity or torsades de pointes. [40] Transvenous pacing also is useful in patients with digitalis-induced dysrhythmias in whom direct current (DC) cardioversion may be dangerous or in patients in whom there is further concern about myocardial depression with drugs.

## CONTRAINDICATIONS

There are no absolute contraindications to transvenous cardiac pacing; however, the severely hypothermic bradycardic patient can often be managed without pacing. Severe hypothermia will occasionally result in ventricular fibrillation when pacing is attempted. Because ventricular fibrillation under these conditions is difficult to defibrillate, caution is advised when considering pacing the severely hypothermic and bradycardic patient. Rapid warming is often recommended first, followed by pacing if the patient's condition does not improve.

## EQUIPMENT

Several items are required to insert a transvenous pacemaker adequately. Like most special procedures, a prearranged tray is convenient. The usual components required to insert a transvenous cardiac pacemaker are listed in Table 13-4 .

**Pacing Generator**

Many different pacing generators are available, but in general, they all have the same basic features. The on/off switch frequently will have a locking feature to prevent the generator from inadvertently being switched off. An amperage knob allows the operator to control the amount of electrical current delivered to the myocardium, usually 0.1 to 20 mA. The pacing control mode is the gain control for the sensing

---

**TABLE 13-4** -- Equipment

| **Pacemaker Tray** |
| --- |
| 10-mL syringe |
| 1% lidocaine |
| Alcohol wipes |
| Povidone-iodine (Betadine) |
| Several gauze pads |
| 4 sterile drapes |
| No. 11 scalpel blade |
| 0.9 normal saline--2 ampules |
| Sterile gloves |
| Needle holder |
| Two 22-ga needles |

| |
|---|
| Scissors (suture) |
| Two 4-0 silk sutures on needles |
| Sterile basin |
| **Electrical Hardware** |
| Insulated connecting wire with alligator clamps at each end (or a male-to-male adapter) |
| Spare 9-V battery |
| Medtronic pacing unit no. 5375 |
| 3 Fr Balectrode Pacing Kit (catalog no. 11--KBE1) |
| 12-lead electrocardiographic machine (well grounded) |

function of the generator. By increasing the sensitivity, one can convert the unit from a fixed rate (asynchronous mode) to a demand (synchronous mode) pacemaker. In the fixed rate mode, the unit fires despite the underlying intrinsic rhythm; the unit does not sense any intrinsic electrical activity. In the full-demand mode, however, the pacemaker senses the underlying ventricular depolarizations, and the unit does not fire as long as the patient's ventricular rate is equal to or faster than the set rate of the pacing generator. A sensing indicator meter and rate control knob are also present. An example of a pacing generator is shown in .

**Pacing Electrodes**

Several sizes and brands of pacing catheters are available. In general, most range from 3 Fr to 5 Fr in size and are approximately 100 cm in length. Along the catheter surface are lines that are marked at approximately 10-cm intervals; these can be used to estimate catheter position during insertion. Two basic types of pacing catheters are currently in use: the flexible semifloating or floating catheter, and the rigid fixed-position catheter. [40]

The flexible catheters are more advantageous than the rigid catheter in their ability to be inserted in low flow states, as well as in their decreased tendency to perforate the ventricle. For emergency pacing, the 4 Fr semifloating bipolar electrode with or without the balloon tip is used most frequently (Fig. 13-2) . The balloon holds approximately 1.5 mL of air, and some of them have a locking lever to secure balloon expansion. Before insertion, the balloon is checked for air leakage by inflating it and immersing it in sterile water. The presence of an air leak is noted by a stream of bubbles arising at the surface of the water. An inflated balloon helps the catheter "float" into the heart in low flow states but is obviously not advantageous in the cardiac arrest situation.

For all practical purposes, temporary transvenous pacing is accomplished with a bipolar pacing catheter. The terms *unipolar* and *bipolar* refer to the number of electrodes in contact with that portion of the heart that is to be stimulated. All pacemaker systems must have both a positive (anode) and a negative (cathode) electrode; hence, *all stimulation is bipolar.* In the typical bipolar catheter used for temporary transvenous pacing, the cathode (stimulating electrode) is at the tip of the pacing catheter. The anode is located 1 to 2 cm proximal to the tip, and the two electrodes may be separated by a balloon or an insulated wire. The electrodes are usually platinum rings that encircle the pacing catheter. When properly positioned, both electrodes will be within the right ventricle so that a field of electrical excitation is set up between the electrodes. With the bipolar catheter, the cathode does not need to be in direct contact with the endocardium for pacing to occur, although it is preferable to have direct contact.

A unipolar system is also effective but is infrequently used for temporary transvenous pacing. In a unipolar system, the cathode is at the tip of the pacing catheter, and the anode is located (1) in the pacing generator itself, (2) more proximal on the catheter (outside the ventricle), or (3) underneath the skin on the patient's chest. The bipolar system may be converted to a unipolar system by simply disconnecting the positive proximal connection of the bipolar catheter from the pacing generator and running a new wire from the positive (pacing generator) terminal to the patient's chest wall. Such a conversion may be required in the unlikely event of failure of one lead of the bipolar system.

Theoretically, the field of electrical stimulation of a pacing catheter is equal to the distance between the electrodes. If the field of excitation is not close enough to the myocardium, depolarization will not occur. When a catheter is passed blindly in an emergency, it seems advantageous to ensure the best chance of capture by separating the electrodes by more than the standard 1 to 2 cm. A pacing catheter that uses this configuration (Davison pacing lead, Electro-Catheter Corporation) is a hybrid of the standard bipolar and unipolar catheters. This catheter has the cathode at the tip, but the

anode is situated 19 cm proximal to the tip. This configuration allows pacing with a very wide field of excitation. Pacing has been reported to occur with this catheter when the catheter is placed anywhere within the thoracic venous system. The catheter is a hybrid because both electrodes are present on the same catheter (bipolar), but both electrodes will not be positioned in the same cardiac chamber (unipolar).

### Electrocardiographic Machine

An ECG can be used to record the heart's inherent electrical activity during pacer insertion and to aid in localization of the catheter tip without fluoroscopy. The ECG machine must be well grounded to prevent leakage of alternating current, which can cause ventricular fibrillation. Such leakage should be suspected if interference of 50 to 60 cycles per second (Hz) is noted on the ECG.

The ECG machine should be placed in such a manner as to allow easy visibility of the rhythm during insertion. One method is to place the machine on the same side of the patient as the operator at the level of the midthorax (Fig. 13-3) . Note that the operator stands at the head of the patient during internal jugular or subclavian vein passage of the catheter and at the midabdomen for femoral or brachiocephalic vein insertion.

### Introducer Sheath

An introducer set or sheath is required for venous access (see Chapter 24) . Some pacing catheters are prepackaged with the appropriate equipment, whereas others require a separate set. The introducer set is used to enhance passage of the pacing catheter through the skin, subcutaneous tissue, and vessel wall. To allow passage of the pacing catheter, the sheath must be one size larger than the pacing catheter. A makeshift sheath can be made with an appropriate-sized intravenous (IV) catheter. For the 3 Fr balloon-tipped catheter, a 14-ga, 1.5- to 2-in. IV catheter is suitable. The 4 Fr balloon-tipped catheter will also fit through a 14-ga catheter or needle.

A balloon-directed pulmonary artery catheter (Paceport pacing system, American Edwards Laboratories, American Hospital Supply Corporation) has been developed that has a separate lumen that allows the passage of a transvenous pacing catheter. [42] This catheter is 7.5 Fr and has an opening 19 cm from the catheter tip that allows passage of the 2.4 Fr pacing wire. This stainless steel wire is Teflon coated for easy passage and has a flexible tip. Combination pulmonary artery or pacemaker catheters are also available but are not widely used in the emergency setting.

Overall, the key to success with this procedure is preparation. It is imperative that one examine all the components of the tray before starting the procedure and ensure that all wires, sheaths, dilators, and syringes fit as expected.

## PROCEDURE

### Patient Preparation

Patient instruction is an extremely important aspect of any procedure. Frequently there is not enough time to give patients a detailed explanation. Nonetheless, sufficient information should be provided so that the patient feels at ease. Patients should be assured that they will feel no discomfort after the venipuncture site has been anesthetized and that they will feel better when the catheter is in place and is functional. Continued reassurance is required during the procedure, because patients are usually facing away from the operator; because their faces are often covered, they may be unsure of what is occurring.

All operators should wear surgical masks, caps, gloves, and gowns to decrease the risk of infection before catheter placement. This aseptic precaution should also be explained to the patient.

**Site Selection**

The four venous channels that provide an easy access to the right ventricle are the brachial, subclavian, femoral, and internal jugular veins (Table 13-5) . The route selected is often one of personal or institutional preference. The right internal jugular and the left subclavian veins have the straightest anatomic pathway to the right ventricle and are generally preferred for temporary transvenous pacing. In some centers a particular site is preferred for permanent transvenous pacemaker placement, and, if possible, this site should be avoided for temporary placement.

The subclavian vein can be accessed by both an infraclavicular and a supraclavicular approach; the infraclavicular approach is most commonly reported for all temporary transvenous pacemaker insertions. This route is preferred because of its easy accessibility, close proximity to the heart, and ease in catheter maintenance and stability. The supraclavicular approach has been described in the literature for several years and has gained popularity among some physicians. [43] The left subclavian vein is preferred because of the less acute angle traversed when compared with the right-sided approach.

Some physicians believe the internal jugular approach is as easy as and safer than subclavian catheterization. [44] The right internal jugular vein is preferred because of the direct line to the superior vena cava. Problems with this approach include dislodgment of the pacemaker with movement of the head, carotid artery puncture, and thrombophlebitis (see Chapter 24) .

During cardiopulmonary resuscitation, the use of the right internal jugular vein and the left subclavian veins for pacemaker insertion have been demonstrated to result in the highest rates of proper placement in the right ventricle. [45] The right internal jugular vein is the more direct route of the two and may be the most appropriate site. Because of the extremely low flow state during cardiopulmonary resuscitation, a larger (5 Fr), semirigid catheter may be a more appropriate choice than the 3 to 4 Fr catheters commonly used.

Femoral veins, like neck veins, are reusable and easily catheterized. Problems include

easy dislodgment, infection, and increased risk of thrombophlebitis. [46] [47]

Brachial vein catheterization is easy to perform but results in a high incidence of infection and vessel thrombosis. [48] In addition, the catheter is easily dislodged with arm motion. This approach is seldom used in the emergency setting.

**Skin Preparation and Venous Access**

The skin over the venipuncture site is cleaned twice with an antiseptic solution such as povidone-iodine and isopropyl alcohol. A wide area is prepared because of the tendency for guide wires and catheters to spring from the hands of the unsuspecting operator. Preparation of the skin is shown in Chapter 24 . Similarly, wide draping is carried out in the standard manner to maintain a sterile field and to allow clear visibility of the venipuncture site.

The infraclavicular approach is used in this chapter to illustrate venous access, although the mechanics are generally the same for other vascular approaches. The reader is referred to Chapter 24 for the specific techniques of venous access.

| | **TABLE 13-5** -- Advantages and Disadvantages of Pacemaker Placement Sites | |
|---|---|---|
| **Venous Channels** | **Advantages** | **Disadvantages** |
| Brachial | Very safe route Vessel easily accessible, either by cutdown or percutaneous approach | Often requires cutdown Easily displaced and poor patient mobility Not reusable if cutdown technique is performed Catheter is more difficult to advance than with central or larger vessels |

| Subclavian | Direct access to right heart (especially via leftsubclavian) Rapid insertion time Reusable Good patient mobility | Pneumothorax and other intrathoracic trauma are possible |
|---|---|---|
| Femoral | Direct access to right heart Rapid insertion time Reusable | Increased incidence of thrombophlebitis Can be dislodged by leg movement and poor patient mobility Infection |
| Internal jugular | Direct access to right heart (especially via right internal jugular) Rapid insertion time Reusable | Possible carotid artery puncture Dislodgment with movement of the head Thrombophlebitis |

Occasionally a patient who already has a central venous line in place requires the emergent placement of a pacing catheter. An existing central venous pressure (CVP) line can be used to place the pacing catheter if the catheter lumen is large enough to accept a guide wire. The CVP line should be withdrawn 1 to 2 in. to expose an area of sterile tubing. The tubing is transected through a sterile area while being held firmly at the skin level. A guide wire can then be passed through the tubing, and the tubing can be withdrawn, leaving only the wire in the vein. The guide wire and the tubing should never be released, because embolization may result. An introducer unit can then be passed over the guide wire, as is done in the Seldinger technique (see Chapter 21) , and the pacing catheter can be placed (Fig. 13-4) .

**Pacemaker Placement**

**Electrocardiographic Guidance**

The patient should be connected to the limb leads of an ECG machine, and the indicator should be turned to record the chest (V) lead.

With newer ECG machines, the pacemaker may be attached to any of the V leads (usually $V_1$ or $V_5$ ) that are displayed during rhythm monitoring. As the tracing on the ECG is slightly delayed with the newer devices, advancement of the catheter after initial

insertion must be carefully evaluated.

The pacing wire should be inserted about 10 to 12 cm into the selected vein. The *distal terminal* of the pacing catheter (the cathode or lead marked negative, "-") must be connected to the V lead of the ECG machine by a male-to-male connector or by an insulated wire with an alligator clip on each end (Fig. 13-5) . The pacing catheter is thus an exploring electrode that creates a unipolar electrode for intracardiac ECG recording. The ECG recorded from the electrode tip *localizes the position of the tip of the pacing electrode*. If a balloon-tipped catheter is used, the balloon is inflated with air *after* the catheter enters the superior vena cava.

The pacing catheter should be advanced both quickly and smoothly. The V lead should be monitored, and the P wave and QRS complex should be observed to ascertain the location of the pacing catheter tip. The use of an ECG to guide the placement of a pacing catheter is based on two concepts. First, the complex will vary in size depending on which chamber is entered. For example, when the tip of the pacing catheter is in the atrium, one will see large P waves, often larger than the corresponding QRS complex. Second, the sum of the electrical forces will be negative if the depolarization is moving away from the catheter tip and positive if the depolarization is moving toward the catheter tip. Therefore, if the catheter tip is *above* the atrium, both the P wave and the QRS complex will be negative (i.e., the electrical forces of a normally beating heart will be moving away from the catheter tip). As the tip progresses inferiorly in the atrium, the P wave will become isoelectric (biphasic) and will eventually become positive as the wave of atrial depolarization advances toward the catheter tip. The ECG resembles an aVR lead initially when in the left subclavian vein (Fig. 13-6 (Figure Not Available) *A*) or midsuperior vena cava (Fig. 13-6 (Figure Not Available) *B*). At the high right atrium, both the P wave and QRS complex are negative; the P wave is larger than the QRS complex and is deeply inverted (Fig. 13-6 (Figure Not Available) *C* and *D*). As the center of the atrium is approached, the P wave becomes large and biphasic (Fig. 13-6 (Figure Not Available) *E*). As the catheter approaches the lower atrium (Fig. 13-6 (Figure Not Available) *F*), the P wave becomes smaller and upright. The QRS complex is fairly normal. When striking the right atrial wall, an injury pattern with a P-Ta segment is seen (Fig. 13-6 (Figure Not Available) *G*). As the electrode passes through the tricuspid valve, the P wave becomes smaller, and the QRS complex becomes larger (Fig. 13-6 (Figure Not Available) *H*). Placement in the inferior vena cava may be recognized by a change in the morphology of the P wave and a decrease in the amplitude of both the P wave and the QRS complex (Fig. 13-6 (Figure Not Available) *I*).

Once the pacing catheter is in the desired position, the balloon is deflated by unlocking it and allowing the air to passively fill the syringe. One should avoid drawing back on the syringe, because this may cause balloon rupture. If the plunger does not move back spontaneously, the operator should assume that the balloon ruptured and should not subsequently place more air into the port. The pacing catheter should be withdrawn and the balloon checked for leaks. If a leak is found, a new pacing catheter should be used.

After successful placement of the catheter within the right ventricle, the tip should be advanced until contact is made with the endocardial wall. When this occurs, the QRS segment will show ST segment elevation (Fig. 13-6 (Figure Not Available) *J*). Ideally,

the tip of the catheter should be lodged in the trabeculae at the apex of the right ventricle; however, pacing may be successful if the catheter is in various other positions within the ventricle or outflow tract.

If the pacer enters the pulmonary artery outflow tract, the P wave again becomes negative, and the QRS amplitude diminishes (Fig. 13-6 (Figure Not Available) *K*). If the catheter is in the pulmonary artery, the pacing catheter should be withdrawn into the right ventricle and readvanced. Sometimes a clockwise or counterclockwise twist of the catheter will redirect its path in a more favorable direction. If catheter-induced ectopy develops, the catheter should be slightly withdrawn until the ectopy stops; then it should be readvanced. Occasionally an antidysrhythmic drug such as lidocaine may need to be given to desensitize the myocardium. Once ventricular endocardial contact is made, the catheter is disconnected from the ECG machine. The proximal positive and negative leads are connected to their respective terminals on the pacing generator. The pacing generator is then set to a rate of 80 beats/min or 10 beats/min faster than the underlying ventricular rhythm, whichever is higher. The full-demand mode is selected, with an output of about 5 mA. The pacing generator is then turned on. If complete capture does not occur or if it is intermittent, the pacer will need to be repositioned. When proper capture occurs, the pacer is tested for optimal positioning. This is done by testing the thresholds for sensing and pacing and with chest radiographs, physical examination, and ECG.

**Catheter Placement Without an Electrocardiograph**

Occasionally it is necessary to use a transvenous pacemaker in an emergency setting when a well-grounded ECG machine is not available. Blind insertion of the transvenous pacing catheter is a safe and effective alternative to placement with ECG guidance. In this technique, the pacing catheter is placed 10 to 12 cm into the venous port and is connected to the pacing generator as noted previously. The pacing rate is selected at *twice* the intrinsic heart rate, and the output is set at an amperage that is too low to capture the ventricle, usually <0.2 mA. The unit is then turned on to first *sense* but *not* to pace. On entering the ventricle, the pacer will sense on every other beat. The balloon can then be deflated, the amperage can be increased to 4 to 5 mA for initiating pacing, and the pacemaker can be advanced to capture the ventricle. If this does not occur within an additional 10 cm, the pacing catheter should be withdrawn to its original position and then advanced again. As with ECG placement, proper positioning must be ensured.

Fluoroscopy is a valuable tool in the placement of transvenous pacemakers. Its use depends on the operator's preference, the patient's condition, and its availability. Generally transvenous pacemakers are not inserted under fluoroscopy without ECG monitoring because of the high incidence of ventricular dysrhythmias. [40]

If the cardiac output is too low to "float" a pacing catheter or if the patient is in extremis, there may not be enough time to advance a pacing catheter using the previously described techniques. Such a situation would be asystole or complete heart block with malignant ventricular escape rhythms (although one can make a case for transthoracic or transcutaneous pacing in such conditions). In such emergent situations, the pacing

catheter is connected to the energy source, the output is turned to the maximum amperage, and the asynchronous mode is selected. The catheter is then blindly advanced in the hope that it will enter the right ventricle and that pacing will be accomplished. The pacing catheter is rotated, advanced, withdrawn, or otherwise manipulated according to the clinical response. The right internal jugular approach is the most practical access route in this situation. In such instances, there is the theoretic advantage of using the previously described Davison catheter, because one is interested in rapid capture only until the patient is stabilized.

**Dual Chamber Pacing**

Synchronous pacing of the atria and ventricle through a *dual-chamber pacing catheter* that senses and paces both atria and ventricles (DDD mode) results in more physiologic pacing with further improvement in cardiac output. It also allows for variations in the atrial rate and improved coordination of atrial and ventricular contractions. This catheter is placed in the same manner as the ventricle-only pacing catheter (VVI mode) but requires greater skill and experience because of the need to properly place the catheter in both the atria and ventricle. When attaching the catheter to the pacing generator, it is important to ensure that the atrial catheter is connected to the atrial electrode and the ventricular catheter is placed to the ventricular electrode. The Medtronic Model 5346 dual-chamber pacemaking generator is available for this purpose. It is capable of pacing in a variety of modes, including DDD mode. [17]

**Testing Threshold**

The threshold is the minimum current necessary to obtain capture. Ideally, this is <1.0 mA, and usually it is between 0.3 and 0.7 mA. If the threshold is in this ideal range, good contact with the endocardium can be presumed.

To determine the threshold, the pacing generator should be placed in the full-demand mode at 5 mA with a rate of approximately 80 beats/min. The amperage (output) should then be reduced slowly until capture is lost. This current is the threshold. This maneuver should be carried out 2 or 3 times to ensure that this value is consistent; the amperage should then be increased to 2½ times the threshold to ensure consistency of capture (usually between 2 and 3 mA).

If one reduces the output to below the threshold and then slowly increases it, there may be a difference in the point at which capture returns. This difference is called *hysteresis* and represents the time interval between sensing and pacemaker firing. If the difference in capture current is >20%, the pacing catheter should be repositioned, because serious dysrhythmias may result if the pacemaker fires during the vulnerable period of repolarization.

The sensing function should be tested in patients who have underlying rhythms. The pacemaker system is again set in full-demand mode with complete capture, and the rate is decreased until it is suppressed by the patient's intrinsic rhythm. This is done several

times to ensure accuracy of the sensing function.

In bipolar systems, another method of evaluating the sensing mode is to take a unipolar ECG from each end of the bipolar lead on a chest lead at one-fourth standardization to permit observation of the entire complex. [40] The voltage of the QRS complex is multiplied by 4 and, if adequate, should be greater than the sensing threshold by >1 mV (Fig. 13-7) (Figure Not Available) . Another method is to set the ECG machine on lead I and to connect the wires from the proximal electrode to the right arm lead and the left arm lead to the distal electrode. A lead I is created, which, when the QRS voltage is multiplied by 4, should also be at least 1 mV more than the sensing threshold.

### Securing and Final Assessment

After the pacemaker's position has been tested for electrical accuracy, the introducer sheath should be withdrawn (Fig. 13-8) and the catheter secured to the skin with suture (e.g., 4-0 nylon or silk). A fastening suture should be sewn to the skin and the catheter tied securely in place. The excess pacing catheter should be coiled and secured in a sterile manner along with the introducer. A large sterile dressing should be applied. Pacemaker function should again be assessed, and a chest film should be taken to ensure proper positioning. Ideal positioning of the pacing catheter is at the apex of the right ventricle (Fig. 13-9) (Figure Not Available) .

A 12-lead ECG should be obtained after transvenous pacemaker placement. If the catheter is within the right ventricle, a left bundle branch pattern with left axis deviation should be evident in paced beats (Fig. 13-10) . If a right bundle-branch block pattern is noted, coronary sinus placement or left ventricular pacing due to septal penetration should be suspected.

With a properly functioning ventricular pacemaker, large cannon waves will be noted on inspection of the venous pulsations at the neck. This is caused by the atria contracting against a closed tricuspid valve. On auscultation of the heart, a slight murmur secondary to tricuspid insufficiency from the catheter interfering with the tricuspid valve apparatus may be evident. [50] A clicking sound heard best during expiration following each pacemaker impulse may also be noted here and is believed to represent either intercostal or diaphragmatic muscular contractions caused by the pacemaker. [51] Note that this can also be a sign of cardiac perforation. [52] On auscultation of the second heart sound, paradoxical splitting may be noted. This represents a delay in closure of the aortic valve because of delayed left ventricular depolarization.

As in any procedure, the patient should then be assessed for improvement in his or her clinical status. An evaluation of vital signs, mentation, improvement in congestive symptoms, and urinary output must be noted. In addition, complications secondary to the procedure should be sought and treated as needed.

## COMPLICATIONS

The complications of emergency transvenous cardiac pacing are numerous and

represent a compendium of those related to central venous catheterization, those related to right heart catheterization, and those unique to the pacing catheter itself (Table 13-6) .

## Problems Related to Central Venous Catheterization

Inadvertent arterial puncture is a well-known complication of the percutaneous approach to the venous system. [53] This problem is usually recognized quickly because of the rapid return of arterial blood. Firm compression over the puncture site will almost always result in hemostasis in 5 minutes or less.

Venous thrombosis and thrombophlebitis are also potential problems with central venous catheterization. Thrombophlebitis, which occurs early after insertion, is said to be a rare complication. Some experts believe that it can be managed without removal of the catheter or anticoagulation. [48] When thrombophlebitis occurs in long-term implanted pacemakers, removal and anticoagulation may be required. In one series, only 0.1% of permanent pacemakers were in this category, and in a small percentage of these, occult malignancies were found. [48] Complete thrombosis of the innominate vein is also a rare problem, with pulmonary embolism an even more uncommon event. [54] Femoral vein thrombosis, however, appears to be a much more common event associated with femoral vein catheterization.  Studies using noninvasive techniques have shown a 37% incidence of femoral vein thrombosis, with 55% of these having ventilation-perfusion scan evidence of pulmonary embolism. [55] Thrombosis in the right atrium may also occur and has been treated successfully with thrombolytic agents. [56]

Pneumothorax is consistently a problem with the various approaches to the veins at the base of the neck. The decision to place a chest tube in patients with this complication depends on the extent of the air leak and the clinical status of the patient (see Chapter 9) . In addition, laceration of the subclavian vein with hemothorax, [57] thoracic duct laceration with chylothorax, air embolism, wound infections, pneumomediastinum, hydromediastinum [58] and hemomediastinum, phrenic nerve injury, [59] fracture of the guide wire with embolization, [60] [61] and catheter or guide wire knotting [62] [63] are all potential complications.

## Complications of Right Heart Catheterization

A common complication of the pacing catheter is dysrhythmia, with premature ventricular contractions being a common occurrence in my experience. One study noted a 1.5% incidence of serious dysrhythmias with a balloon-tipped catheter using ECG guidance, compared with a 32% incidence with the semirigid catheter using fluoroscopic guidance, suggesting that the balloon catheter was the preferred type of catheter. [15] Another study noted a 6% incidence of ventricular tachycardia during insertion. [46] The ischemic heart is more prone to dysrhythmias than the nonischemic heart. [64] The therapy for catheter-induced ectopy involves withdrawing the catheter from the ventricle. This usually stops the ectopy; however, if after repeated attempts it is found that the catheter cannot be passed without ectopy, myocardial suppressant therapy may be used to desensitize the myocardium.

Misplacement of the pacing catheter has been well studied. Passage of the catheter into the pulmonary artery can be diagnosed cardiographically by observing the return of an inverted P wave and the decrease in the voltage of the QRS complex. Misplacement in the coronary sinus may occur and

**Figure 13-10**Electrocardiogram pattern of right ventricular pacemaker.

| | | | | | TABLE 13-6 -- Complications of Transvenous Cardiac Pacing |
|---|---|---|---|---|---|

| Year | Author | No. of Patients | Catheter | Route | Result |
|---|---|---|---|---|---|
| 1969 | Rosenberg et al [1] | 111 | Flexon steelwire vs unipolar semifloating (ECG) | 96 Subclavian 5 Basilic 1 External jugular | 12 inconsistent pacing, 3 local infection, 2 pneumothorax, 1 subclavian artery puncture; 16% complication rate |
| 1973 | Schnitzler et al [14] | 17 | 3 Fr bipolar semifloating balloon (ECG) | Antecubital vein | 2 PVCs, stable pacing, no thrombophlebitis |
| 1973 | Weinstein et al [47] | 100 | 6 Fr bipolar (fluoroscopy) | Femoral | 2 ventricular tachycardia, 2 perforations, 2 required repositionings, 1 questionable thrombophlebitis and pulmonary embolism, 1 local infection |

| | | | | |
|---|---|---|---|---|
| 1973 | Lumia & Rios [87] | 142 insertions in 113 patients | Bipolar (fluoroscopy) | 61 Brachial 81 Femoral | 12 ventricular tachycardia and fibrillation in 9 patients, 3 perforations in 2 patients; local hematoma, abscess, and bleeding in 30%; 16.9% complication rate |
| 1980 | Pandian et al [88] | 20 | 5 Fr bipolar (fluoroscopy) | Femoral | 25% deep venous thrombosis |
| 1980 | Nolewajka et al [55] | 29 | 6 Fr cordis (fluoroscopy) | Femoral | 34% venous thrombosis by venogram with 60% of these with pulmonary embolism by VQ scan |
| 1981 | Lang et al [15] | 111 | Balloon, semifloating vs. semirigid | Subclavian | Serious dysrhythmia: 1.5% balloon-tipped, 20.4% semirigid Catheter displacement: 13.6% ± 4.4 days balloon-tipped; 32% ± 1.9 day semirigid |
| 1982 | Austin et al [46] | 113 insertions in 100 patients | 4-7 Fr bipolar (fluoroscopy) | Brachial Femoral | Failure to sense or pace in 37%; repositioning in 37% of brachial insertions; repositioning in 9% of femoral insertions; fever, sepsis, local infection only in femoral insertions; 20% complication rate |

ECG, electrocardiogram; PVC, premature ventricular contraction; VQ, ventilation-perfusion.

should be suspected in the patient in whom a paced right bundle branch pattern on the ECG is seen with right ventricular pacing (Fig. 13-11) . Rarely, a right bundle branch

pattern can be seen with a normal right ventricular position; therefore, all right bundle branch patterns do not represent coronary sinus pacing. [65] Further evidence for coronary sinus location can be obtained by viewing the lateral chest film. Normally, the catheter tip should point anteriorly toward the apex of the heart; however, with coronary sinus placement, the catheter tip is displaced posteriorly and several centimeters away from the sternum (Fig. 13-12) (Figure Not Available) . Other potential forms of misplacement include left ventricular pacing through an atrial septal defect or a ventricular septal defect, septal puncture, extraluminal insertion, and arterial insertions.

Perforation of the ventricle is a well-described complication that can result in loss of capture, [67] hemopericardium, and tamponade. [68] [69] Reported symptoms and signs of this problem include chest pain, pericardial friction rub, and diaphragmatic or chest wall muscular pacing. [70] At least 1 case of a postpericardiotomy-like syndrome and 2 cases of endocardial friction rub have been reported without perforation.

Pericardial perforation is suggested radiographically when the pacing catheter is outside or abuts the cardiac silhouette and is not in proper position within the right ventricular cavity (Fig. 13-13) (Figure Not Available) . [73] ECG clues include a change in the QRS and T wave axis or a failure to properly sense. In suspected cases, a two-dimensional echocardiogram usually demonstrates the catheter's extracardiac position. Uncomplicated perforation can usually be treated by simply pulling back the catheter and repositioning it in the right ventricle.

During the insertion of a temporary pacing catheter when a nonfunctioning permanent catheter is in place, there is a small risk of entanglement or knotting. This potential also exists with other central lines and Swan-Ganz catheters. Frequently these lines can be untangled under fluoroscopy using specialized catheters.

Local and systemic infections, [48] balloon rupture, pulmonary infarction, [74] phrenic nerve pacing, [75] and rupture of the chordae tendineae are also potential complications. [74]

**Complications of the Pacing Electrode**

The complications related to the pacing electrode can be separated into three groups: mechanical, organic, and electrical.

Mechanical failures include displacement, fracture of the catheter, and loose leads. Displacement can result in intermittent or complete loss of capture or improper sensing, malignant dysrhythmias, diaphragmatic pacing, or perforation. Displacement should be suspected with changes in amplitude, with vector changes >90°, or with a change in threshold. [76] Frequently catheter fractures may be detected by a careful review of the chest film or may be suspected because of a change in the sensing threshold. As with displacement, intermittent or complete loss of capture may result.

Organic causes of pacemaker failure result in changes in the threshold or sensing function. [77] Progressive inflammation, fibrosis, and thrombosis may result in more than a doubling of the original threshold. [78] This may occur in 3 to 4 weeks and should be

expected in prolonged temporary pacemakers. Physiologic and pharmacologic factors that affect the threshold have been studied. Sleeping, eating a heavy meal, lowered aldosterone concentration, potassium infusions, [79] and myxedema [80] all increase the threshold by raising the resting membrane potential. The threshold for cardiac pacing tends to decrease with exercise, sympathetic amines, glucocorticoids, and toxic levels of procainamide. [81]

In some patients the atrial contribution to ventricular filling is extremely important. Transvenous ventricular pacing results in loss of the atrial kick and ultimately a decrease in left ventricular stroke volume. This phenomenon is called *postpacer syndrome* and occasionally is severe enough to preclude the use of a pacemaker. [82] A bichamber sequential pacemaker that stimulates the atria and ventricles in sequential fashion is a viable alternative for patients unable to tolerate the loss of the atrial kick. [83]

Electrical problems with pacing include pacemaker generator failure, dysrhythmias, and outside interference. Electrical interference is of occasional importance during aeromedical transport. [84] Usually converting the unit to a fixed mode will permit continued pacing. Although ventricular tachycardia and ventricular fibrillation have been reported to result from pacemakers, these dysrhythmias are rare. Because of this, patients who present with such dysrhythmias should be evaluated for a nonpacemaker etiology. [85] Direct current cardioversion and electroshock therapy are safe procedures to carry out in patients who have pacemakers as long as the current does not go directly over the generator pack.

## CONCLUSION

Temporary transvenous pacing is a rapid, safe, and reliable method for achieving effective electrical stimulation of the heart. It can be mastered by any physician who is responsible for the care of critically ill or injured patients. Symptomatic bradycardias unresponsive to pharmacologic treatment and some tachycardias are indications for its use. In acute myocardial infarction, it serves both a therapeutic and a prophylactic function.

# Chapter 12 - Assessment of Implanted Pacemaker/AICD Devices

**David W. Munter**

Patients with permanent implanted pacemakers or automatic implantable cardioverter-defibrillators (AICDs) are commonly seen in the emergency department (ED). At least 500,000 patients in the United States have permanent pacemakers, [1] with as many as 300,000 pacemakers placed or replaced annually. [2] More than 10,000 AICDs have been implanted in patients. [3]

Recipients of these devices have underlying cardiac disease, which is often severe, and therefore frequently use emergency departments for cardiac complaints. Additionally, complications from the devices are not uncommon, with 7.4 to 15% of pacemakers failing in the first year. [4] AICD complication rates, including inadvertent shocks, occur in up to 34% of patients with the device. [5]

Although the basic evaluation and treatment of patients with pacemakers or AICDs presenting with cardiac complaints is not substantially different from that of patients without the devices, a knowledge of the range of potential problems and complications and techniques for evaluating or inactivating pacemakers or AICDs is important for emergency physicians.

## BACKGROUND

The relationship between applied electricity and ventricular rhythms and arrhythmias has been understood since the late 1800s. [3] With the onset of cardiac monitoring techniques in the 1940s, the relationships between sudden death, cardiac syncope, ventricular fibrillation, and bradycardias began to be understood. [6]

The first pacemakers were developed by Zoll in the 1950s. [6] The units were transcutaneous and used primarily for Stokes-Adams disease (syncope secondary to bradycardia or asystole). These early units could only be used transiently and only in a hospital setting, as they created severe patient discomfort. [6] In 1958, the first long-term implanted pacemaker was used. [7] The indications for pacemaker use were rapidly expanded, as was the technology of the devices.

The first human defibrillation was performed intraoperatively in 1947 and the first transthoracic defibrillation in 1956. [8] Rapid defibrillation is now the standard of care for ventricular fibrillation, and its use has significantly increased survival of cardiac arrest. However, patients who survive cardiac arrest have a high risk of recurrent arrest, estimated at 22% to 60% in the first year. [9] [10]

AICDs were developed by Mirowski in the late 1960s, but they were not accepted by the medical community until the late 1970s. [3] In 1980, the first AICD was implanted in a human, followed by implantation in a series of patients [11] in whom an average of 52%

decrease in mortality was seen. [12] Between 1985 and 1990, more than 10,000 patients received AICDs, and the number continues to increase

## PACEMAKER CHARACTERISTICS

Several hundred types of pacemakers are in use today. The typical generator is a hermetically sealed device weighing from 30 to 130 g. [13] Modern power sources are almost exclusively lithium-based batteries, [14] which have the advantage of a slow, steady, predictable decay rate. Half-lives of lithium batteries range from 75 to 144 months. [14] The generator is connected to sensing and pacing electrodes that are placed in varying locations in the heart depending on the configuration of the pacemaker. Newer models are programmable for rate, output, sensitivity, refractory period, and modes of response, [13] and they can be reprogrammed radiotelemetrically after implantation.

Pacemakers are classified according to a standard code developed by the North American Society of Pacing and Electrophysiology/British Pacing and Electrophysiology Group (Table 12-1) . The first letter designates the chamber that actually receives the pacing current, whereas the second letter denotes which chamber the pacemaker uses to sense intrinsic cardiac electrical activity. The third letter denotes the pacemaker's response to sensed intrinsic electrical activity (usually inhibited). The fourth letter refers to the pacemaker's rate modulation and programmability, whereas the fifth letter describes antitachycardiac features of the pacemaker. In normal practice, only the first three letters are used to describe the pacemaker (e.g., VVI or DDD). [15]

**TABLE 12-1** -- North American Society of Pacing and Electrophysiology/British Pacing and Electrophysiology Group Generic Pacemaker Code (NBE Code)

| I | II | III | IV | V |
|---|---|---|---|---|
| Chamber Paced | Chamber Sensed | Response to Sensing | Rate Modulation and Programmability | Antitachycardia Features |
| 0--None | 0--None | 0--None | 0--None | 0--None |

| A--Atrium | A--Atrium | I--Inhibited | I--Inhibited | P--Antitachycardiac pacing |
|---|---|---|---|---|
| V--Ventricle | V--Ventricle | T--Triggered | M--Multiple | |
| D--Dual | D--Dual | D--Dual | C--Communicating | S--Shock |
| | | | R--Rate modulation | D--Dual |

Pacemaker wires are embedded in plastic catheters, and the terminal electrodes may be unipolar or bipolar. They travel from the generator unit to the heart via the venous system. The typical entry point is the subclavian or cephalic vein. The terminal electrodes are placed in the proper chambers, either the right ventricle, or both the right ventricle and atrium, under fluoroscopic guidance. Proper lead placement is checked by electrograms checking sensing and pacing thresholds. [16] The typical radiographic appearance of an implanted pacemaker is seen in Figure 12-1 .

The pacemaker rate is typically programmed between 60 and 80 beats/min. The pulse generator output is the product of volts, milliamperes, and the duration of stimulus, all of which may be preset or programmed. Sensing of intracardiac electrical activity is a combination of recognizing the characteristic waveforms of P waves or QRS complexes while also discriminating these from T waves or external interfering signals, such as muscle activity or movement. The pacing electrical stimulus is a triphasic wave consisting of an intrinsic deflection, far-field potential, and an injury current, which typically delivers a current of 0.1 to 20.0 mA for 2 msec at 15 V. [17]

Pacemakers have a reed switch, which may be closed by placing a magnet over the generator; this inactivates the sensing mechanism of the pacemaker, which then reverts to an asynchronous rate, termed the *magnet rate*. The magnet rate is usually, but not always, the same as the programmed rate.

## AICD CHARACTERISTICS

The AICD consists of a pulse generator with sensing mechanism, battery, and attached lead system with sensing and shocking abilities. The pulse generator and battery are contained in a hermetically sealed titanium case weighing 250 to 300 g. This case is approximately 10.8 × 2 × 7.6 cm, with variations depending on the manufacturer. The battery is typically a lithium battery with a life span of approximately 100 shocks, [18] [19] although a maximum life span of 22 months has been noted in one study. [9]

A variety of sensing and shocking leads are in use. Most current AICDs use two bipolar sensing electrodes that are secured in the epicardium, with two ventricular patches for shock delivery, which are usually placed anteriorly and posteriorly over the left ventricle (Figs. 12-2 (Figure Not Available) and 12-3) .

A new AICD with a single transvenous lead containing both sensing and shocking components is also in use (Fig. 12-4) . This model offers the advantage of obviating the need for cardiac surgery.

The electrical components of the AICD continuously monitor both rate and waveform [20] through separate sensing channels. Different models place a different emphasis on these channels, depending on the patient's underlying disease. The rate-detection channel determines if a preset limit, individualized for each patient, is exceeded. The second channel analyzes the morphology of the waveform of intrinsic cardiac electrical activity. Essentially, variance from a baseline is measured to determine if a shockable rhythm is present. Normal sinus rhythm has periods of isoelectric activity (the baseline), but ventricular fibrillation or sinusoidal tachydysrhythmias typically have a lack of these isoelectric segments.

A malignant ventricular tachydysrhythmia is usually present for 5 to 15 seconds before the preset rate and morphologic criteria are met. [18] At this time, the AICD charges its capacitor and delivers a shock of 25 J. This shock is synchronized in the case of ventricular tachycardia and unsynchronized in ventricular fibrillation. The time from the initiation of the arrhythmia to delivery of the shock is from 10 to 35 seconds.

If the arrhythmia persists after the first shock, subsequent shocks of 30 J are delivered, with each subsequent detection and shocking period lasting from 10 to 35 seconds. A total of four shocks are delivered if the rhythm is unchanged, after which the AICD does not deliver more shocks unless a subsequent change occurs in the rhythm that lasts more than 35 seconds. [5]

The AICD case is implanted in a subcutaneous pocket in the abdominal wall (Figs. 12-5 (Figure Not Available) and 12-6) . The wires are run through a subcutaneous tunnel to the chest. A variety of surgical approaches to the heart are used. [21] The ventricular patches are sutured to the epicardium or parietal pericardium in roughly anterior and posterior positions on the left ventricle, depending on the surgical approach. The sensing electrodes may be placed in either the left or right ventricle.

In those patients in whom the new transvenous lead is used, thoracotomy is not needed. In these patients the AICD case is still in the typical abdominal position, and a submuscular patch is placed in the chest wall.

AICDs may be inactivated by a magnet, either purposely or inadvertently. They may also be interrogated via a radiotelemetry device, which is a procedure that is normally only performed by the cardiologist.

# INDICATIONS FOR IMPLANTABLE DEVICE USE

Current indications for permanent pacemaker use include complete heart block, symptomatic type II second-degree block, second-degree block with episodic ventricular arrhythmias, sick sinus syndrome, symptomatic bradycardias with syncope or presyncope, hypersensitive carotid artery syndrome, type I block with infra-His bundle block, and certain subgroups of patients with triphasic and biphasic blocks at risk of developing sudden high-degree block. [16] [22]

The use of AICDs is generally limited to patients who are at high risk of sudden cardiac death from ventricular arrhythmias. Currently accepted indications are a documented episode of hemodynamically significant, sustained ventricular tachycardia or fibrillation; ventricular arrhythmia refractory to standard antiarrhythmic therapy as demonstrated electrophysiologically; persistent electrophysiologically inducible ventricular arrhythmia despite best available drug therapy; and recurrent syncope in a patient with electrophysiologically inducible ventricular arrhythmia in whom no effective drug is tolerated or available. [3] [18] [23]

Contraindications to AICD use are life expectancy of less than 6 months, New York Heart Association class IV heart failure, treatable causes of ventricular arrhythmias, or incessant or very frequent ventricular arrhythmias that result in rapid battery depletion.

# COMPLICATIONS OF PERMANENT PACEMAKERS

Complications are commonly seen with permanent pacemakers. Failure rates in the first year postimplantation range

**Figure 12-5** (Figure Not Available) Typical external appearance of AICD implanted in the abdominal wall. *(Courtesy of Lawrence B. Stack, M.D. From Munter DW, DeLacey WA: Automatic implantable cardioverter-defibrillators. Emerg Med Clin North Am 12:579-595, 1994. Used with permission.)*

from 7.4 to 15.0%, with most occurring within the first 30 days. [4] After this initial period, approximately 6% of pacemakers fail each year. [24]

Pacemaker failure can be categorized as failure to pace, failure to sense, failure to capture, inappropriate pacemaker rate, and other complications (e.g., vascular or infectious) (see Table 12-2) . Failure to sense is the most common problem, accounting for 32 to 57% of failure cases.

## Failure to Pace

This condition is characterized by the lack of production of pacemaker spikes despite the lack of intrinsic cardiac electrical activity or an intrinsic cardiac rate falling below the threshold for pacing. Causes of failure to pace include lead fracture or disconnection, battery depletion, component failure, and oversensing.

Lead fracture or disconnection may occur months to years after pacemaker implantation and may be due to inherent stress at the lead connection site. Blunt trauma may also cause lead fracture. [26] [27] [28]

Battery depletion is normally a gradual process and is usually detected before complete failure of the pacemaker.

| TABLE 12-2 -- Complications of Permanent Pacemaker Use |
| --- |
| **Failure to pace (no pacemaker activity present)** |
| Lead fracture |
| Lead disconnection |
| Battery depletion |
| Component failure |
| Oversensing |
| External interference |
| **Failure to sense (constant pacemaker spikes despite ongoing intrinsic cardiac electrical activity)** |
| Lead dislodgement |

| |
|---|
| Lead fracture |
| Fibrosis around lead tip |
| Battery depletion |
| Pacer in asynchronous mode |
| External interference |
| Low-amplitude intracardiac signal |
| **Failure to capture (pacemaker spikes but no subsequent cardiac activity)** |
| Lead dislodgement including perforation |
| Lead fracture |
| Lead disconnection |
| Poor lead position |
| Fibrosis around lead tip |

| |
|---|
| Battery depletion |
| Metabolic abnormalities |
| Medications |
| **Inappropriate pacemaker rate (runaway pacemaker)** |
| Pacemaker reentrant tachycardia |
| Resetting from external interference |
| Battery depletion |
| **Other** |
| Infections: pocket, wires |
| Lead displacement: cardiac perforation, tamponade, pericarditis, vascular perforation |
| Vascular complications: thrombosis, superior vena cava syndrome |
| Psychiatric: anxiety, panic attacks |

Patients who have not had regular follow-up may present with previously undetected

battery depletion.

Component failure may be due to a variety of external influences including blunt trauma, therapeutic radiation, electrocautery, transthoracic defibrillation, diathermy, electroshock therapy for depression, magnetic resonance imaging (MRI), and extracorporeal shock-wave lithotripsy.

Pacemakers may oversense, or misinterpret non-QRS complex electrical activity (e.g., P waves, T waves, muscular activity, or chest thumping). [25] If this activity is interpreted as a QRS complex, the ventricular spike will be inhibited. If, however, the pacemaker is dual chambered and the electrical activity is misinterpreted as a P wave, it will stimulate firing of the ventricular electrode, which can lead to overpacing. External stimuli can also be misinterpreted and lead to oversensing. This interference can include electrocautery, MRI, diathermy, transcutaneous electrical nerve stimulation (TENS), electroshock therapy, ultrasound dental scalers, static electricity, or vibration (e.g., from a tractor or helicopter).

## Failure to Sense

This condition is characterized by the presence of constant pacemaker spikes despite ongoing intrinsic cardiac electrical activity. Failure of the pacemaker to sense cardiac electrical activity can be due to lead dislodgement, lead fracture, normal development of fibrosis around the lead tip that occurs with time, battery depletion, external interference, or an intrinsically low-amplitude cardiac signal.

Lead dislodgement, the most common reason for failure to sense, can be due to an enlarged right ventricle, poor initial lead positioning, blunt trauma, or patient manipulation of the generator unit in the chest wall pocket (pacemaker twiddler's syndrome).

Fibrosis normally develops around the tip of the electrode, and this fibrosis can lead to abnormal sensing or higher required thresholds for pacing. [1]

Several cardiac and metabolic abnormalities cause the intrinsic cardiac electrical activity to be of lower than normal amplitude, causing undersensing of the QRS complexes.

## Failure to Capture

This condition is the appropriate presence and timing of pacemaker spikes, but without resultant cardiac activity. Reasons for failure to capture include lead dislodgement (e.g., myocardial perforation), lead fracture, lead disconnection, poor lead position, lead tip fibrosis, battery depletion, and metabolic abnormalities (e.g., hyperkalemia) or medications (e.g., lidocaine, flecainide) that make the myocardium less responsive to electrical impulses. Lead dislodgement is the most common reason for failure to capture. [25]

### Inappropriate Pacemaker Rate

This condition, also known as a *runaway pacemaker*, is a rare complication that is usually seen in dual-chamber pacemakers.  In dual-chamber pacers, it is caused by an endless loop reentry tachycardia, often initiated by a retrograde P wave. [41] In older, or single-chamber pacemakers, it can be due to component failure or battery depletion. [42] Component failure is rare, as almost all pacemakers have circuitry to prevent high discharge rates.

### Other Complications

Infections including localized skin or pocket infections, more complicated infections along the route of the wires, or endocarditis occur in 1 to 15% of patients. These infections are initially treated with broad-spectrum antibiotics against *Staphylococcus aureus*, but they often require removal of the pacemaker.

Lead displacement, in addition to causing failure to sense or capture, can cause injury to the myocardium including cardiac perforation with resultant tamponade or restrictive pericarditis,  and it is usually seen early after pacemaker implantation.

Fracture of the J-shaped retention wire within the lead can result in a protrusion of the wire outside the protective plastic coating. This protruding wire can then puncture the superior vena cava or right atrium resulting in bleeding or cardiac tamponade. [48] Figure 12-7 (Figure Not Available) illustrates this fracture.

Vascular complications include thrombosis or superior vena cava syndrome. Many pacemaker patients have a benign thrombosis of the upper arm or shoulder, but only about 2% have a serious thrombotic or embolic event. [45] Superior vena cava syndrome is a rare complication. [49]

Psychiatric complications include panic attacks and anxiety, which may prompt ED evaluation.

## COMPLICATIONS OF AICDs

The most common complication is the delivery of inappropriate shocks by the device when not indicated by a tachydysrhythmia. Up to 35% of AICD patients receive inappropriate

**Figure 12-7** (Figure Not Available) *A* and *B*, Appearance of fracture of atrial "J" retention wire. *A*, Radiographic appearance of fracture of atrial "J" retention wire. *B*, Diagram of fracture site. *(Courtesy of Telectronics Pacing Systems, Englewood, Colo.)*

shocks. [50] Causes of inappropriate shock delivery include misinterpretation of sinus tachycardia, atrial fibrillation, muscular activity (e.g., shivering), T waves, or extraneous sources (e.g., pacemaker spikes or vibrations) as a shockable tachydysrhythmia.

Likewise an unsustained tachydysrhythmia also may be shocked. Component failure, such as electrode failure or migration, may result in false sensing and resultant shocking. Pacemaker magnet testing has produced inappropriate shocks resulting in ventricular fibrillation.

Other reported long-term complications include interference with cardiac pacemakers, particularly after AICD discharge [54] [55] ; component failures, such as patch migration or distortion, [56] lead fracture, generator case rotation or fracture, and battery depletion; constrictive pericarditis and pericardial effusions,  cardiac fibrosis, and atrial or ventricular wall perforations after repeated shocks [59] [60] ; abdominal pocket infections [61] ; thrombosis and pulmonary embolism; erosion into the lung with hemoptysis [62] ; patient trauma sustained during falls after delivery of a shock, either due to the physical "jolt" of the shock [63] or to suspected postshock bradycardia and hypotension] ; and psychiatric disorders, such as adjustment disorder, panic attacks, or major depression.

The AICD may be inadvertently inactivated by any strong magnetic force including microwave ovens, industrial engines, metal detectors, magnets in speakers, refrigerator door magnets, bingo wands, and model airplane starters. [5] This inactivation is not normally noted until routine follow-up, but if the patient has a tachydysrhythmia, no shock will be delivered.

## EVALUATION OF PACEMAKER PATIENTS

Pacemaker patients presenting to the ED for any problem that may be associated with the device should have an evaluation that includes investigation of potential pacemaker malfunction.

**Historical Issues**

Pertinent information about the pacemaker unit should be obtained. The brand and manufacturer, type (NBE code), *implantation site, programmed rate, and any changes seen during follow-up at a pacemaker clinic should be noted. The time of implantation is important as certain types of complications such as lead failure, migration, or perforation typically occur within 3 months, whereas generator or battery failure usually occur later. [1]

Some medications, such as lidocaine or flecainide, can raise myocardial thresholds to pacing, [25] and their use should be queried.

Patient symptoms that may be related to pacemaker failure should be ascertained. Chest pain may be due to cardiac ischemia, but pacemaker complications such as cardiac perforation, pericarditis, or infection may also be the cause.

Recent trauma, especially to the chest or back, can cause failure of the generator unit, lead fracture, or lead displacement. Recent cardioversion or defibrillation, MRI, diathermy, lithotripsy, or electroconvulsive therapy for depression can have the same

effect.

Symptoms of decreased cerebral perfusion, such as syncope, near-syncope, or orthostatic light-headedness, may be an indication of pacemaker malfunctioning leading to bradycardias. Runaway pacemaker can also cause these symptoms as a result of low cardiac output during the tachycardic phase.

Palpitations are more typically due to intrinsic cardiac activity, but pacemaker failure to sense can cause an irregular rhythm due to inappropriate generation of pacer spikes, which may be perceived by the patient.

## Physical Examination

The patient should be examined for potential pacemaker complications. The head and neck should be inspected for venous engorgement that may be indicative of thrombosis or superior vena cava syndrome. [1]

The chest wall, and especially the pacemaker pocket, should be inspected and palpated for erythema, edema, tenderness, and location of the pacemaker generator. The initial course of the lead wires should also be palpated. If the generator unit is malrotated or in a position other than the original pacemaker pocket, this may be indicative of "pacemaker twiddler's syndrome" (i.e., purposeful or inadvertent manipulation by the patient), [38] [39] which can lead to lead dislodgement or disconnection.

The cardiac examination may be altered by the functioning of the pacemaker. A paradoxically split second heart sound is normal and is due to the origination of the pacemaker spike in the right ventricle. If the second heart sound is widely split, and widens more with inspiration, a left ventricular origin may be present due to cardiac perforation or lead migration. [1] A pericardial friction rub may be due to pericarditis, potentially from lead perforation. [46] A new murmur may be the result of infective endocarditis. [1]

## X-Ray

A chest radiograph should be obtained and compared to old films, preferably ones taken shortly after implantation. Location of the generator and battery type should be ascertained. Lithium batteries are seen as a single radiopaque portion of the generator unit, whereas older pacemakers have 4 to 5 button batteries. Location of the leads should be noted and migration should be looked for. Lead presence outside the myocardial shadow is indicative of perforation. The connection site at the generator unit should be examined for disconnection. The lead wires should be traced and examined for fracture, or J retention wire fractures.

## Electrocardiogram

The electrocardiogram (ECG) contains important information about pacemaker function and should be compared to old ECGs. An asynchronous mode pacemaker (i.e., VOO)

will have regular pacer spikes followed by QRS activity. A left bundle-branch pattern is normal, as the lead is usually in the right ventricle. Absence of pacer spikes in the asynchronous mode is termed *failure to pace*. Absence of a QRS complex after a pacer spike represents failure to capture. Some pacemakers have a programmed feature called hysteresis, in which the intrinsic rate below which pacing is triggered is somewhat lower than the resultant pacing rate. Hence the pause following a spontaneous QRS complex may be longer than an R-R interval at normal pace.

Pacemakers that sense intrinsic cardiac activity and are inhibited by same (i.e., VVI) should only generate pacer spikes if the patient's intrinsic rate falls below the programmed rate. In this case, pacer spikes followed by QRS complexes, again in a left bundle-branch pattern, should be noted. Failure to pace or capture may be seen. Pacer spikes occurring despite an intrinsic rate above the programmed rate represent failure to sense.

Dual chamber pacemakers (i.e., DDD) can have a variety of paced rhythms. [1] This includes the patient's intrinsic rhythm, atrial pacer spikes followed by a P wave and then an intrinsic QRS, or two pacer spikes sequentially followed by a P wave and then a QRS complex. Failure to pace, capture, or sense may again be noted.

The electrical axis of the pacer spike and paced QRS complex should be compared to old ECGs. A change of the pacer spike axis may be a sign of lead migration. [1] Normally, the paced QRS complex is a left bundle-branch pattern. A right bundle-branch pattern may be due to left ventricular lead placement, but a change from old ECGs from a left to a right bundle-branch pattern may be due to migration of the lead or perforation of the ventricular septum.

Paced rates that are higher than the programmed rate can be due to pacemaker-mediated tachycardia, a reentrant tachycardia normally seen only in dual-chambered pacemakers, [40] [41] or runaway pacemaker, normally seen only in older pacemaker models and due to battery depletion or component failure. [42] External electrical sources can mimic pacemaker spikes and be misinterpreted as an inappropriate pacing rate. [1]

Patients who are found to have failure to sense or failure to capture should undergo the basic evaluation outlined above; in addition, serum electrolyte assays should be performed to look for metabolic abnormalities. These patients and patients with infections, lead displacement, disconnection or fracture, or vascular complications will require cardiology consultation for intervention as determined by the etiology of the malfunction.

Patients who have failure to pace should undergo a

**Figure 12-8** Ring magnet.

**Figure 12-9** Typical location of pacemaker pocket in upper chest wall.

magnet test in the ED to rule out battery depletion or oversensing.

Patients with pacemaker-mediated tachycardia or runaway pacemaker need prompt cardiology evaluation, and those whose condition is unstable require acute intervention, as outlined below, in the ED.

### Use of a Magnet in Pacemaker Assessment

**Indications**

Magnet testing of a pacemaker is indicated to assess patients with "failure to pace" to assess for battery depletion, component failure, or oversensing. It is also indicated for pacemaker-mediated tachycardia or runaway pacemaker in an attempt to terminate the rhythm.

**Equipment and Setup**

Typically, a ring magnet (Fig. 12-8) is used for this procedure. Different brands of magnets are also available for specific pacemakers, but in almost all cases, a standard pacemaker ring magnet will suffice. The patient should undergo a baseline ECG. The patient should be on a cardiac monitor. Placement of a magnet on a pacemaker closes a reed switch and reverts the pacemaker to an asynchronous mode.

**Technique**

The location and orientation of the pacemaker generator should be ascertained by palpation. It is typically in the left or right upper chest wall (Fig. 12-9) . The magnet is placed directly over the pacemaker generator (Fig. 12-10) . A repeat ECG is obtained to compare to the baseline (Fig. 12-11 and 12-12) . Some brands of pacemakers have either a several-second delay or a series of 2 to 3 rapid pacer spikes before reverting to the asynchronous mode. [1] If the patient has an underlying bradycardia associated with failure to pace that is corrected by magnet placement, the magnet should be left over the generator. Likewise, if a pacemaker-mediated tachycardia or runaway pacemaker is reverted to a normal rate or rhythm, the magnet should be left in place pending consultation with a cardiologist.

**Complications**

Incorrect alignment of the magnet may cause only intermittent pacing in the asynchronous mode. Some pacemakers

**Figure 12-10** Application of ring magnet over pacemaker generator.

react erratically to a non-brand-specific magnet; if this is considered to be an issue, attempt to obtain a brand-specific magnet.

### Interpretation

In the case of failure to pace, three outcomes are possible: (1) The pacemaker will fire at its programmed rate (this is the expected outcome). In the case of failure to pace, this indicates that the pacemaker was oversensing and was inappropriately inhibited. The cause of the oversensing should then be investigated. (2) The pacer may not produce any spikes at all. This is indicative of component failure and requires cardiology consultation. (3) The pacemaker may produce spikes, but at a rate lower than the programmed rate, which is indicative of battery depletion; this also requires cardiology consultation.

## Management of Pacemaker-Mediated Tachycardia or Runaway Pacemaker

### Indications

These maneuvers are indicated for correction of an inappropriately high pacemaker rate.

### Equipment and Setup

IV access should be obtained and the patient should be placed on a cardiac monitor. A baseline ECG should be obtained. Because the definitive treatment often requires radiotelemetric reprogramming of the pacemaker, an immediate ("stat") cardiology consultation should be obtained. A ring magnet will be needed. A transcutaneous pacemaker unit with pads may be needed. If open lead disconnection is required, local anesthetic, sterile drapes, scalpel blade and handle, hemostats, and Mayo scissors or wire cutters will be needed as well as either a transcutaneous pacemaker or portable pacemaker unit with alligator clip.

### Technique

The initial procedure is to place a ring magnet over the pacer generator as described above. If the pacer then reverts to an asynchronous mode at the appropriate programmed rate, the magnet should be left in place.

If magnet placement has no effect, then isometric pectoral exercises should be attempted. [15] [40] This is done by having the patient place his hand from the same side as the pacemaker generator onto the opposite shoulder and push against the shoulder as long and as hard as possible. This creates rapid muscle activity in the pectoral muscle surrounding the pacemaker generator, which may be interpreted as ventricular activity and inhibit the pacemaker, [40] [41] terminating the reentrant tachycardia.

If this maneuver is unsuccessful and the patient's condition is stable, continue to monitor the patient and await cardiology consultation. If the patient's condition is unstable, the next maneuver is to attempt a standard chest *(precordial)* thump. [15] [40] This is performed by firmly striking the midsternum with a clenched fist from a distance of 30 to 38 cm. This procedure may be repeated once if needed. [40]

If the chest thump is unsuccessful, attach transcutaneous pacemaker pads anteriorly and posteriorly, and attach the leads to the transcutaneous pacemaker generator. Pace the patient at an initial output of 2 to 5 mA and a rate of 40 beats/min. [40] This will stimulate chest wall movement that may be interpreted as ventricular activity and inhibit the pacemaker, terminating the reentrant tachycardia. Unipolar DDD pacemakers are normally inhibited at a low output. Bipolar DDD pacemakers often need higher outputs of up to 10 to 20 mA. [40] If increasing the output is unsuccessful, raise the transcutaneous pacing rate slightly to attempt to deliver chest wall stimulation outside the pacemaker's particular ventricular refractory period. [40]

If these maneuvers are unsuccessful and the patient is hemodynamically unstable due to the pacer-induced tachycardia, the solution of last resort is to cut the pacer leads from the generator unit. The generator unit should be palpated to ascertain position and orientation. Apply transcutaneous pacemaker pads and connect them to the transcutaneous pacemaker. This is a precautionary measure, as cutting the leads may lead to profound bradycardia or asystole. A portable pacemaker generator should be available. Use povidone-iodine for sterile preparation of the site over the generator and drape with sterile drapes. Instill local anesthesia in the skin and subcutaneous tissue overlying the generator in the area of the lead connections. Make a skin incision with a scalpel, and expose the lead wires with blunt dissection. Alternatively, make an incision through the previous scar and remove the pacemaker unit. [42] Cut the lead wires close to the generator using Mayo scissors or wire cutters. At this point, the patient may require transcutaneous pacing. An alternate is to insert a needle into the cathode (negative) lead wire (this is the only wire on unipolar models, and it is identified on bipolar units either by marking or by a white band). [42] Connect an alligator clip to the needle, and connect the alligator clip to the negative terminal of the portable pacemaker generator. Ground the positive terminal via an alligator clip to the subcutaneous tissue of the incision, and pace the patient using the portable generator. [42]

## Complications

Incorrect alignment of the magnet may cause pacing only intermittently in the asynchronous mode. Some pacemakers will react erratically to a non-brand-specific magnet; if this is a problem, attempt to obtain a brand-specific magnet. There are no potential complications from isometric pectoral muscle exercise. Chest thumps can result in sternal or rib fractures, myocardial contusion, pulmonary contusion, extrasystolic

complexes, or ventricular tachydysrhythmias. [40] Transcutaneous pacing to stimulate chest wall movement is normally uncomfortable for the patient and can also result in diaphragmatic or arm muscle stimulation as well. Open disconnection of the pacemaker

lead wires can be complicated by unanticipated bleeding, difficulty exposing the generator unit, and subsequent infection. A more common complication is the termination of the paced rhythm resulting in a profound bradycardia or asystole that requires ongoing transcutaneous or portable generator pacing. [42]

## Interpretation

Correction of the pacer-induced tachycardia by lead disconnection only is generally indicative of component failure or severe battery depletion, whereas termination by noninvasive procedures is more indicative of a pacemaker-induced endless loop reentrant tachycardia

# EVALUATION OF AICD PATIENTS

Patients with an AICD who present to the ED can have noncardiac complaints unrelated to the AICD, cardiac complaints, or AICD-related problems, including AICD shocks. The evaluation should focus on potential AICD problems.

## Historical Issues

Patients with AICDs have severe underlying cardiac disease, and will often present with cardiac chest pain, shortness of breath, or congestive heart failure. Potential AICD complications such as pericarditis, pericardial effusion, cardiac fibrosis, atrial or ventricular perforation, and infections of the wires or leads can all present with chest pain. Mortality in AICD patients is typically due to their underlying disease, and any complaints of chest pain must be pursued aggressively.

Generator pocket infections or wound infections will produce complaints of pain and fever.

The most common AICD-related complaint in patients presenting to the ED is that of one or more AICD shocks. Patients describe the shock as a feeling of being kicked or punched in the chest. [69] [70] The number of shocks received should be ascertained. Associated symptoms of syncope or near-syncope indicate a probable tachydysrhythmia and appropriate shock. Many patients fall down when they experience an AICD shock, and the presence of any fall-related trauma should be queried.

## Physical Examination

Inspection and palpation of the abdominal pocket and subcutaneous tunnel should be done for signs of infection. Heart and lung sounds should be auscultated. A pericardial rub is indicative of pericardial fluid, which may be a result of pericarditis from the AICD. The patient should be examined for any signs of trauma if they sustained a fall.

## X-Ray

A chest radiograph is generally not helpful, but one should be obtained and compared to old films to look for electrode fracture, displacement of sensing electrodes, and patch migration or distortion.

## Electrocardiograms

An ECG should be obtained. Immediately after an AICD shock, the ECG often shows abnormalities such as ST-segment elevations or depressions. [60] [71] If these changes are due solely to the shock, they will resolve within 15 minutes. Otherwise, the ECG should be examined and compared to old studies for evidence of ischemia.

## Use of a Magnet for AICD Inactivation

The patient who is experiencing inappropriate AICD discharges in the ED can be treated by magnet inactivation of the device similar to the approach described earlier for the pacemaker patient.

### Technique

The method for inactivating an AICD device is outlined in Table 12-3 (Table Not Available) . The orientation of the device in the abdominal pocket should be determined, with the lead connections normally cephalad. A ring magnet is then placed over the corner adjacent to the lead connections (usually the upper righthand corner of the device) (Fig. 12-13) . A series of beeping tones will sound, which correspond to the sensed QRS complexes. In the absence of organized QRS activity, random beeps will sound. [69] When the magnet is left in place for 30 seconds, a continuous beep is heard. This indicates that the AICD is inactivated. The magnet should then be removed, and the AICD will remain inactivated. The AICD may be reactivated by applying the magnet for 30 seconds and removing it when the steady beep changes to intermittent beeping.

## Clinical Follow-up

The AICD patient who has component failure, such as patch migration or lead fracture or dislodgement; infection; vascular complications, such as thrombosis or perforation; or cardiac complications, such as perforation or pericarditis, require cardiology consultation for admission.

The AICD patient who received a single shock and had prodromal symptoms indicative of low cardiac output should be evaluated for myocardial infarction, electrolyte imbalance,

| TABLE 12-3 -- Method for Inactivation of AICD |
|:---|
| *From Munter DW, DeLacey WA: Automatic implantable cardioverter-defibrillators.* <br> *emerg Med Clin North Am 12:579-595, 1994. Used with permission.* |
| (Not Available) |

or drug toxicity, as well as for any sustained trauma. If findings of this evaluation are normal, the patient is usually released home after discussion with the cardiologist. [5]

The patient who received a single shock without related symptoms consistent with a ventricular tachydysrhythmia requires a more extensive evaluation including cardiac monitoring, determination of drug levels as appropriate, and electrolyte level measurement. [69] [70] [72] If the ED evaluation is normal, patients require cardiology consultation. The cardiologist normally interrogates the AICD using either phonogram or telemetry units. The decision to admit or release the patient with potential alterations of antiarrhythmic drug therapy is made based on the results of this testing.

Patients whose condition is unstable, who report more than one shock in succession or more than two single shocks in a 1-week period, or who have evidence of ischemia, electrolyte imbalance, or drug toxicity require admission to a monitored setting for further evaluation and continued monitoring.

## THE PACEMAKER/AICD PATIENT IN CARDIAC ARREST

Patients with pacemakers in cardiac arrest may require defibrillation or cardioversion, depending on their presentation. Although most pacemakers have circuitry that protects them from high current flows, a variety of pacemaker-related problems can develop from defibrillation or cardioversion. These include damage to circuitry resulting in complete destruction, decrease in output, or runaway pacing; acute or chronic increases in the pacing threshold, which is normally temporary; undersensing lasting up to 10 days; reprogramming; resetting to a different mode, usually asynchronous; lead displacement; and myocardial thermal or electrical burns at the electrode-myocardium interface leading to ventricular fibrillation. [29]

Due to these potential complications, Barold and colleagues have suggested guidelines for defibrillation and cardioversion in pacemaker patients. [29] The first of these is the use of anterior-posterior paddle position, if possible, preferably with specific anterior and posterior paddles. When this is not possible, the paddles should be placed along a line perpendicular to the line between the pulse generator and the tip of the ventricular lead. For a patient with a pacemaker generator in the left upper chest wall, the appropriate paddle placement would be left lateral chest wall and right midsternal border. For a patient with a pacemaker generator in the right upper chest wall, the appropriate paddle placement would be left upper chest wall and right lower chest wall. Second, the

paddles should be placed at least 10 cm from the pulse generator or lead. Third, because of the potential for damage to the pacemaker or leads, a transcutaneous pacemaker and pads should be readily available if needed, and standby cardiology consultation should be arranged in case emergent reprogramming is needed. Fourth, the patient must be admitted to the hospital and pacemaker functioning must be monitored carefully with repeated threshold testing.

Because of the lower current used by AICDs, the possibility of pacemaker damage from AICD discharge in patients with both is remote. [29]

The AICD patient who is in cardiopulmonary arrest may be managed in the same manner as patients without the device, with minor modifications. Cardiopulmonary resuscitation (CPR) and transthoracic defibrillation will not harm the AICD. [69] The AICD may spontaneously discharge without warning if the patient's rhythm meets the preset criteria for shocking. In the case of an AICD shock, medical personnel performing CPR may experience a mild electrical shock, [73] but to date no reports of injury to medical personnel from such a shock have been noted.

Some authors recommend leaving the AICD activated during cardiopulmonary arrest, [69] allowing it to discharge up to the maximum of four times allowed by the circuitry; but deactivation may be necessary to alleviate fears of medical personnel, to avoid AICD shock-induced arrhythmias, or if temporary cardiac pacing is needed. [5]

Defibrillation does not harm the device or circuitry, and may be performed with the usual technique, although a theoretical possibility exists that the epicardial patches can shield the myocardium from the delivered energy. For this reason, paddle placement over the apex and right sternum may be indicated for AICD patients with anterior and posterior myocardial patches, with anterior and posterior paddle placement in AICD patients with a single patch over the cardiac apex.

## CONCLUSION

Many patients have implanted pacemakers or AICDs. These patients have underlying cardiac disease and may present to the ED with a variety of complaints. Complications associated with these devices are not uncommon. The evaluation of these patients must be directed toward the potential complications. Pacemaker function may be evaluated by ECG and magnet testing, but most problems will require cardiology consultation and intervention. Runaway pacemaker represents a true emergency and must be dealt with expeditiously. AICD complications can be initially evaluated in the ED, but they normally require specialized interrogation devices to analyze the problem. Patients with pacemakers or AICDs in cardiopulmonary arrest require modifications of standard paddle placement for defibrillation or cardioversion, and if an AICD is present, it may need to be deactivated with a magnet.

# Chapter 14 - Emergency Transcutaneous Cardiac Pacing

**Scott A. Syverud**

**Jerris R. Hedges**

Transcutaneous cardiac pacing (TCP) is a rapid, minimally invasive method of treating severe bradycardias and asystole. Electrodes are applied to the skin of the anterior and posterior chest walls, and pacing is initiated with a portable pulse generator. In an emergency setting, this pacing technique is faster and easier to initiate than transvenous pacing. Pulse generators are sufficiently portable to be used in emergency departments, hospital wards, intensive care units, and mobile paramedic vehicles.

In 1872, Duchenne de Boulogne reported a successful resuscitation of a child by attaching 1 electrode to a limb while a second electrode was rhythmically touched to the precordium of the thorax. [1] Successful overdrive pacing of the human heart, using a precordial electrode, was reported by VonZiemssen in 1882. [2]

In 1952, Zoll introduced the first practical means of TCP. Using a ground electrode attached to the skin and a subcutaneous needle electrode over the precordium, he reported the successful resuscitation of 2 patients in ventricular standstill. [3] One patient was paced for 5 days and subsequently was discharged from the hospital. Zoll later introduced a machine that delivered impulses lasting 2 msec through 3-cm-diameter metal paddles pressed firmly against the anterior chest wall. This device was the first commercial transcutaneous cardiac pacemaker. During the 1950s, Zoll and Leatham demonstrated the effectiveness of TCP in patients with bradycardia and asystole. [4] [5] [6] [7] Leatham used larger electrodes (4 × 6 cm) and a longer pulse duration (20 msec) to successfully pace 2 patients with bradydysrhythmias. [7]

Until the late 1950s, TCP was the only clinically accepted method of cardiac pacing. The original technique using bare metal electrodes had adverse effects, including local tissue burns, muscle contraction, and severe pain. [3] [7] With the development of the first implantable pacemakers from 1958 through 1960 and the improvement of transvenous electrodes during the early 1960s, TCP was rapidly discarded. [8]

Refinements in electrode size and pulse characteristics have led to the reintroduction of TCP into clinical practice. [9] [10] Increasing the pulse duration from 2 to 20 msec or longer was found to decrease the current output required for cardiac capture. [11] [12] Longer impulse durations also make the induction of ventricular fibrillation less likely. [11] Larger surface area electrodes (8-cm diameter) decrease the current density at the underlying skin and therefore decrease pain and the possibility of tissue burns. [9]

## INDICATIONS AND CONTRAINDICATIONS

General indications for cardiac pacing are discussed in Chapter 13 . TCP is technically

the fastest and easiest method of emergency pacing. This technique is useful for initial stabilization of the patient in the emergency department who requires emergency pacing while arrangements for transvenous pacemaker insertion are being made. The use of the equipment is readily mastered, and the procedure is fast and minimally invasive. [10] [13] Refinements in equipment have made TCP the emergency pacing procedure of choice. TCP also is gaining widespread out-of-hospital use in helicopter ambulance programs and inhospital use in the cardiac catheterization laboratory, operating room, intensive care unit, and on general medical floors. [14] [15] [16] The technique may be preferable to transvenous pacing in patients who have received thrombolytic agents. No central venous puncture, with the attendant risk of hemorrhage, is required. Limited experience suggests that TCP also may be useful in the treatment of refractory tachydysrhythmias by overdrive pacing.  Although small pediatric electrodes for TCP have been developed, experience with pediatric TCP has been limited.

TCP is indicated for the treatment of hemodynamically significant bradydysrhythmias that have not responded to atropine therapy. *Hemodynamically significant* implies hypotension, anginal chest pain, pulmonary edema, or evidence of decreased cerebral perfusion. This technique is a *temporary* technique that is indicated for short intervals as a bridge until transvenous pacing can be initiated or until the underlying cause of the bradyarrhythmia (e.g., hyperkalemia, [13] drug overdose [24] ) can be reversed. Although often unsuccessful, TCP may be attempted in the treatment of asystolic cardiac arrest. In this setting the technique is efficacious only if used early after arrest onset (generally within 10 minutes). TCP is not indicated for treatment of prolonged arrest victims with a final morbid rhythm of asystole.

Delay from the onset of arrest to the initiation of pacing

is a major problem that limits the usefulness of TCP in out-of-hospital care. Hedges and colleagues reported that everyday availability of pacing increased the number of patients who received pacing within 10 minutes of hemodynamic decompensation and increased long-term patient survival as well. [28] Out-of-hospital pacing may be most useful in treatment of the patient with a hemodynamically significant bradycardia who has not yet progressed to cardiac arrest (e.g., heart block in the setting of acute myocardial infarction) or in the patient who arrests after the arrival of out-of-hospital providers. [27] [28]

In conscious patients with hemodynamically stable bradycardias, TCP may not be necessary. It is reasonable to attach electrodes to such patients and to leave the pacemaker in standby mode against the possibility of hemodynamic deterioration while further efforts at treatment of the patient's underlying disorder are being made. This approach has been used successfully in patients with new heart block in the setting of cardiac ischemia. [26] Generally when a transvenous pacemaker becomes available, transvenous pacing is preferred because of better patient tolerance.

## EQUIPMENT

Few medical product lines have changed as rapidly as commercial transcutaneous pacemakers. Patent controversy, corporate acquisitions, and rapid product evolution

have all contributed to this rapid change. [34] Of 8 commercial transcutaneous pacemakers reviewed in a 1988 product review, [35] 4 are no longer available. Several manufacturers no longer produce transcutaneous pacemakers or have gone out of business.

Despite this instability in the marketplace, transcutaneous pacemakers are now standard equipment in most emergency departments and are rapidly spreading to other inhospital and out-of-hospital care settings. The pacemakers introduced in the early 1980s tended to be asynchronous devices with a limited selection of rate and output parameters. Units introduced more recently have demand mode pacing and more output options and are more likely to be combined with a defibrillator in a single unit. Combined defibrillator-pacers offer advantages in both cost and ease and rapidity of use when compared with stand-alone devices. An example of 1 combined unit is shown in Fig. 14-1 (Figure Not Available) . A full-featured stand-alone pacemaker is illustrated in Fig. 14-2 (Figure Not Available) .

All transcutaneous pacemakers have similar basic features. Most allow operation in either a fixed rate (asynchronous) or a demand mode. Most allow rate selection in a range from 30 to 200 beats/min. Current output is usually adjustable from 0 to 150 mA. If an electrocardiography (ECG) monitor is not an integral part of the unit, *an output adapter to a separate monitor is required* to "blank" the large electrical spike from the pacemaker impulse and allow interpretation of the much smaller ECG complex. Without blanking protection, the standard ECG machine is swamped by the pacemaker spike and is uninterpretable. This could be disastrous, because the large pacing artifacts *can mask treatable ventricular fibrillation* (Fig. 14-3) . Pulse durations on available units vary from 20 to 40 msec and are not adjustable by the operator.

Two sets of patient electrodes are usually required for

**Figure 14-1** (Figure Not Available) Combined defibrillator-transcutaneous pacemaker unit (Zoll-PD). The unit defibrillates through standard hand-held paddles and has additional cable connections for electrocardiograph monitoring electrodes and for pacing electrodes. *(Courtesy of ZMI Corporation, Cambridge, Mass.)*

operation of the device. One set of standard ECG electrodes is used for monitoring. The much larger pacing electrodes deliver electrical impulses through an 8-cm-diameter conducting surface. One pacing electrode is placed over the mid-dorsal spine, and the other is placed over the left anterior chest. The posterior electrode serves as the ground.

One currently marketed combined defibrillator-pacemaker (Medac ALS System 4) uses a single set of electrodes for ECG monitoring, pacing, and defibrillation. Although this approach makes use of the device simpler, its clinical utility remains to be proved. Because the impedance characteristics of the ideal pacing electrode differ from those of the ideal ECG and defibrillation electrode, this approach may not prove to be ideal.

## TECHNIQUE

**Pad Placement**

The pacing electrodes are applied as shown in Figure 14-4 (Figure Not Available) and are attached to the instrument cable. The anterior electrode (cathode, or negative electrode) is placed as close as possible to the point of maximal impulse on the left anterior chest wall. This 8-cm-diameter electrode adheres to the skin and has a large surface area for electrical contact. The second electrode is placed directly posterior to the anterior electrode. Failure to capture may be due to misplacement of the electrodes, and failure to pace may be rectified with a small change in anterior electrode position. Electrocardiogram electrodes are placed on the chest wall and connected to the instrument cable. Some physicians prophylactically apply pacing electrodes to all critically ill patients with bradycardia to facilitate immediate TCP should decompensation occur.

**Figure 14-2** (Figure Not Available) "Stand alone" transcutaneous pacemaker (Zoll NTP). This unit has a built-in monitor and strip chart recorder. *(Courtesy of ZMI Corporation, Cambridge, Mass.)*

There is little risk of electrical injury to health care providers during TCP. Power delivered during each impulse is less than 1/1000 of that delivered during defibrillation. [36] Chest compressions (cardiopulmonary resuscitation) can be administered directly over the insulated electrodes while pacing. [29] Inadvertent contact with the active pacing surface results only in a mild shock.

**Pacing Bradycardic Rhythms**

To initiate TCP, the pacing electrodes are applied, and the device is activated. In the setting of bradyasystolic arrest, it is reasonable to turn the stimulating current to maximal output and then decrease the output as appropriate after capture is achieved. In a patient who has a hemodynamically compromising bradycardia but is not in cardiac arrest, the operator should slowly increase the output from the minimal setting until capture is achieved. Rate and output selections are adjustable (Fig. 14-5) . Generally a heart rate of 60 to 70 beats/min will maintain an adequate blood pressure (by blood pressure cuff or arterial catheter) or the desired degree of mentation.

Assessment of electrical capture can be made by monitoring the ECG on the filtered monitor of the pacing unit (Fig. 14-6) . Ideally, pacing should be continued at an output level just above the threshold of initial electrical capture. One study in 16 normal male volunteers who were paced without sedation noted cardiac capture at a mean current of 54 mA (range, 42 to 60 mA). [37] Most subjects could tolerate pacing at their capture threshold; only 1 subject required discontinuation of pacing at 60 mA because of intolerable pain. Heller and associates compared subjective pain perception and capture thresholds in 10 volunteers paced with 5 different transcutaneous pacers. [38] Capture rates (40 to 80%), thresholds (66.5 to 104 mA), and subjective discomfort varied from pacemaker to pacemaker.

Failure to capture with TCP may be related to electrode placement or patient size. Patients with barrel-shaped chests and large amounts of intrathoracic air conduct

electricity poorly and may prove refractory to capture. A recent thoracotomy has been found to nearly double the pacing threshold. [39] A large pericardial effusion or tamponade also will increase the output required for capture. [40] Failure to electrically capture with a transcutaneous device in these settings is an indication to consider immediate transvenous pacer placement.

Patients who are conscious or who regain consciousness during TCP will experience discomfort because of muscle contraction. [26] [37] [38] Analgesia with incremental doses of an opioid agent, sedation with a benzodiazepine compound, or both, will make this discomfort tolerable until transvenous pacing can be instituted.

**Overdrive Pacing**

Overdrive pacing [17] [18] [19] [20] [21] of ventricular tachycardia or paroxysmal supraventricular tachycardia is performed in patients who are stable enough to tolerate the brief delay associated with the necessary preparation for this technique. The patient is sedated as above, pacing and monitoring electrode pads are placed in the standard position as detailed earlier, and brief trains (6 to 10 beats) of asynchronous pacing are applied. The pacer rate must be set approximately 20 to 60 pulses/min greater than the dysrhythmia rate. [41] Generally, an impulse rate of 200 pulses/min is used for ventricular tachycardias (rate generally 150 to 180 beats/min), and a rate of 240 to 280 pulses/min is used for paroxysmal supraventricular tachycardias (rate commonly 200 to 250 beats/min).

For many commercial devices, the maximum device rate achievable is 120 pulses/min, thus prohibiting use of TCP for overdrive pacing. Interestingly, Altamura and colleagues have reported some success with "underdrive" pacing (i.e., TCP at a rate less than the tachydysrhythmia), although underdrive pacing seems less effective than the overdrive technique. [19]

Usually a current output considerably above the ventricular capture threshold is chosen to ensure that a wide area of myocardium is simultaneously depolarized. Generally a current output of 120 mA is used. After each train of impulses, the device is turned off, and the patient's cardiac rhythm is monitored. Generally no more than 3 trains of impulses are required. This technique may be combined with pharmacologic therapy.

Because rhythm acceleration is possible during overdrive pacing, it is essential that full resuscitation equipment, including a defibrillator, be available at the bedside.

## COMPLICATIONS

The major potential complication of TCP is *failure to recognize the presence of underlying treatable ventricular fibrillation*. This complication is primarily due to the size of the pacing artifact in the ECG screen, a technical problem inherent in systems without appropriate dampening circuitry.

A theoretical complication of TCP is the induction of dysrhythmias. In animal models

using epicardial electrodes, the threshold current required to induce ventricular fibrillation decreases as electrical impulse duration increases. At a 10-msec impulse duration, ventricular fibrillation can be induced with currents as low as 25 mA delivered through epicardial electrodes. [11] Because TCP impulses are of even longer duration (20 msec) and of higher current (50 to 200 mA), there has been concern about possible induction of ventricular fibrillation during TCP. Studies of fibrillation thresholds using large *precordial* electrodes have shown the relationship of threshold to impulse duration to be the opposite of that seen with *epicardial* electrodes. With *cutaneous* precordial electrodes, the current required to induce ventricular fibrillation *increases* as pulse duration *increases.* [12] The apparent paradox may be explained by the differing nature of the electrodes. Epicardial electrodes are localized over a small area of the myocardium, whereas transcutaneous

electrodes deliver a broad electrical charge to the myocardium as a whole. The implication is that longer impulse durations, although more dangerous with internal pacing, seem to decrease the chance of inducing ventricular fibrillation with TCP. Nonetheless, asynchronous TCP for tachydysrhythmias has been associated with rhythm acceleration and development of ventricular fibrillation. [19]

Experience with prolonged TCP in humans has not been extensive. Zoll and colleagues reported 25 humans paced for up to 108 hours with impulses of 20-msec duration. [6] Pacer-induced dysrhythmias did not occur. Leatham and associates paced 1 patient for 68 hours with impulses of 20-msec duration. [7] The patient died 2 days after pacing was discontinued. Pathologic examination revealed no evidence of pacer-induced myocardial damage. Madsen and colleagues paced 10 healthy volunteers at threshold for 30 minutes and found no enzyme or echocardiographic abnormalities. [42]

Studies of repetitive direct current countershocks in dogs have induced tissue damage with energy levels that are 1000 times greater than those required to pace the heart transcutaneously. In a canine study, 10 animals with chronic heart block that were paced for 60 minutes (20-msec duration at 80 beats/min with 8-cm-diameter cutaneous electrodes) did not develop pacer-related dysrhythmias. Serial cardiograms and cardiac enzymes revealed no evidence of ischemia or infarction. [43] Examination of the canine hearts after the dogs were sacrificed 72 hours after pacing did not reveal clinically significant myocardial damage. [44] A single primate paced for 1 hour with 20-msec impulses of 400 mA similarly had no evidence of tissue damage at autopsy and at microscopic examination after sacrifice 24 hours later. [45] Based on these studies, TCP appears unlikely to produce cardiac injury with short-term use in humans.

Soft tissue discomfort with the potential for injury may still occur with current transcutaneous pacemakers. One study with the Zoll transcutaneous pacemaker found only 2 of 30 subjects (patients and volunteers) who were paced while conscious required discontinuation of pacing owing to discomfort. [37] Most reported the discomfort as "mild or moderate and easily tolerable." Sedation would presumably improve a conscious patient's ability to tolerate TCP. Nonetheless, prolonged use may still induce local cutaneous injury; Pride and McKinley reported one 7-week-old child who was paced for 45 hours without a pad change and who developed third-degree burns.

## CONCLUSIONS

Devices that pace the heart have been available for clinical use since 1952. Technologic improvements have minimized the complications associated with earlier use of the transcutaneous route and have enabled the reapplication of this relatively old pacing technique to a selected subset of cardiac emergencies. The introduction of combined defibrillator-pacemakers promises to make pacing more available in out-of-hospital and health care settings. Although the technique is still not universally available, it is rapidly becoming the standard of care for resuscitation protocols and equipment. Pacing instituted earlier in the course of bradycardiac rhythms, including the out-of-hospital phase of care, may improve the poor survivial rate currently associated with these rhythms.

# Chapter 15 - Pericardiocentesis and Intracardiac Injections

**Richard J. Harper, Michael L. Callaham**

## Pericardiocentesis

Pericardiotomy under direct vision was first done in 1815, and in 1840, the first blind approach using a trocar was carried out successfully on a patient with tamponade from malignancy. [1] By the end of the 19th century, the trocar-and-cannula method of pericardiocentesis was commonly used. The subxiphoid approach was first described in 1911.

Blind, electrocardiography (ECG)-assisted pericardiocentesis has a significant morbidity rate, reportedly as high as 15 to 20%. [2] [3] For this reason ultrasound diagnosis of pericardial effusion with fluoroscopic or ultrasound guidance has become the norm for *elective* pericardiocentesis because of its lower (0.5 to 3.7%) [4] [5] incidence of complications. The ECG-assisted blind pericardiocentesis technique remains the standard procedure for truly *emergent* pericardiocentesis when a lengthy delay may be associated with obtaining and organizing ultrasound or fluoroscopic assistance.

### CAUSES OF PERICARDIAL EFFUSION AND TAMPONADE

The medical literature concerning pericardiocentesis tends to address two distinct categories of pericardial fluid collection: acute hemopericardium (largely secondary to trauma) and pericardial effusion from other causes. This separation is not entirely artificial, as these two clinical entities are quite different in their time course, etiology, and treatment.

#### Acute Hemopericardium

Acute hemopericardium has several causes, including coagulopathies, cardiovascular catastrophes, and acute injury resulting from either blunt or penetrating trauma. All of these causes result in rapid accumulation of whole blood in the pericardial sac. The fluid accumulates too fast for the relatively inelastic pericardial sac to stretch and accommodate the fluid. The result is cardiac tamponade produced by small fluid volumes and with an essentially normal pericardial size.

#### Penetrating Trauma

Traumatic tamponade due to penetrating trauma may result from obvious external injury such as knife or gunshot wounds, or it may be insidious, as seen with iatrogenic cardiac perforation during cardiac or vascular procedures.

In external penetrating trauma, tamponade is most commonly the result of a stab wound

to the heart, [6] presumably because the pericardium seals itself after a stab wound but cannot do so after gunshot wounds. [6] [7] Approximately 80 to 90% of stab wounds to the heart demonstrate tamponade, [6] [8] compared with 20% of gunshot wounds. Larger pericardial wounds from gunshots generally drain into the pleural space and produce a hemothorax. [9] Cardiac tamponade is often suspected with anterior chest wounds, but it is imperative to remember that any penetrating wound of the lateral chest, back, or upper abdomen also may involve the heart.

Iatrogenic causes of cardiac tamponade are relatively uncommon but well-known complications of invasive or diagnostic procedures. Pacemaker insertion (either transthoracic or transvenous) and cardiac catheterization, including valvuloplasty and angioplasty, are two of the main causes, resulting in tamponade when cardiac chambers or coronary vessels are inadvertently penetrated. [10] [11] [12] Such penetration of vascular structures is common during transthoracic pacemaker placement. [13] Tamponade is also seen as a complication after cardiac surgery, although it is usually anticipated, and

mediastinal or pericardial drainage helps to control and prevent it. [10] [14] Pericardiocentesis itself can cause tamponade by lacerating myocardium or coronary vessels. [15] [16]

Cardiac tamponade may result from perforation of the right atrium or, less commonly, of the right ventricle or superior vena cava by a central venous pressure (CVP) catheter or subclavian hemodialysis catheter. [17] This event is usually not diagnosed early and is therefore often fatal. [18] Perforation may occur during placement or, more commonly, 1 to 2 days later, when the catheter erodes through tissue, particularly if a catheter made of stiff material is used or when the left internal jugular vein approach is used. [19] Tamponade from CVP line placement is seldom seen in the emergency department but must always be considered when there is sudden decompensation in a patient with a CVP line in place. Tamponade should always be considered when a patient deteriorates hemodynamically after an invasive diagnostic or therapeutic procedure involving the heart.

**Blunt Trauma**

Blunt trauma may cause hemopericardium. Usually it occurs in the presence of a major chest injury with associated bruises or rib and sternal fractures. Cases have been reported, however, in which tamponade occurred in blunt trauma with no obvious signs of injury to the thorax. [20] Such incidents may be more common than is clinically recognized, judging by the reports of constrictive pericarditis and pericardial defects found months to years later in trauma patients who were not originally noted to have effusion. Pericardial effusion due to blunt trauma may also be a late finding, becoming symptomatic 12 to 15 days after trauma. [21]

Severe deceleration injury may cause tamponade as a result of aortic dissection. This appears to be an uncommon development, with two case series reporting tamponade in 3.6% (1 of 28 patients) and 2.3% (1 of 43 patients) of victims of aortic injury. [22]

Theoretically, cardiopulmonary resuscitation (CPR) can cause pericardial effusion secondary to the blunt trauma of chest compressions, broken ribs, or intracardiac injections. Early studies reported pericardial effusion in 1 to 3% of CPR survivors. [23] Echocardiographic studies showed small cardiac effusions (but not tamponade) in 12% of survivors, only 4% of whom had received intracardiac injections. [24] Thus, although case reports of tamponade exist, [25] [26] it would appear that CPR and intracardiac drug injections are unlikely to cause significant effusion, much less tamponade.

**Nontraumatic Hemopericardium**

Nontraumatic but acute hemopericardium caused by a bleeding diathesis, aortic dissection and ventricular rupture behaves much like traumatic tamponade because of its acute nature. These types of hemopericardium are less obvious in etiology than hemopericardium caused by external trauma.

Bleeding diathesis may cause spontaneous bleeding into the pericardial sac. The incidence of spontaneous pericardial tamponade in anticoagulated patients has been reported to range from 2.5 to 11%. [10] [27] Thrombolytic therapy has also been implicated in tamponade secondary to bleeding diathesis. Among 392 patients, only 4 (1%), all with large anterior myocardial infarctions, developed tamponade secondary to hemopericardium without ventricular rupture. [28]

A dissection of the ascending aorta may extend around the base of the aorta into the pericardial sac, causing dramatic, rapid, and often fatal tamponade. This pathologic abnormality may be due to conditions such as syphilis, Marfan syndrome, or atherosclerosis. Infection may create pseudoaneurysms of the aorta, which also can present as tamponade. [29]

Ventricular rupture after myocardial infarction is a common source of acute hemopericardium. Although the prognosis is often grim, survival is possible with prompt recognition and definitive treatment. [30] [31]

**Nonhemorrhagic Effusions**

The etiology of nonhemorrhagic pericardial effusions determines the aggressiveness, type, and speed of treatment required. Patients with nonhemorrhagic etiologies usually accumulate effusions slowly, which allows the pericardium to stretch and accommodate up to 2000 mL of fluid. [32] This slower accumulation, often over weeks to months, means that even in a moderately hypotensive patient, more time may be available for evaluation and treatment. [33] [34] In many cases of small nonhemorrhagic effusion, tamponade does not occur, and the effusion may resolve with treatment of the underlying disease or may be managed successfully by elective pericardiocentesis.

Many disease processes, ranging from the common to the rare (Table 15-1) , can cause pericardial effusion. The cause of nonhemorrhagic tamponade may not be obvious on examination in the emergency department, and tamponade is frequently misdiagnosed

as congestive heart failure or respiratory disease. Although neoplasm has generally been the most common underlying cause of nonhemorrhagic effusion, [35] [27] some reports [36] [37] have identified infectious complications of the human immunodeficiency virus (HIV) as a common etiology of large nonhemorrhagic pericardial effusion and tamponade (Table 15-2) .

HIV-related effusions have been ascribed to many opportunistic bacterial and viral infections, with mycobacterial infections being the most common. [36] [37] [38] Noninfectious pericardial effusions in HIV have been caused by Kaposi sarcoma and lymphoma. [39] [40]

Cancer is a prominent cause of nonhemorrhagic effusions; the pericardium is involved in 20% of patients with disseminated tumors [41] and 8% of all patients with cancer. [42] There is primary pericardial involvement in 69% of acute leukemias, in 64% of malignant melanomas, and in 24% of lymphomas; however, the incidence of actual tamponade in these malignancies is not known. Of metastases to the pericardium, 35% originate in the lung, 35% in the breast, 15% in lymphomas, and <3% in each of the other cancers. [42] Thus, any patient who is known to have one of these malignancies should be considered at risk for tamponade. Metastasis to the heart is usually a late finding in cancer, and other foci located elsewhere are usually evident. [43] Classic findings of tamponade, such as pulsus paradoxus, are frequently absent in cancer patients with tamponade, and their symptoms are usually attributed to their malignancy. [42]

Radiation pericarditis, particularly after treatment for Hodgkin's disease, is a common cause of effusion. [32] Effusion occurs in approximately 5% of those patients who receive 4000 rad to the heart.

Approximately 15 to 20% of patients on dialysis for renal failure develop pericarditis, and 35% of those with pericarditis develop tamponade. [44] [45] Up to 7% of patients on

chronic dialysis may have effusions, sometimes of 1 L or more. [45] Some series have reported tamponade in 34% of uremic patients who have effusions. [45] Pericardial effusion in renal failure may be managed with dialysis alone in many cases.

Thirty percent of myxedema patients may have pericardial effusions, but few have tamponade. [35] Most of the other

---

**TABLE 15-1** -- Causes of Pericardial Effusion
*Data from Guberman BA, Fowler NO, Engel PJ, et al: Cardiac tamponade in medical patients. Circulation 64:633, 1981; and Pories WJ, Caudiani VA: Cardiac tamponade. Surg Clin North Am 55:573, 1975.*

| | |
|---|---|
| Neoplasm | Mesothelioma<br>Lung<br>Breast<br>Melanoma<br>Lymphoma |
| Pericarditis | Radiation (especially after Hodgkin's disease)<br>Viral<br>Bacterial<br>*Staphylococcus*<br>*Peumococcus*<br>*Haemophilus*<br>Fungal<br>Tuberculosis<br>Amebiasis<br>Toxoplasmosis<br>Idiopathic |
| Connective tissue disease | Systemic lupus erythematosus<br>Scleroderma<br>Rheumatoid arthritis<br>Acute rheumatic fever |
| Metabolic disorders | Myxedema<br>Uremia<br>Cholesterol pericarditis<br>Bleeding diatheses |
| Cardiac disease | Acute myocardial infarction<br>Dissecting aortic aneurysm<br>Congestive heart failure<br>Coronary aneurysm |

| | | |
|---|---|---|
| Drugs | Hydralazine<br>Phenytoin<br>Anticoagulants<br>Procainamide<br>Minoxidil | |
| Trauma | Blunt<br>Major trauma<br>Closed-chest CPR<br>Penetrating<br>Major penetrating trauma<br>Intracardiac injections<br>Transthoracic and transvenous pacing wires<br>Pericardiocentesis<br>Cardiac catheterization<br>CVP catheter | |
| Miscellaneous | Serum sickness<br>Chylous effusion<br>Loffler syndrome<br>Reiter syndrome<br>Behcet syndrome<br>Pancreatitis<br>Postpericardiotomy<br>Amyloidosis<br>Ascites | |

| **TABLE 15-2** -- Etiology of Pericardial Effusion in Two Studies [*] | | |
|---|---|---|
| | **Krikorian[27]<br>(120 Patients)<br>(%)** | **Guberman[35]<br>(56 Patients)<br>(%)** |
| Neoplastic disease | -- | 32 |

| | | |
|---|---|---|
| Pericardial invasion | 16 | -- |
| Radiation pericarditis | 7.5 | 4 |
| Etiology uncertain | 18 | -- |
| Traumatic hemopericardium | 9 | -- |
| Hemopericardium, nontraumatic | 2.5 | -- |
| Rheumatic disease | 12 | 2 |
| Uremia/dialysis | 5 | 9 |
| Bacterial infection | 2.5 | 12.5 |
| Congestive heart failure | 1.5 | -- |
| Uncertain etiology | 12.5 | -- |
| Idiopathic pericarditis | 13.5 | 14 |
| Cardiac infarction | -- | -- |

| Iatrogenic diagnostic procedures | -- | 7.5 |
|---|---|---|
| Myxedema | -- | 4 |
| Aneurysm | -- | 4 |
| Anticoagulation and cardiac disease | -- | 11 |
| Postpericardiotomy | -- | 2 |

* Note: Various complications related to human immunodeficiency virus (HIV) infections are now probably the most common causes of large nonhemorrhagic pericardial effusions. Effusions related to bacterial, viral, and mycobacterial infections and Kaposi's sarcoma and lymphoma are common.

etiologies listed in Table 15-1 are isolated case reports, and their exact incidences have not been determined.

**Other Causes of Pericardial Tamponade**

An interesting but rare cause of cardiac tamponade is pneumopericardium. Pneumopericardium is most commonly seen with pneumothorax and pneumomediastinum as a complication of respiratory therapy in infants, but it may also occur from similar barotrauma in adults. [46] Pneumopericardium also occurs spontaneously in asthma, [47] after blunt chest injury, [48] and even after high-speed motorcycle rides. [49] Pneumopericardium is usually benign, but tension pneumopericardium has been reported as a cause of life-threatening tamponade after blunt chest trauma [50] and after pericardiectomy. [51] The appearance of life-threatening pneumopericardium and tamponade has also been described immediately [52] and 6 days after penetrating chest trauma. [53]

## PATHOPHYSIOLOGY OF TAMPONADE

The pericardium is a tough, leathery sac that encloses the heart. It normally contains about 25 to 35 mL of serous fluid. [54] The pericardium is not rapidly elastic, although it does demonstrate stress relaxation within minutes of increased intrapericardial pressure, providing a slight ability to accommodate sudden increases in fluid. [55] As fluid accumulates, the first 80 to 120 mL is easily accommodated without significantly affecting pericardial pressure (Fig. 15-1) (Figure Not Available) . [56] However, if an

additional 20 to 40 mL is rapidly accumulated, the intrapericardial pressure almost doubles, often leading to sudden decompensation. With chronic effusions

that develop over weeks to months, the pericardium lengthens circumferentially to a huge size and can accommodate liters of fluid.

Pericardial compliance varies considerably in different individuals and various disease states. This compliance helps to determine the pressure-volume response curve (Fig. 15-2) (Figure Not Available) . [54] The pressure-volume relationship demonstrates hysteresis; the withdrawal of a quantity of fluid drops the pressure more than the addition of the fluid raised it.

As pericardial fluid accumulates, the increased intrapericardial pressure is transmitted across the myocardial wall and causes compression of the atria and perhaps the vena cava and pulmonary veins. This reduces right ventricular filling in diastole, producing decreased stroke volume and cardiac output. [57] Pulse pressure narrows as reflex sympathetic stimulation increases. Severe tamponade is produced with intrapericardial pressures of 15 to 20 mm Hg. [58]

Because stroke volume is decreased, heart rate increases to maintain cardiac output. Sympathetic discharge causes both arterial and venous vasoconstriction. [58] [59] Vasoconstriction increases venous pressure, which helps to restore the normal venous-atrial and atrioventricular filling gradients. These compensatory mechanisms are often effective and may permit establishment of a new homeostasis with normal cardiac output.

In chronic effusion and in early tamponade, cardiac contractility is not affected, and myocardial perfusion is normal. [57] [60] [61] As pressure continues to increase, coronary perfusion pressure drops, so in its later stages, tamponade is an ischemic event for the heart. By the time hypotension is measurable, left ventricular blood flow has already decreased 37%. [62] For comparable degrees of hypotension, experimental animals in hemorrhagic shock have five times greater coronary blood flow than animals in cardiac tamponade. [62] Severe

**Figure 15-1** (Figure Not Available) Production of cardiac tamponade by injections of saline into the pericardial sac. Although pericardial space can acutely accommodate 80 to 120 mL of fluid without a significant increase in pericardial pressure, note steep increases in pressure and drop in blood pressure at about 200 mL of saline. Once critical volumes are reached, very small increases cause significant hemodynamic compromise. *(From Fowler NO: Physiology of cardiac tamponade and pulsus paradoxus. II: Physiological, circulatory, and pharmacological responses in cardiac tamponade. Mod Concepts Cardiovasc Dis 47:116, 1978. Reproduced by permission of the American Heart Association, Inc.)*

**Figure 15-2** (Figure Not Available) Relationship of intrapericardial pressure to volume of pericardial fluid. Note that pressure drops more rapidly when fluid is removed than when it accumulates. (From Pories W, Gaudiani V: Cardiac tamponade. Surg Clin North Am 55:573, 1975. Reproduced by permission.)

experimental tamponade is followed by large increases in creatine kinase MB and microscopic evidence of cardiac injury resulting from ischemia. [63]

As intrapericardial pressure continues to rise, the heart's compensatory mechanisms fail. Myocardial ischemia and perhaps lactic acidosis from poor tissue perfusion may be the triggering events that disrupt the uneasy equilibrium. [64] Atrial pressure rises rapidly (Fig. 15-3) (Figure Not Available) . The atria and pulmonary circulation, being at much lower pressure than the systemic arterial pressure, are more vulnerable to the rising intrapericardial pressure. A "pressure plateau" occurs in which right atrial pressure, right ventricular diastolic pressure, pulmonary artery diastolic pressure, and pulmonary capillary wedge pressure are virtually identical.

This equalization of pressures leads to the echocardiographic hallmark of tamponade: right ventricular collapse. At this point hypotension is severe, bradycardia is common, and pulseless electrical activity (PEA) may occur. Unless intrapericardial pressure is immediately decreased, pulmonary blood flow ceases, and cardiac arrest follows. [64]

Total blood volume affects cardiac compensation, and it is possible to encounter a "low-pressure" cardiac tamponade. [65] The hypovolemic patient with tamponade has a decreased venous pressure, which not only decreases cardiac output, but also may obscure the diagnosis, because *distended neck veins or an elevated CVP are not present*. In a patient with a chronic pericardial effusion, the onset of hypovolemia can lower filling pressure enough to precipitate tamponade, and conversely, providing additional volume may temporarily offset increased pericardial pressure.

Ventilation and blood $CO_2$ levels have significant effects on cardiac tamponade. This is of particular significance, because trauma and other patients with tamponade may also have respiratory impairment. Pericardial pressure decreases 3 to 6 mm Hg with a hypocarbia of 24 torr and increases 2 to 4 mm Hg when the $P_{CO_2}$ reaches 57 torr. [66] This degree of hypercarbia-induced pericardial pressure rise can decrease

**Figure 15-3** (Figure Not Available) Summary of physiologic changes in tamponade. RV, right ventricle. (From Shoemaker WC, Carey JS, Yao ST, et al: Hemodynamic monitoring for physiological evaluation, diagnosis, and therapy of acute hemopericardial tamponade from penetrating wounds. J Trauma 13:36, 1973; and Spodick D: Acute cardiac tamponade: Pathologic physiology, diagnosis, and management. Prog Cardiovasc Dis 10:65, 1967. Reproduced by permission.)

cardiac output by 25%. Similarly, fluctuations in intrapleural pressure induced by intermittent positive-pressure ventilation are transmitted to the pericardial space and can reduce cardiac output another 25%. [67] The clinical implications of these findings are that patients suspected of having tamponade should normally be allowed to breathe spontaneously under careful monitoring and should not be ventilated with positive pressure unless it is absolutely necessary, as their hemodynamic status may deteriorate precipitously.

## DIAGNOSIS OF CARDIAC TAMPONADE

### Patient Profile and Symptoms

The clinical diagnosis of pericardial effusion can be difficult. In contrast, pericardial

tamponade is a clinical diagnosis, but specific clinical signs are often inaccurate. Particularly in the setting of acute hemorrhagic tamponade, the time from the first signs of tamponade to full arrest may be brief, and a high level of suspicion to invoke ultrasound or invasive diagnostic testing must be maintained. [68]

Classic clinical findings have been described for tamponade. However, these findings are often clearly present only when the patient is in fulminant tamponade. Ideally, tamponade is diagnosed early, when the patient suffers no more than dyspnea, weakness, or sometimes right heart failure. A common pitfall is to attribute respiratory symptoms (e.g., dyspnea on exertion) to a more common condition such as heart failure or pulmonary pathology and to overlook pericardial effusion until the classic late signs (e.g., hypotension) appear. [69]

Acute pericardial tamponade may resemble tension pneumothorax, acute hemothorax, hypovolemia, pulmonary edema, or pulmonary embolism. Severe right ventricular contusion can mimic the findings of tamponade. [70] The patient is often agitated or panic stricken, confused, uncooperative, restless, cyanotic, diaphoretic, and acutely short of breath. In the late stages, the patient is moribund. Hypotension in the presence of severe cyanosis and distended neck veins is a helpful but late finding.

**Physical Signs**

The classic physical findings of tamponade were first characterized by Beck in 1935. He described two triads, one for acute and one for chronic compression. [71] The chronic compression triad consists of high CVP; ascites; and a small, quiet heart. The triad in acute compression consists of high CVP, decreased arterial pressure, and muffled heart sounds. Unfortunately, in most major trauma series, only about one third of patients demonstrate the complete acute triad, [64] [72] although almost 90% have one or more signs. [6] *The simultaneous occurrence of all three physical signs is a very late manifestation of tamponade and is usually seen only shortly before cardiac arrest* (see Fig. 15-3) (Figure Not Available) .

Careful hemodynamic monitoring reveals much earlier changes that indicate the progression of tamponade (Table 15-3) (Table Not Available) . [73] In grade I tamponade, cardiac output and arterial pressure are normal, but CVP and heart rate are increased. In grade II tamponade, blood pressure is normal or slightly decreased, CVP is increased, and tachycardia persists. In grade III tamponade, the classic findings of Beck's acute triad occur. Although this sequence represents the natural history of acute tamponade, the time course varies. Some patients are stable at a given stage for hours; others proceed to cardiac arrest within minutes. [64] [73] Unfortunately, not all patients with early tamponade respond with a predictable pattern of change in vital signs. Brown and coworkers found that 6 of 18 patients with tamponade, defined through right heart catheterization, responded to tamponade with elevated systolic blood pressure. [74] After pericardiocentesis, these patients had a marked reduction in systolic blood pressure accompanied by increased cardiac output. All of these patients had previously been hypertensive.

The measurement of pulsus paradoxus is a procedure in its own right (see Chapter 70) . Pulsus paradoxus is defined as an exaggeration of the normal inspiratory fall in blood pressure. [59] [72] A paradoxical pulse (pressure) is one of the classic physical signs of tamponade, but it is not pathognomonic. It is also caused by pulmonary emphysema, asthma, labored respirations, obesity, cardiac failure, constrictive pericarditis, pulmonary embolism, and cardiogenic shock. [6] [33] [64] Measuring the paradoxical pulse is difficult and time consuming, and any frightened, hypotensive patient with labored breathing can demonstrate this finding (Fig. 15-4) .

If the difference between inspiratory and expiratory systolic blood pressures is >12 mm Hg, the paradoxical pulse is abnormally high. [75] Most patients with proven tamponade will demonstrate a difference of 20 to 30 mm Hg or more during the respiratory cycle. [6] [33] [64] This may not be true of patients with very narrow pulse pressures (typical of grade III tamponade); they will have a "deceptively small" paradoxical pulse of 5 to 15 mm Hg. The decrease in magnitude of pulsus paradoxus with hypotension occurs because the paradoxical pulse is a function of actual pulse pressure, and the inspiratory systolic pressure may be below the level at which

---

**TABLE 15-3** -- Shoemaker System of Grading Cardiac Tamponade
*From Shoemaker WC, Carey SJ, Yao ST, et al: Hemodynamic monitoring for physiologic evaluation, diagnosis, and therapy of acute hemopericardial tamponade from penetrating wounds. J Trauma 13:36, 1973.*

(Not Available)

---

diastolic sounds disappear. [59] For this reason, the ratio of paradoxical pulse to pulse pressure is a more reliable measure. A paradoxical pulse >50% of the pulse pressure is abnormal. [59]

Pulsus paradoxus has been correlated with the amount of impairment of cardiac output by tamponade. In atraumatic patients, a 15% pulsus paradoxus in the face of relative hypotension was found in 97% of patients with moderate or severe tamponade and only 6% of patients with absent or mild tamponade. [75] A similar study of right ventricular diastolic collapse by echocardiography found that an abnormal pulsus paradoxus had a sensitivity of 79%, a specificity of 40%, a positive predictive value of 81%, and a negative predictive value of 40%. [76]

The absence of a paradoxical pulse does not rule out tamponade. Although the mean paradoxical pulse was 49 mm Hg in one series of nonhemorrhagic tamponade, [35] 23% of the patients had a paradoxical pulse of <20 mm Hg, and 1 patient had no measurable paradoxical pulse. An abnormal pulsus paradoxus has been reported to be absent in tamponade when there is an atrial septal defect, aortic insufficiency, localized collections of pericardial blood, or extreme tamponade with hypotension. [65] It may also be absent when left ventricular diastolic pressure is intrinsically elevated owing to poor left ventricular compliance. This was seen in one

**Figure 15-4** Normally systolic blood pressure drops slightly during inspiration. To measure pulsus paradoxus, the patient breathes normally while lying at a 45° angle. The blood pressure cuff is inflated well above systolic pressure and slowly deflated. When the pulse is first heard only during expiration, this is the upper value. The cuff is deflated until the pulse is heard during both inspiration and expiration, and this is the lower value. The difference in the two values is the amount of pulsus paradoxus. A difference of more than 12 mm Hg is abnormal.

half of uremic patients with tamponade. [45] [77] In traumatic tamponade, pulsus paradoxus is deemed unreliable. [6] [77] [78] [79] In one study, only 35% of trauma patients had an abnormal paradoxical pulse when elevated CVP and decreased heart sounds were present. [79] In another study of 197 traumatic cases, only 8.6% of the diagnoses of tamponade were made by finding an abnormal pulsus paradoxus. [80]

Although the absence of a paradoxical pulse rules against severe tamponade, it does not completely rule it out. Whether time is taken to determine pulsus paradoxus depends on the patient's status. If the patient is moribund or rapidly deteriorating, taking time to check this parameter is obviously a poor choice of priorities.

**Venous Distention**

Venous distention, reflecting increased CVP, is also a late sign in cardiac tamponade (see Fig. 15-3) (Figure Not Available) . It may be masked by venoconstriction as a result of vasopressors (e.g., dopamine), intrinsic sympathetic discharge, or hypovolemia. [33] [64] [73] [78] Neck vein distention may be obvious clinically, but the measured CVP is more reliable than the presence of venous distention. The CVP reading should take into account positive-pressure ventilation and the effects of a Valsalva maneuver. Most patients with significant tamponade will have a CVP of 12 to 14 cm $H_2$ O. [78] *Hypovolemia changes the intrapericardial pressure-volume curve in tamponade and will lower the CVP reading at any given stage in the tamponade process.*

Animal studies have documented that right atrial pressure can be normal in tamponade when hypovolemia is present. One case of low-pressure cardiac tamponade was reported in a patient with no jugular venous distention, no paradoxical pulse, and a right atrial pressure of 8 mm Hg. [65] Thus although the initial CVP reading is useful and diagnostic if grossly elevated (e.g., 20 to 30 cm $H_2$ O), [51] [78] a series of CVP readings looking for an upward trend is the most sensitive diagnostic tool. [78] A rising CVP, especially when there is persistent hypotension, is extremely suggestive of tamponade in the trauma patient. In the rare case of the hypovolemic patient in whom tamponade is

suspected but who demonstrates a low CVP, a fluid challenge will help clarify the situation and will also improve cardiac output at least temporarily. [65]

**Ancillary Testing**

Routine chest radiographs and ECGs may be useful in increasing the level of suspicion for pericardial effusion and

tamponade. Noninvasive diagnosis of effusion, however, must be made by computed tomography or, preferably, cardiac ultrasound.

### Chest Radiographs

Chest radiographs are not useful in the diagnosis of acute traumatic tamponade, because *the cardiac size and shape do not change acutely.* However, the radiographs may reveal hemothorax, bullet location, or even pneumopericardium.

In the nontrauma patient with chronic effusion, a chest film often reveals an enlarged, sac-like "water bottle" cardiac shadow. Unfortunately, it is difficult to differentiate pericardial from myocardial enlargement, and radiographs cannot be used to distinguish between simple pericardial effusion and tamponade. One finding that is useful in identifying effusion on the plain chest film is the epicardial fat pad sign. The water density space between the radiolucent epicardial fat and the mediastinal fat represents the pericardial tissues and is normally <2 mm. An increase in this width suggests pericardial fluid or thickening (Fig. 15-5) . This sign may be seen in 41% of upright lateral and 23% of frontal chest films in proven pericardial effusion. [81] The diagnostic value may be enhanced by using a supine rather than upright cross-table, lateral chest radiograph. Obtaining a supine lateral film increases the sensitivity of the epicardial fat pad sign from 31 to 51%. [82]

### Electrocardiograms

ECGs may suggest, but should *not* be used to diagnose, pericardial effusion or cardiac tamponade. Most ECG changes, such as PR-segment depression, low-voltage QRS complexes, and electrical alternans, have acceptable specificity but poor sensitivity for pericardial effusion or tamponade. [33] [38] Low voltage is defined as a QRS amplitude 5 mV in all limb leads (or a sum of the limb lead QRS amplitude 30 mV), and PR depression is defined as 1 mV in at least 1 lead other than aVR. In 1 study correlating the ECG with echocardiographic evaluation, ECG signs had an overall sensitivity of only 1 to 17% and a specificity of 89 to 100% for pericardial effusion. [33] Others have demonstrated significantly higher sensitivity, i.e., in the range of 32 to 68% for voltage criteria. [84] PR-segment depression is the most common ECG finding in pericardial tamponade, and low voltage is most commonly associated with a moderate to large effusion. It is important to note that none of the ECG findings differentiate tamponade from effusion.

Electrical alternans is caused by pendular motion of the heart within the pericardial sac.

[85] Alternans of the QRS complex has been seen in about 22% of medical tamponade cases [69] but in only 5% of cancer patients with tamponade. [42] Electrical alternans of both the P wave and the QRS complex (total electrical alternans) is a rare finding but when seen is thought to be pathognomonic of tamponade (Fig. 15-6) (Figure Not Available) . [33] [86] Alternans does not always appear in the standard ECG leads; a bipolar chest lead (Lewis lead) may be needed to detect it.

**Echocardiography**

Echocardiography is the best available tool for diagnosing pericardial effusion and has the further advantage of being noninvasive. [87] Echocardiography is very sensitive in the diagnosis of pericardial effusion and tamponade. [88] [89]

The disadvantages of echocardiography are that it requires ultrasound equipment and is dependent on a skilled operator who is specifically trained in echocardiography. Even when immediately available, echocardiography may take at least 5 minutes, which may be too much time for a patient who is deteriorating rapidly.

**Figure 15-5** Epicardial fat pad sign. The water density space between the radiolucent epicardial fat and the mediastinal fat represents the pericardium and its contents and should be 2 mm or less. An increase suggests pericardial fluid or thickening. *A,* Left anterior-oblique chest film. *B,* Lateral chest film. On acute tamponade, the chest radiograph has very minimal diagnostic value.

**Figure 15-6** (Figure Not Available) Overall, the ECG has a low sensitivity for pericardial effusion or tamponade, but PR depression, low voltage, or electrical alternans may be seen. Lewis lead electrocardiogram (ECG) showing total electrical alternans of both amplitude and configuration of P and QRS complexes. This is rarely seen but is almost pathognomonic of tamponade. Note that electrical alternans may not be evident in standard ECG leads. (From Sotolongo RP, Horton JD: Total electrical alternans in pericardial tamponade. Am Heart J 101:854, 1981. Reproduced by permission.)

The diagnosis of effusion is easily made by visualizing a large area of fluid, often best seen behind the heart. This view will also demonstrate the ventricular wall and quickly distinguishes the patient with a large effusion from the patient with congestive heart failure and a dilated ventricle. False-positive results may be obtained if a large amount of subepicardial fat is present. [90]

Effusion is not synonymous with tamponade, and the volume of fluid needed to produce tamponade in an individual depends on the thickness of the ventricular myocardium, the rate of fluid accumulation, and the distensibility of the pericardium. Echocardiographic diagnosis of tamponade is best made on the observation of right ventricular diastolic collapse. [91] [92] Other useful echocardiographic suggestions of tamponade are observation of the heart swinging rhythmically in the pericardial effusion, excessive respiratory variation in the size of the ventricles, greatly decreased right ventricular size, and pseudoprolapse of the mitral valve. [77]

Although right ventricular or atrial collapse has been touted to be highly specific and sensitive for tamponade, [76] when the definition of tamponade is based on clinical

criteria, the picture becomes less clear. Whereas echocardiography is very sensitive for tamponade, it may be nonspecific. [89] [93] [94] For example, in a group of 50 patients with an echocardiograph diagnostic for tamponade, 94% had a systolic blood pressure 100 mm Hg and 58% had a cardiac index 2.3 L/min/m [2] . [89] It is unclear whether the subset of patients with electrocardiographically "proven" tamponade but no clinical signs of tamponade have a natural history that differs from those with a large, nontamponade pericardial effusion. One author has suggested, "In some of these patients, when the etiology is known and the disease can be treated effectively with medication . . . pericardial drainage may not be necessary." [94]

If echocardiographic determination of tamponade is overly sensitive, do the echocardiographic signs associated with tamponade provide predictive value regarding outcome? In a retrospective evaluation of 187 hospitalized patients with pericardial effusion, the effusion size alone was found to have a strong predictive value for patient outcome when measured in terms of eventual tamponade or the combined end point of tamponade, surgical drainage, or pericardiocentesis. [95] The addition of criteria for tamponade, right atrial and ventricular collapse, and inferior vena cava (IVC) plethora added little, if any, prognostic value. Wall and coworkers, in an analysis of 57 patients with new, large pericardial effusions, found that these additional findings did not differentiate patients with and without tamponade, although cardiac tamponade occurred in almost 50% of patients, regardless of these findings. [96]

The risk of performing pericardiocentesis without echocardiographic determination of pericardial fluid was demonstrated by the Krikorian series. [27] Of patients with a clinical picture of tamponade, 17% actually had constrictive pericarditis, 16% had congestive heart failure and fluid overload, and 5% had obstruction of the superior vena cava. None of these patients could be expected to benefit from pericardiocentesis, and all were at risk for complications.

**Computed Tomography**

Although in most circumstances computed tomography (CT) is less readily available than echocardiography and requires that the patient be transported to the site of the CT equipment, CT is effective in defining the presence and extent of pericardial effusion in the stable patient. [97] In certain circumstances, CT can provide a more definitive diagnosis than echocardiography. In one series, eight equivocal echocardiograms were evaluated by CT. [98] Two patients thought to have pericardial effusion by ultrasound were found by CT to have pleural effusions. Another patient with pericardial effusion by ultrasound was found by CT to have an epicardial lipoma. Three loculated pleural effusions not seen by ultrasound were defined by CT. A final 2 patients had hemopericardium visualized by CT but not ultrasound. In circumstances where the patient is stable and ultrasound produces equivocal results or is not available, CT may provide a definitive diagnosis of pericardial effusion.

## INDICATIONS FOR PERICARDIOCENTESIS

There are two indications for pericardiocentesis: (1) to diagnose the cause or presence of a pericardial effusion and (2) to relieve tamponade. The former is an elective

procedure and ideally should be accomplished under ultrasound guidance. The latter may be semi-elective and performed with ultrasound guidance or emergent and performed blind with ECG assistance.

**Diagnostic Pericardiocentesis**

The use of pericardiocentesis for diagnosis of the etiology of nonhemorrhagic effusions is widespread, although opinions of its utility vary. [32] [99] [100] Neoplastic cells, blood, bacteria, viruses, and chyle can be sought. Measurement of pericardial fluid pH can be helpful, because inflammatory fluid is significantly more acidotic than noninflammatory fluid. [101] When a specific etiology is suspected, additional diagnostic testing may be useful (e.g., adenosine deaminase in tuberculosis, and carcinoembryonic antigen in suspected malignancy). [102]

The diagnostic accuracy of pericardiocentesis varies greatly from series to series, depending on the vigor with which a definitive etiology was sought and the prevalence of certain etiologies in the patient population under consideration. In one large series, fluid was obtained in 90% of the taps, but a specific etiologic diagnosis was obtained in only 24% of the fluid specimens. [27] Certain diagnoses are unlikely to be made from pericardial fluid. Pericardial fluid has been shown to give false-negative cytologic results in several cases of lymphoma and mesothelioma. [27] In HIV patients, effusions caused by Kaposi sarcoma and cytomegalovirus have been diagnosed by pericardial biopsy after fluid studies were nondiagnostic. [103] [104]

An alternative diagnostic tool is subxiphoid pericardiotomy. This technique, performed in the operating suite, obtains both fluid and a pericardial biopsy specimen. It is more likely to provide a definite diagnosis and has been performed safely without general anesthesia. [105] [106] In a prospective series of 57 patients, 36% obtained a definitive diagnosis; 40%, a probable diagnosis; 16%, a possible diagnosis; and 7% remained undiagnosed with subxiphoid pericardiotomy. [107] Although it is uncertain whether this technique is safer than ultrasound-guided pericardiocentesis, published reports show a low rate of complications. [107]

Regardless of technique, the need to sample small effusions or obtain pericardial tissue has been questioned. A prospective series found a diagnostic rate of 6% with pericardial fluid and 5% with pericardial tissue when a small persistent effusion was sampled for the specific purpose of diagnosis. In contrast, when patients from the same population had therapeutic intervention for tamponade, the yields from fluid and tissue were 54% and 22%, respectively. [100]

The use of pericardiocentesis as a diagnostic tool in traumatic tamponade is limited. When used diagnostically to determine the presence of pericardial bleeding in trauma, the procedure has a false-negative rate of between 20% and 40%. [78] [108] [109] [110] The reason for the high false-negative rate (defined as no blood aspirated) is well demonstrated by typical stab wounds of the heart. [8] [111] Ninety-six percent of the patients had blood in the pericardium, but it was clotted in 41% of the patients and partially clotted in another 24%. In only 19% was the blood completely fluid and thus

capable of giving a true-positive result on pericardiocentesis.

**Therapeutic Pericardiocentesis**

### Tamponade of Uncertain Etiology

The most common reason for performing pericardiocentesis in the emergency department (ED) is as part of the treatment for cardiac arrest or in peri-arrest situations. In particular, the presentation of pulseless electrical activity (PEA) with elevated jugular venous pressure should cause immediate consideration of pericardiocentesis. In this setting, blind, ECG-guided pericardiocentesis can be life-saving. However, the overwhelming majority of patients with PEA have neither significant effusion nor tamponade, and other etiologies for the PEA also should be sought. Pericardiocentesis also may be considered in other presentations of effusion with existing or incipient tamponade.

### Tamponade Caused by Nonhemorrhagic Effusions

Pericardiocentesis is often, at least temporarily, therapeutic in cardiac tamponade. Most nonhemorrhagic effusions are liquids that can be drained easily through a small needle. Removal of even a small amount of fluid can immediately and dramatically improve blood pressure and cardiac output. Pericardiocentesis relieves tamponade due to nonhemorrhagic effusions in 60 to 90% of cases. [35] [27] [42] Patients in whom it fails often have purulent pericarditis or malignant invasion of the pericardium.

Pericardiocentesis without catheter placement may be much less useful for long-term management of these patients; 26% of the patients in the study by Guberman and coworkers eventually required pericardial resection. [35] In Krikorian's series, 24% of the patients were managed successfully with one pericardiocentesis, 37% needed multiple taps or an indwelling catheter, and 39% required surgical drainage. [27] Fifty-five percent of the last group had traumatic hemopericardium.

Patients with renal failure and pericardial effusion may be better managed by dialysis than pericardiocentesis. In one series, 63% of these patients were successfully managed with dialysis alone, and only 6% needed surgical treatment over the long term. [27] Tamponade is less frequent with pericarditis when it occurs within the first months of dialysis, and such patients are much more likely to be successfully managed without invasive intervention. [44] When invasive treatment is needed for dialysis patients, pericardiocentesis is probably a poor choice; 9 of 10 patients who received it had complications in one series, and it was the only invasive treatment that resulted in death. [44]

An algorithm for the urgent management of nonhemorrhagic cardiac tamponade is shown in Figure 15-7 .

### Use in Hemorrhagic Tamponade

*Pericardiocentesis is never the definitive treatment in hemorrhagic tamponade.* [112] [113] Although aspiration of a small quantity of fluid may cause dramatic improvement, blood usually reaccumulates. [33] [86] Thus, patients with pericardial hemorrhage ultimately require thoracotomy to explore and repair the cardiac injury.

One of the greatest potential drawbacks of pericardiocentesis in traumatic tamponade is that it may delay thoracotomy. In one study of 25 trauma patients with cardiac injury, [112] all of those who were operated on within 2 hours of injury survived, regardless of age or type of wound. With greater delay, none survived. Sugg and colleagues, in a study of 459 similar patients, found a mortality rate of 43% when pericardiocentesis was the sole treatment, but only 16% when surgery was performed. [109] Most investigators agree that with early thoracotomy and little or no reliance on pericardiocentesis, the number of deaths due to stab wounds has decreased. [78] [110] [113] [114] Sugg and associates reported that 10 of 18 patients with traumatic tamponade who were managed by repeated pericardiocentesis alone died within 1 to 2 hours. [109] At autopsy, all patients had completely repairable wounds.

Nonetheless, while other temporizing treatments are instituted (see discussion below) and arrangements for definitive surgical treatment are being made, pericardiocentesis may temporarily improve the patient's hemodynamic situation (Fig. 15-8) . Some clinical evidence supports the usefulness of pericardiocentesis as a temporizing measure. In a study of 174 patients with tamponade from penetrating trauma, 96 underwent operating room thoracotomy, 44 underwent emergency department thoracotomy, and 34 received

**Figure 15-7** Management of *nontraumatic* cardiac tamponade. IV, intravenous line; CVP, central venous pressure; ECG, electrocardiogram.

only pericardiocentesis followed by observation. [80] Of those who underwent operating room thoracotomy, 68% were hemodynamically unstable, and preoperative pericardiocentesis decreased the mortality rate from 25 to 11%. Ninety-one percent of those who underwent emergency department thoracotomy were unstable, and pre-thoracotomy pericardiocentesis decreased the mortality rate from 94 to 63%. For the unconscious and hypotensive or agonal patient,

**Figure 15-8** Management of *traumatic* cardiac tamponade. IV, intravenous lines; CVP, central venous pressure; ECG, electrocardiogram; R/O, rule out.

emergency thoracotomy is the preferred treatment (see Chapter 17) .

When a trauma patient's condition is relatively stable, but a high level of suspicion for a penetrating cardiac wound is present, an alternative to thoracotomy is the subxiphoid pericardial window. [105] [115] [116] The procedure has been done under local anesthesia, and although it is possible to perform the procedure in the ED, [116] most authors believe the procedure should be reserved for the operating suite. [117] [118] [119]

## CONTRAINDICATIONS

There is no absolute contraindication to pericardiocentesis. It should not be performed when better treatment modalities are immediately available (e.g., dialysis for uremic patients and immediate surgery for trauma patients). For diagnostic or nonemergency pericardiocentesis, the absence of echocardiographic or CT diagnosis is a relative contraindication, because the complication rate increases dramatically under such circumstances. If ultrasound or fluoroscopic guidance is available, it should be used in nonemergent situations.

## EQUIPMENT FOR PERICARDIOCENTESIS

### Fluoroscopic or Ultrasound Guidance

Pericardiocentesis is ideally performed in the cardiac catheter laboratory under fluoroscopic guidance. In the emergency department, as well as elsewhere in the hospital, echocardiography is useful for directing pericardiocentesis. With ultrasound, the area of the heart with the greatest fluid accumulation can be accurately identified and its relationship to the body wall clarified. [120] [121] An entry site and angle of penetration can then be chosen that have the greatest likelihood of obtaining fluid while simultaneously avoiding vital structures. Ultrasound also can be used to identify when the needle tip enters the pericardial space, although visualizing the needle with ultrasound can be difficult. [122] Specially made guides are available to allow visualization of the needle during the procedure and reduce complications. [123] Without

**Figure 15-9** Equipment for emergent pericardiocentesis: long, 18-ga spinal needle; wire with alligator clips for connection to the electrocardiograph machine; and syringe (three-way stopcock optional). Sterile skin preparation and local anesthetic are also required.

**Figure 15-10** An example of the contents of a prepackaged pericardiocentesis set: finder needle, Seldinger wire, dilator, catheter guide, and pigtail catheter. Sterile skin preparation and local anesthetic are also required.

ultrasound or fluoroscopy, pericardiocentesis is blind, and vital structures are vulnerable to injury. These techniques are beyond the scope of this text, and the clinician should enlist the assistance of a skilled imager when using them.

### Electrocardiographic Assistance

Although the procedure can be performed with only a syringe and a spinal needle, ECG monitoring is desirable. An alligator clamp is used to connect the needle to *any* of the precordial leads (V leads) of a properly grounded ECG device (Fig. 15-9) . Generally the V lead (usually $V_1$ or $V_5$ ), which permits a continuous display during rhythm monitoring,

is used.

**Other Equipment**

The traditional needle choice has been a 7.5- to 12.5-cm (3- to 5-inch), 18-ga spinal needle with an obturator. It is best to leave the obturator in the needle during initial passage through the skin to avoid obstruction of the needle lumen. More recently, the shorter Teflon-sheathed Intracath needle has been used. Alternatively, the clinician can use the guide wire (Seldinger) technique, inserting a plastic catheter over a flexible guide or J wire. With this technique, an 18-ga, thin-walled needle is used for placement of the wire. The catheter (after removal of the accompanying introducer) may be left in place for prolonged drainage, if needed. [124] [125]

For drainage of blood, pus, or other viscous effusions, a large catheter such as a No. 7 to 9 Fr Cordis sheath should be inserted. [126] Alternatively, the guide wire technique can be used to insert a radiopaque, 16-ga, flexible, fenestrated, central venous catheter, which can then be connected to closed suction drainage and left in place for long periods of time. [127] Pigtail catheters with side and end holes or nephrostomy drainage catheters also can be used. [125] Multilumen catheter patency can be maintained by slow continuous flush with heparinized saline solution. [125] Complete "sets" containing necessary equipment for placing a catheter using the guide wire technique are commercially available (Fig. 15-10) , including sets designed for pediatric use. [128]

A three-way stopcock may be attached to the needle or catheter to allow removal of more than one filled syringe without much movement of the needle. The continuous motion of the heart may require minor changes in needle or catheter position during the procedure. Lengthy or repeat drainage is much safer if the steel needle is withdrawn and a plastic catheter is left in place.

## PROCEDURE

**Temporizing Measures**

While preparing for pericardiocentesis in the unstable patient or attempting to stabilize the patient while the operating suite is readied for thoracotomy or subxiphoid pericardiotomy, temporizing measures should be considered. In the patient with suspected tamponade and without jugular venous distention, the administration of a fluid bolus may improve hemodynamics. [65] In the setting of nonpenetrating tamponade, a fluid challenge has been recommended [56] [129] ; animal experiments have found this to be beneficial, with or without nitroprusside for afterload reduction. [130] However, a follow-up prospective evaluation in patients with tamponade found no benefit from either fluid challenge or nitroprusside; cardiac output remained unchanged at a mean of 5.1 L/min, in contrast to 9.1 L/min after pericardiocentesis. [131] In the trauma patient with penetrating cardiac injury, fluid resuscitation may produce improvement or deterioration. Animal experiments indicate that the response depends on whether fluid infusion produces recurrent bleeding from the cardiac wound. [132] One report found that dextran solution for volume expansion produced significant hemodynamic improvement in patients with subacute ventricular free wall rupture after acute myocardial infarction. [30]

In summary, judicious volume expansion may produce temporary beneficial hemodynamic results, but this is not uniformly true.

Pressors also have been recommended as a temporizing measure in tamponade. Dopamine, dobutamine, norepinephrine, and isoproterenol have been evaluated. Norepinephrine produced increased cardiac output in animal models of tamponade [133] [134] but failed to increase cardiac output in patients with malignant effusion. [133] Isoproterenol produced increased cardiac output in animal models but detrimentally affected cardiac blood flow. [133] Both dopamine and dobutamine have produced increased cardiac output and other improvements in hemodynamics in the setting of tamponade. [20] [134] Either of these agents may be helpful as a temporizing agent in tamponade, but dobutamine may be preferable on theoretical grounds because of its greater beta activity. [134]

**Preparation**

All necessary equipment must be checked and laid out in advance. Full resuscitation equipment must be on hand, including a defibrillator. The patient must have an IV line in place and be attached to a cardiac monitor. The nonemergency patient may require sedation, but in an emergency, pericardiocentesis is usually performed on patients who are already obtunded or unresponsive as a result of low cardiac output. Use of sedation in these patients not only is unnecessary, but also carries a high risk of hemodynamic or respiratory deterioration. Premedication of the patient with atropine may help to prevent vasovagal reactions. When possible, the presence of pericardial effusion and the optimal anatomic approach should be determined in advance by echocardiography. If surgery may be needed, preparations should already be under way to ensure prompt availability of both an operating room and a surgeon.

If the patient's clinical condition permits, the chest should be elevated at a 45° angle to bring the heart closer to the anterior chest wall. If the abdomen is distended because of gastric contents or previous positive-pressure ventilation, a nasogastric tube should be used to decompress the stomach. The entire lower xiphoid and epigastric area should be carefully prepared with 10% povidone-iodine solution and sterilely draped, if time permits.

If the patient is awake, the skin and the proposed route of the pericardial needle should be anesthetized by infiltration with 1% plain lidocaine or 0.5% bupivacaine. Note that the pericardium is very sensitive and should be anesthetized in patients who are awake. [124]

**Anatomic Approach**

The choice of anatomic approach in the past has been governed largely by conjecture and theory, not by actual study of patients with pericardial effusion. Traditionally the subxiphoid approach was preferred and is widely touted in most texts and articles as by far the optimal choice. However, two-dimensional echocardiography allows direct visualization in the individual patient of both the areas of maximal effusion and the location of vital structures, and studies of echocardiography-directed pericardiocentesis have found that the intercostal space near the heart apex is usually the best site for

puncture, not the traditional subxiphoid approach. [121] [126] Careful cadaver studies have corroborated this finding, demonstrating greater safety with a parasternal approach in the fifth intercostal space and showing that the greatest number of injuries (usually to the right atrium) occurred with any variant of the subxiphoid approach. [13] In contrast, studies of intracardiac injection using the same routes have found an increased incidence of pneumothorax when parasternal or intracostal approaches are used (see discussion of complications of pericardiocentesis). This risk may increase with underlying lung disease. Whenever time and the patient's condition permit, clinicians should rely on echocardiography to define the extent of and optimal approach to pericardial effusion. When time or circumstances prevent the use of ultrasound, the clinician should use the approach with which he or she is most familiar.

**Parasternal Approach**

In this approach, the needle is inserted perpendicular to the skin in the left fifth intercostal space medial to the border of cardiac dullness (Fig. 15-11) . Older texts identify the puncture site as being at least 3 to 4 cm lateral to the sternal border to avoid the internal mammary artery. However, anatomic studies indicate that penetration immediately lateral to the sternum is less likely to cause this complication. [13]

**Subxiphoid Approach**

In the traditional subxiphoid approach, the needle is inserted between the xiphoid process and the left costal margin at a 30° to 45° angle to the skin (Fig. 15-12) . Because the heart

**Figure 15-11** Parasternal approach for pericardiocentesis. The patient is depicted in a supine position, although a preferable position would be sitting at a 45° angle, if the patient's clinical condition permits.

is an anterior structure, an angle >45° may intercept the liver or stomach. In this approach, the needle enters the pericardium at the angle at which it becomes the diaphragmatic pericardium. Recommendations as to where to aim the needle vary from the right shoulder to the left shoulder and all points in between, including the sternal notch. [124] [129] The only anatomic study that has been done demonstrated that in fact, the subxiphoid approach is likely to injure the thin-walled

**Figure 15-12** *A* and *B*, Xiphosternal approach for pericardiocentesis. The needle is aimed for the sternal notch or the left shoulder. Note the electrocardiography monitor. Although the patient is shown in a supine position, a preferable position would be sitting at a 45° angle, if the patient's condition permits. This general approach is also used for intracardiac injection of advanced cardiac life support drugs.

right atrium when one aims for the right shoulder. [13] Aiming for the left shoulder directs the needle toward either the left ventricle or the anterior wall of the right ventricle (Fig. 15-13) (Figure Not Available) .

**Apical Approach**

In the less commonly used apical approach, the needle is inserted 1 cm outside the apex beat in the intercostal space below the apex beat and within the area of cardiac dullness, and aimed toward the right shoulder. [124] If the apex cannot be palpated, the needle is inserted just inside the area of cardiac dullness. This area is close to the lingula and left pleural space, and pneumothorax is more frequent; a concomitant pleural effusion may be inadvertently tapped. In theory, this technique is used because the coronary vessels are small at the apex, and if a ventricle is entered, it is the thick-walled left ventricle, which is more likely to seal off any ventricular injury. Data are insufficient to say whether these theoretical advantages are important.

**ECG Monitoring**

After the skin has been punctured but before the pericardial needle is advanced, any obturator in the needle is removed, and an aspirating syringe is attached. At this time, ECG monitoring is begun by attaching a sterile cord with alligator clips (see Fig. 15-9) from the pericardial needle to any precordial lead (V lead) of the ECG machine. The V lead is then recorded, as the needle becomes an "exploring electrode." The machine must be properly tested and internally grounded. Small current leaks can induce dysrhythmias. [33] The purpose of the ECG monitoring is to prevent ventricular puncture. When the needle touches the epicardium, a current-of-injury pattern is noted on the ECG (Fig. 15-14) . This current of injury may be local and could be missed if a lead other than a V lead is monitored or if a cardiac monitor (which has a lower frequency response than the ECG machine) is used. Usually one notes ST-segment elevation on contact with the heart or pericardium in the absence of an effusion, but a premature contraction or other ventricular dysrhythmia also may be induced by direct mechanical stimulation of the ventricular epicardium by the needle. Contact with the atrium can cause atrial dysrhythmias, marked elevation of the PR segment, or atrioventricular dissociation. [15] If there is abnormal myocardial scarring secondary to infarction or other diseases or if there is malignant infiltration of myocardium, no current of injury may be generated. [16] Thus, ECG monitoring is not infallible in preventing myocardial penetration. In addition, the incessant motion of the heart makes it almost impossible to merely *touch* the epicardium.

With constant ECG monitoring, the operator slowly advances the needle and syringe while gently aspirating with the syringe. The needle will penetrate the pericardium (a barrier whose penetration usually cannot be palpated) at about 6 to 8 cm below the skin in adults and 5 cm or less below the skin in children. [54] The patient who is awake may complain of sharp chest pain as the sensitive pericardium is entered. When pericardial content is aspirated, the needle should not be advanced further. If a current of injury is noted (see Fig. 15-14) , the needle is touching epicardium and can easily lacerate myocardium or coronary vessels. The needle should be withdrawn a few millimeters until the

**Figure 15-13** (Figure Not Available) During "blind" pericardiocentesis, the subxiphoid approach is recommended. A short needle (16- or 18-ga) is inserted into the left xiphocostal angle perpendicular to

the skin and 3 to 4 mm below the left costal margin *(A)*. After advancing the needle to the inner aspect of the rib cage, the needle's hub is depressed so that the needle points toward the patient's left shoulder. The needle is then cautiously advanced about 5 to 10 mm until fluid is reached *(B)*. The fingers may sense a distinct "give" when the needle penetrates the parietal pericardium. Successful removal of fluid confirms the needle's position. The syringe is then disconnected from the needle, and the flexible tip of the guide wire is advanced into the pericardial space under fluoroscopic guidance *(C)*. The needle is withdrawn and replaced with a soft, multihole pigtail catheter (No. 6 to 8 Fr) using the Seldinger technique. After dilating the needle tract, the catheter is advanced over the guide wire into the pericardial space *(D)*. Once the catheter is properly positioned, aspiration of fluid should result in rapid improvement in blood pressure and cardiac output, a decrease in atrial and pericardial pressures, and a decrease in the degree of any paradoxical pulse *(E)*. Electrical alternans, if present, also decreases or disappears. (From Spodick DH: The technique of pericardiocentesis. J Crit Illness 2:91-96, 1987.)

**Figure 15-14** Current of injury. There is an obvious change in the electrocardiogram when the pericardiocentesis needle touches the epicardium. Following slight withdrawal *(arrow)*, the ST-segment elevation diminishes.

current of injury disappears. At this point, the needle should be safely positioned in the pericardial space, although heart motion may quickly bring it back into contact with the myocardium. This is particularly a risk if the presence of a large effusion has not been demonstrated by ultrasound.

**Fluid Aspiration and Evaluation**

An attempt is then made to drain pericardial fluid or blood. If blood is obtained, the possibility of cardiac puncture should be entertained. If fluoroscopy is available, the injection of a small amount of contrast will quickly disclose intracardiac placement. In other circumstances, the needle may need to be repositioned and the aspirate reexamined. Laboratory tests may help distinguish circulatory blood from hemorrhagic pericardial fluid. The latter should have a lower hematocrit measurement than venous blood. Substantially different hematocrit values rule out the possibility that the needle was in a cardiac chamber. Hemorrhagic pericardial fluid usually is about 0.10 pH unit more acid than simultaneously obtained arterial blood. [101] Bloody pericardial fluid may clot, particularly in traumatic situations in which bleeding is brisk, so clotting of the aspirated blood does not eliminate the possibility of a pericardial source. Nonclotting blood is indicative of defibrinated pericardial blood. Practically, however, there is rarely time for such analysis.

If an indwelling catheter is to be placed, a guide wire should be advanced through the needle (see Fig. 15-13) (Figure Not Available) . A dilator is passed over the needle to expand the needle tract. The guide wire should be maintained in sight and stabilized at all times. If intracardiac placement of the needle or guide wire is suspected, positioning must be verified by ultrasound or fluoroscopy or by using the techniques described above *before* the needle tract is dilated. Once the tract has been dilated, the pigtail catheter is placed over the guide wire. If the dilator is not used, particularly with the subxiphoid approach, the pigtail catheter tip may hang in the subcutaneous (SQ) tissue, making placement difficult.

After the catheter is placed, or if a decision is made to do a single aspiration, as much

fluid as possible should be aspirated from the pericardium. The removal of even 30 to 50 mL may result in marked clinical improvement in patients with tamponade. The catheter may be placed for continuous or intermittent drainage. A chest film should be obtained after the procedure to rule out iatrogenic pneumothorax. Patients should be monitored closely for 24 hours for signs of reaccumulating fluid or iatrogenic complications from the procedure. Repeat ultrasound examination is wise. Diagnostic evaluation of nonhemorrhagic fluid is similar to the analysis of pleural fluid (see Chapter 8) .

## COMPLICATIONS

The failure of pericardiocentesis to yield fluid ("dry tap") may be considered a complication, as the procedure has failed to achieve its desired result. If a dry tap is considered a complication, it is by far the most frequent one. In addition, the pericardial needle can injure any organ within its reach, causing pneumothorax or myocardial or coronary vessel laceration and, thus, hemopericardium. [114] Venous air embolism may be caused by air entering the heart. [135] The pericardial needle can also induce dysrhythmias from direct irritation of the epicardium or from small currents leaking from the connected ECG machine. [2]

Assessing the frequency of complication from pericardiocentesis is not as simple as it might appear. Changes in diagnosis of effusion by ultrasound and guidance of the procedure by ultrasound or fluoroscopy have greatly reduced the likelihood of complication. [100] [126] For example, Wong

and colleagues reported that most complications occurred in patients who were found retrospectively to have no effusion at all. [3] Unfortunately, ultrasound remains highly operator dependent, and the chances of misadventure are in large part dependent on operator skill. The procedure is also often performed on a moribund patient, and distinguishing between a poor outcome resulting from a poorly performed procedure and a poor outcome resulting from the underlying condition can be difficult.

Six studies summarized in Table 15-4 demonstrate that the risks of pericardiocentesis remain quite significant. The major complications will be discussed individually.

### Cardiac Arrest and Death

Cardiac arrest and death occurred in approximately 2% of patients in the larger series listed in Table 15-4 , but in none of a series of patients whose pericardiocentesis was directed solely by ultrasound. However, an exact causal relationship between pericardiocentesis and sudden death is difficult to substantiate. For example, in one series of 52 patients, the only death occurred in a patient in cardiogenic shock who had a nonproductive pericardiocentesis and who, on postmortem examination, had severe arteriosclerotic heart disease, not tamponade. [3] An additional case of cardiac arrest (successfully resuscitated) in this series was in a patient with a nonproductive pericardiocentesis; the cause of the arrest was not discussed. [3]

In two studies comprising 197 patients who underwent pericardiocentesis under echocardiographic guidance, there have been no cardiac arrests or deaths. [100] [126] In a series of 352 pericardiocenteses performed under fluoroscopic guidance, only two deaths resulted. [4] Ultrasound or CT confirmation of effusion was used in all but 15 cases. The two deaths occurred during or after the procedure, but whether they should be attributed to the procedure is unclear. One patient with aortic rupture penetrating into the pericardial space died of cardiac arrest immediately after the puncture. The other death, in a postmyocardial infarction patient with left ventricular aneurysm, was due to ventricular fibrillation that occurred about 15 minutes after the procedure.

**Cardiac Chamber, Vessel, or Lung Laceration**

Cardiac chamber, vessel, or lung laceration occurs in 6 to 9% of patients, even in the hands of experienced physicians under controlled situations. Nonfatal cardiac puncture, pneumothorax, and pneumoperitoneum have been reported, [45] as well as suppurative costochondritis. Most cardiac perforations occur in the right ventricle, but left ventricular [4] as well as atrial punctures have been reported. [15]

In Krikorian and Hancock's series, 13 of 123 patients developed hemopericardium as a result of pericardiocentesis, 1 as a result of a lacerated coronary artery. [27] One patient died from a punctured ventricle. Surgical control was necessary for 4 patients who developed tamponade, whereas 8 patients with hemopericardium did not develop tamponade and were managed conservatively. Several cases of induced tamponade occurred in patients with platelet counts >50 × 10 [9] /L.

Guberman and coworkers reported 3 right ventricular lacerations in 46 patients; one was fatal. [35] Wong and colleagues found 5 right ventricular punctures, 4 in patients with nonproductive pericardiocentesis, but none causing any adverse sequelae. [3] In a series of dialysis patients, 9 of 10 receiving pericardiocentesis had serious complications, including 3 deaths and 2 myocardial lacerations. [44] Duvernoy and coworkers [4] reported 23 penetrations (all right ventricular except 2 in which the right and left ventricles had been perforated), along with 4 cases of significant arterial bleeding in a series of 352 procedures.

Researchers differ in their opinions as to the adverse effects of ventricular puncture. Most ventricular punctures during the procedure occur in the lower aspect of the right ventricle. Because the pressure is lower there than in the left ventricle, [54] there should be less bleeding; however, the ventricular wall is also thinner and more vulnerable to laceration. In a series of patients whose pericardiocentesis was exclusively directed by ultrasound, ventricular puncture still occurred in 1.5% but was without consequence due to small needle size. [126] In another study, right ventricular laceration occurred in 1 patient despite the use of echocardiography, producing tamponade and necessitating emergency surgery. [100]

| | Permanyer-Miralda[100] (1985) | Wong[3] (1979) | Guberman[35] (1981) | Krikorian[27] (1978) | Kwasnik[45] (1978) |
|---|---|---|---|---|---|
| No. of cases | 80 | 52 | 56 | 123 | 34 |
| Environment | Cardiology service in Spain, all echoed | Cath lab with echo, fluoroscopy, no trauma | Cardiology service with echo, fluoroscopy, etc., no trauma | University hospital, most in cath lab, 9% trauma | All uremic patients |
| Success in obtaining fluid (%) | 88 | 69 | 87 | 86 | -- |
| Diagnosis from taps (%) | 19 | 50 | 60 (malignancy only) | 18 | -- |
| Cardiac arrest (% resuscitated) | 0 | 2 | 2 | -- | -- |
| Death (%) | 0 | 2 | 2 | 1.6 (3.2 *) | -- |
| Ventricular puncture or laceration (%) | 1.2 | 9 | 6.5 | -- | -- |

**TABLE 15-4** -- Incidence of Complications of Pericardiocentesis

| | | | | | |
|---|---|---|---|---|---|
| False-negative taps (%) | 5.3 | 7.6 | -- | -- | -- |
| Surgery needed for tamponade (%) | 1.2 | -- | 26 | 39 | -- |
| New hemopericardium (%) | 1.2 | -- | -- | 10.5 | -- |
| Major dysrhythmias (%) | 0 | -- | -- | 0.08 | -- |
| Hypotensive episode (% vasovagal) | -- | -- | -- | 2 | -- |
| Pneumothorax (%) | 0 | -- | -- | -- | 3 |
| Pneumoperitoneum (%) | 0 | -- | -- | -- | 3 |

* 1.6% indicates directly attributable deaths; 3.2% also includes contributory deaths

Of the 23 perforations in the series by Duvernoy and colleagues, [4] only 3 were considered "major" complications, with 2 of the patients requiring thoracotomy.

A small number of pneumothoraces and pneumopericardium have been reported in various series (see Table 15-4) but have been without clinical consequence. A single case of tension pneumothorax has been reported after pericardiocentesis, but the cause-and-effect relationship was unclear. [136]

**Dysrhythmias**

Serious dysrhythmias induced by pericardiocentesis are rare. Premature ventricular contractions (PVCs) occur commonly during the procedure and are benign in most cases. Wong and coworkers, [3] Guberman and colleagues, [35] Callahan and investigators, [126] and Kwasnik and coworkers [45] reported no dysrhythmias. Krikorian and Hancock reported only one episode of ventricular tachycardia and several "hypotensive vasovagal reactions," which were associated with bradycardia and responded to atropine and fluid loading. [27] Duvernoy and colleagues [4] reported 1 case of ventricular tachycardia and 1 case of atrial fibrillation among 352 procedures.

**Adverse Physiologic Consequences**

There have been a few case reports of adverse consequences even when pericardiocentesis inflicts no injury. Most of these have to do with the fact that during pericardiocentesis, the stroke volume of the previously collapsed right ventricle increases 77% with the first 200 mL of fluid removed. [57] Generally, this increase in stroke volume is greater initially than that demonstrated by the left ventricle. This can have significant consequences for both right and left ventricular function. In 3 of 6 patients in whom large effusions were removed by pericardiocentesis, there was right ventricular dilation and overload, with abnormal septal motion and either no increase in right ventricular ejection fraction or a decrease. [137] These patients returned to normal hemodynamic status slowly.

Sudden pulmonary edema also has been reported after pericardiocentesis, presumably due to a sudden increase in venous return to the left ventricle at a time when peripheral vascular resistance is still high from compensatory catecholamine secretion. [138] [139] [140] Supporting evidence for this explanation is that right ventricular stroke volume increased 76% after relief of tamponade, greater than the stroke volume increase of the left ventricle. [60] Circulatory collapse with persistently low arterial blood pressure has been reported in a patient who was drained of 700 mL of clear fluid at a rate of 100 mL/min. [141] These authors suggest that ketamine anesthesia may have played a role, but the relative ischemia created by tamponade, coupled with the sudden increase in left-sided preload, created a persisting imbalance. They recommend that pericardial drainage rate not exceed 50 mL/min. Given the rare occurrence of pulmonary edema or primary cardiac compromise, it is unclear whether this limitation is justified.

A case of brief profound bradycardia and rebound hypertension has also been reported after surgical relief of tamponade. [142] Such responses have not been noted in large series of patients receiving pericardiocentesis.

## SUMMARY

In *nontraumatic patients,* tamponade should always be considered in the differential diagnosis of shock, especially in patients who are on anticoagulants, who have had recent myocardial infarction, or who have had pericardial disease; when malignancy is present; when aortic dissection is suspected; or when a CVP catheter is in place. Tamponade should also be considered in the differential diagnosis when hypotension

persists following closed-chest CPR or attempts at cardiac pacing.

In any patient with blunt or penetrating chest or upper abdominal trauma, the possibility of *traumatic tamponade* must also be considered. If clinical deterioration occurs in the emergency department pending operative care, temporizing pericardiocentesis should be considered if other therapy fails. When such a patient arrives with no obtainable blood pressure or in profound shock and unconscious, immediate thoracotomy and pericardiotomy are indicated after intubation. [113] [143] [144] Pericardiocentesis may cause a dangerous delay in this situation and has a low success rate.

Management of tamponade requires a sound understanding of pathophysiology; an ever-vigilant evaluation; and the willingness, if necessary, to perform relatively high-risk procedures such as pericardiocentesis in critically ill or injured patients.

## Intracardiac Injection

Because intracardiac injection (ICI) is related procedurally to pericardiocentesis (i.e., both techniques require transthoracic penetration of the pericardium with a needle using similar anatomic approaches), these techniques are presented together. The major difference is that whereas the goal of pericardiocentesis is to stop needle penetration within the pericardial space, ICI requires penetration of the myocardium for delivery of drug directly into a cardiac chamber.

The technique of ICI is occasionally needed for delivery of cardiac resuscitation drugs when other vascular options are not available in a timely manner. Throughout the 1960s, ICI was recommended as the most expeditious route of drug administration during cardiac arrest. [145] [146] In the years that followed, the technique fell into disfavor as alternative (less invasive) routes for drug delivery were validated and the risks associated with ICI were enumerated.

Although ICI is no longer a preferred route for drug administration during resuscitation, it can still be useful in those cases in which prompt IV access is unattainable and when drug administration by other routes has proven ineffective. The technique of ICI is easily taught and requires little equipment. Studies suggest that when properly performed, ICI carries a low risk of clinically significant complications. [147] [148] [149]

### BACKGROUND

The technique of ICI appears to have originated in the latter part of the 19th century. Fantus attributed its first use to a German physiologist, Schiff, around 1880. [150] By 1930, more than 250 case reports, with an overall success rate of approximately

25%, had appeared in the literature. Yet use of ICIs remained controversial. The report of the Special Committee on Intracardiac Therapy of the Witkin Foundation, published in 1930, concluded that the beneficial effect of intracardiac injection was not from the medication itself, but from the irritant effect of the needle on the myocardium. [151] This concept remained popular until 17 years later, when Beecher and Linton reported a

case of intraoperative cardiac arrest that failed to respond to repeated right atrial punctures until two injections of epinephrine into the right atrium restored a normal sinus rhythm. [152] They concluded that the epinephrine, and not the puncture itself, was responsible for the successful resuscitation.

By the mid-1950s, intracardiac epinephrine had become part of the standard treatment for cardiac arrest, [146] [153] and with the advent of closed-chest cardiac massage in the 1960s, the intracardiac route of drug administration remained popular. [154] [155] [156] By the mid-1970s, the popularity of ICI had declined. Goldberg warned of the numerous potential complications and advised against the use of ICI, except when it was "absolutely necessary" or during open chest cardiac massage. [157] Schechter, in 1975, harshly condemned the ICI of epinephrine and stated that the technique has been obsolete for about 20 years." [158]

The primary reasons for the decline in the use of ICI seem to be as follows:

1. Serious complications resulting from ICI have been well documented. [154] [157] [159]
2. Safe and simple percutaneous techniques have been developed that allow entry into the central venous circulation.
3. Experimental evidence suggests that ICI offers no advantage over injection into peripheral veins. [160]
4. The endotracheal [161] [162] and intraosseous [163] [164] [165] routes of drug administration have been validated.
5. Interruption of CPR during the performance of ICI was thought to be too prolonged. [157] [166]

Although ICI use is seldom reported in the United States, published reports of its use elsewhere continue. In 1990, a Danish trial reported on 543 ICIs in 247 patients using the parasternal approach. [149] The authors concluded that "the risk of ICI with proper technique is sufficiently low to warrant its use during cardiac arrest."

## INDICATIONS AND CONTRAINDICATIONS

Evidence favoring the intracardiac route of injection is theoretical or anecdotal, with several authors suggesting that ICI of epinephrine may be more effective than IV epinephrine in treatment of asystole or electromechanical dissociation. [166] [167] [168] [169] To date, no experimental or clinical data suggest an advantage with the intracardiac drug delivery route during cardiac resuscitation. Thus, the primary indication for ICI is when other vascular access routes for drug administration are not available in a timely manner. A possible secondary indication for ICI is when other forms of drug administration, particularly endotracheal therapy, [170] have failed to resuscitate the patient. In these circumstances a trial of ICI administration would seem warranted.

In the circumstances discussed above, there are no true contraindications to this technique. Certain associated conditions, such as the presence of a preexistent pneumothorax or the hyperinflated chest of chronic lung disease, make the procedure more difficult and may alter the chosen approach. Anticoagulation has been shown to be associated with a greater incidence of hemopericardium, but this rarely results in

hemodynamic embarrassment. [156]

## EQUIPMENT AND DRUGS

Prefilled syringes intended for intracardiac injection are no longer available in the United States. As a result, an 18-ga spinal needle fitted to the end of a regular medication syringe is the best choice for ICI. Spinal needles of narrower gauge may be used, but they are more prone to bending and occlusion by tissue plugs. The primary agent used for ICI is epinephrine (1 mg for adults; 0.01 mg/kg as an initial dose for children). When children are unresponsive to the initial dose, 0.1 to 0.2 mg/kg may be administered. Generally a 1:10,000 epinephrine concentration (1 mg in 10 mL) is used. However, when the higher dose is used for children unresponsive to an initial dose, a 1:1000 concentration (1 mg in 1 mL) is preferred.

## PROCEDURE

Both the subxiphoid and parasternal approaches have been recommended as the primary approach for ICI. The parasternal approach has the advantage of a shorter, more direct route but appears to suffer from a higher rate of complications, particularly pneumothorax. This finding differs from data specific to pericardiocentesis and may be the result of differences in patient positioning during the procedure, changes in cardiac positioning with and without pericardial effusion, or the effects of cardiac arrest and ongoing CPR.

The techniques used for ICI are similar to those used for pericardiocentesis. They differ primarily in setting and the need for ECG or imaging guidance. Whereas pericardiocentesis often takes place with the patient awake and responsive, ICI always occurs with ongoing CPR and for this reason must be done as rapidly as possible to avoid prolonged cessation of CPR. Whereas the goal in pericardiocentesis is to avoid contact with the heart, ICI aims to penetrate the heart. As a result, there is no need for ECG or ultrasound guidance.

**The Subxiphoid Approach**

The technique and landmarks are the same as in pericardiocentesis. Without interrupting CPR, the left costoxiphoid area is prepared with an antiseptic solution. The syringe with the drug to be injected is freed of all air bubbles. At this point, the lungs are allowed to deflate, and CPR maneuvers are stopped. With the bevel up, the tip of the needle is inserted in the xiphocostal notch (approximately 1 cm to the left of the tip of the xiphoid process). The needle is directed cephalad toward the middle of the left clavicle at a 30° to 45° angle with the skin of the abdominal wall (Fig. 15-15) .

Some operators prefer to keep the obturator in the spinal needle until skin puncture has been completed, thus reducing the likelihood of obstructing the lumen with a skin plug. After skin puncture, the syringe is attached and the plunger gently depressed to expel any air in the needle. This technique should not be used when tissue-toxic fluids (e.g.,

**Figure 15-15** Subxiphoid approach to right ventricle for intracardiac injection. It is suggested that the needle be aimed toward the *mid* left clavicle. This figure shows the needle aimed toward the *medial* left clavicle. *A,* Frontal view. *B,* Lateral view. Note the proximity of the stomach and liver to the entrance point.

calcium chloride or sodium bicarbonate) or high-concentration epinephrine (1:1000 concentration) are used.

After the needle has penetrated into the SQ tissue, gentle constant negative pressure is applied to the syringe, and the needle is advanced rapidly. When blood spurts into the syringe, needle advancement is stopped, and the medication is injected as quickly as possible. The needle is withdrawn immediately, and CPR is resumed. *An intracardiac injection should not interrupt CPR for >5 to 10 seconds.* If, after full insertion of the needle, there is no blood return, the needle must be withdrawn immediately, and CPR must be resumed before another attempt is made. The clinician may then attempt penetration again, this time with the needle directed straight up toward the suprasternal notch. If this fails, a third attempt with the needle directed toward the mid-right clavicle is recommended. If this is also unsuccessful, the left parasternal approach should be tried.

**The Left Parasternal Approach**

This approach is completed similarly to pericardiocentesis, but both the fourth and fifth left intercostal spaces may be used. The selected space along the sternal border is prepared with antiseptic solution. After the lungs are allowed to deflate passively, the needle is inserted just over the fifth or sixth rib, immediately lateral to the sternum (see Fig. 15-11) . While gently aspirating with the syringe, the clinician rapidly advances the needle dorsally with slight medial and cranial elevation until an abrupt blood return is observed. [146] [171] At this time the medication is injected as rapidly as possible, the needle is withdrawn, and CPR is resumed immediately.

**Intracardiac Injections in Infants and Children**

The injection technique for infants and children is essentially the same as that for adults. Both the left parasternal and the subxiphoid routes may be used. The chest wall in children is thinner and more pliable than in adults, and injection with a 20- or 22-ga spinal needle has been recommended. [172] [173]

## COMPLICATIONS

As described in the introduction, a major objection to the use of ICI has been the potential for complications. Although case reports of several complications exist, clinical series have failed to document frequent problems (Table 15-5) .

**Coronary Artery Laceration**

Coronary artery laceration is a frequently mentioned potential complication of ICI. Although lacerations have been reported in animal experiments and in human cadaver studies, [13] [174] reports of laceration during resuscitation have been largely anecdotal. [175] Saphir found no coronary artery lacerations at autopsy in 62 patients who had received a total of 155 ICIs during unsuccessful resuscitation attempts. [156] Table 15-5 illustrates further series where only 1 coronary laceration was noted among a total of 842 patients receiving ICI.

**Hemopericardium**

In contrast to coronary laceration, hemopericardium appears to be relatively common following ICI and may lead to tamponade. In a series of left ventriculographies using the intercostal approach, Bjork and colleagues found four instances of hemopericardium among 27 patients who underwent surgery following cardiac puncture. [176] In the same series were six instances of cardiac tamponade among 138 patients. Lehman found three instances of tamponade in more than 300 patients receiving transthoracic ventriculography by the subxiphoid approach. [177] Saphir reported 9 cases

| TABLE 15-5 -- Complications of Intracardiac Injections | | | | | |
|---|---|---|---|---|---|
| **Study** | **Technique** | **Coronary Laceration** * ... | **Hemopericardium** | **Tamponade** | **Pneumothorax** |
| Pondsmenech, 1951 [181] | Subxiphoid | 0 (30) | 0 (30) | 0 (30) | 0 (30) |
| Lehman et al, 1957 [182] | Subxiphoid | 0 (60) | 11 (35) | 0 (60) | 0 (60) |
| McCaughan, Pate, 1957 [178] | Subxiphoid | 0 (29) | -- | 0 (29) | 2 (29) |

| | | | | | |
|---|---|---|---|---|---|
| Lehman, 1959 [177] | Subxiphoid | 0 (300) | -- | 3 (300) | -- |
| Bjork et al, 1961 [176] | Parasternal | 1 (137) | 4 (27) | 6 (137) | 8 (137) |
| Amey et al, 1978 [148] | Parasternal and subxiphoid | 0 (33) | 1 (8) | 0 (8) | 3 (33) |
| Davison et al, 1980 [147] | Subxiphoid | 0 (53) | 12 (39) | 0 (29) | 1 (29) |
| Chen, 1981 [171] | Parasternal | -- | -- | -- | 5 (16) |
| Sabin, 1983 [174] | Parasternal and subxiphoid | 0 (18) | -- | -- | 2 (18) |
| Total | | 1 (660) | 28 (139) | 9 (593) | 21 (352) |

of hemopericardium at autopsy in 62 patients following unsuccessful CPR and noted that this complication was associated with ICI. [156] Davison and coworkers found evidence of hemopericardium in 12 of 39 patients by echocardiography or postmortem examination but no clinical evidence of tamponade in 29 survivors. [147] Jespersen and colleagues [149] found only 3 cases of hemopericardium, with no tamponade attributable to ICI among 182 autopsies following unsuccessful resuscitation, but the series included 24 patients with tamponade due to cardiac or aortic rupture.

**Pneumothorax**

Intuitively, one would expect pneumothorax to be a common occurrence following ICI, particularly when using the left parasternal approach. The studies summarized in Table

15-5 bear this out, with the rate of pneumothorax among patients with parasternal ICI being 10.3% (24/233) as compared to 2.0% (3/148) among patients with subxiphoid ICI. Although pneumothorax can result from CPR alone, in the study by Amey and colleagues, 3 of 29 patients receiving ICI developed a pneumothorax, compared with only 1 of 64 who did not receive ICI during CPR. [148]

**Intramyocardial Injection**

The intramyocardial injection of contrast material has been associated with intractable ventricular fibrillation and death. [177] The combined results of 2 ventriculography series involving the subxiphoid approach showed that 4 of 434 patients underwent intramyocardial injections with subsequent ventricular fibrillation. [177] [178] This situation is probably not comparable with ICI performed during CPR, when the heart is not beating and injection must be made without the benefit of fluoroscopic guidance. It is unlikely that the true incidence of this dire complication, which may result in the same rhythm as that originally being treated, will ever be known.

**Other Potential Complications**

Laceration of other organs is a potential complication that has rarely been reported as a consequence of ICI during human resuscitation. The subxiphoid approach brings the injecting needle close to the stomach and the left lobe of the liver. Smith and colleagues reported hepatic laceration during subxiphoid injection in dog experiments. [179] Although Brown and coworkers [13] reported 14 liver punctures in cadavers following 60 subxiphoid pacemaker insertions, there have been no reports of liver or stomach laceration resulting from the clinical use of ICI. Brown and associates also found a high incidence of inadvertent right atrial puncture that complicated the subxiphoid approach. [13] Based on evidence from clinical studies, it appears that this complication is either rare or of little clinical significance during human resuscitation. [147] [148] [174]

Punctures of the pulmonary artery and aorta have been reported by Sabin and coworkers following atypical parasternal approaches utilizing the second or third intercostal space. [174] Other potential complications include infection and air embolism. Although Bodon in 1923 described a case of fulminant pericarditis following intracardiac injection, [180] no subsequent incidence of pericarditis or endocarditis has been documented. Similarly, air embolism has not been reported.

Careful attention to technique minimizes the risk of some of these complications. To further minimize potential complications following a successful resuscitation, a chest film should be examined for evidence of pneumothorax and the patient observed for signs of hemopericardium or tamponade.

The most common error in technique, and the one stated by the American Heart Association [166] as the major hazard related to ICI, is the prolonged interruption of CPR associated with the procedure. Repeated attempts at ICI after an initial failure *without the reinstitution of CPR* should be avoided.

**SUMMARY**

For more than 100 years, the intracardiac route has been used to deliver medication during cardiac arrest. Although the technique has been shown to be effective, its popularity has declined because of the availability of other effective routes for drug administration and the fear of potential

complications. A review of the literature suggests that these fears are overstated. Although intracardiac puncture is not recommended as the initial route of injection when other vascular access options exist, it should be retained as a valid technique for the administration of emergency drugs during CPR when other routes are not readily available.

# Chapter 16 - Artificial Perfusion During Cardiac Arrest

**Carol S. Federiuk**

**Arthur B. Sanders**

The modern era of cardiopulmonary resuscitation (CPR) was introduced by Kouwenhoven and colleagues [1] in 1960 in a classic paper that brought together the concepts of mouth-to-mouth ventilation, closed-chest compression, and external defibrillation. An increased understanding of the mechanism of blood flow during CPR has led to periodic revision of the recommended standards for CPR and to development of alternative methods of CPR. This chapter discusses the current understanding of the mechanism of blood flow during CPR, standard and alternative techniques of CPR, and methods for evaluating these techniques by assessing perfusion during CPR.

## BACKGROUND: MECHANISM OF BLOOD FLOW

The mechanism of blood flow during CPR has been the subject of much debate since the 1960s. Two mechanisms for blood flow have been proposed: the "cardiac pump" model and the "thoracic pump" model. The current concept of blood flow during CPR is based on these models and provides the theoretical basis for the newer CPR techniques.

Kouwenhoven and colleagues, in their 1960 paper, proposed the traditional cardiac pump mechanism of blood flow. [1] Pressure on the chest compresses the heart between the sternum and the vertebrae, forcing out blood (Fig. 16-1) (Figure Not Available) . [2] The relaxation phase of CPR allows the heart to fill. This model assumes that compression of the ventricles raises intraventricular pressure above that of the aorta and pulmonary artery, creating a pressure gradient that generates forward blood flow. Closure of the atrioventricular valves during chest compression was thought to prevent retrograde blood flow.

The cardiac pump model of blood flow has been the subject of much debate since its introduction. Weale and Rothwell-Jackson, in 1962, showed that chest compression induces almost equivalent increases in arterial and venous pressures in animals. [3] They hypothesized that closed-chest compression creates a generalized increase in intrathoracic pressure that is transmitted equally to the heart and intra- and extrathoracic vessels, because the atrioventricular valves remain open (Fig. 16-2) (Figure Not Available) . [2]

Multiple studies have been published in support of each of these models. Additional evidence for the thoracic pump model has been provided by the reported success of cough CPR. Several studies of simultaneous compression and ventilation also have suggested that increases in intrathoracic pressure produce forward flow of blood. The direct cardiac compression model has received additional support from echocardiographic studies. The data in support of each of these models have been

summarized elsewhere. [4]

Although the debate over the exact mechanism of blood flow during CPR persists, Maier and associates shed light on the subject in their study of the effect of varying the rate, force, and duration of compressions in CPR on large dogs. [5] These investigators demonstrated that the relative contribution of the thoracic pump and direct cardiac compression models to blood flow varied with the CPR technique used. Direct cardiac compression predominated when chest compressions were delivered at higher rates (high-frequency CPR), and the thoracic pump mechanism predominated in low-momentum compression techniques, such as simultaneous compression and ventilation (SCV) CPR. In addition, Babbs and coworkers noted that the optimal technique of CPR varied with the size of the experimental animal and the size of the pad performing chest compressions. [6] Large animals in arrest were more likely to benefit from SCV CPR than smaller animals. Direct cardiac compression was proposed to play a greater role in smaller animals.

In summary, the mechanism of forward blood flow during closed chest compression appears to be multifactorial, with dependence on several factors:

1. Body size of the patient
2. Chest configuration, particularly anteroposterior diameter
3. Previous thoracic surgery
4. Molding of the chest with continued CPR
5. Size of hand or paddle performing chest compressions
6. Rate and force of chest compressions

Knowledge of the mechanisms of blood flow during CPR may allow the clinician to use alternative techniques that provide better perfusion pressures during cardiac arrest. The optimal technique for CPR may not be the same in every patient. For example, obese patients with large anteroposterior diameters may benefit more from SCV; thin patients may benefit from faster compression rates. The key to clinical implementation of these changes is being able to assess perfusion during ongoing CPR. Advances in this area are discussed later in this chapter.

## STANDARD CPR TECHNIQUE

Guidelines for the performance of CPR have been recommended by the American Heart Association and are revised periodically to reflect ongoing research. [7] Current guidelines are summarized in Table 16-1 .

External chest compression is performed over the lower half of the sternum, compressing the sternum 3.8 to 5.0 cm in the normal-sized adult. The pressure is released completely after each compression, and an equivalent amount of time is allotted for relaxation as for compression. The chest compression rate is 80 to 100 per minute. Two ventilations are given after each 15 chest compressions in one-rescuer CPR, and 1.5 to 2.0 seconds are allowed for each breath in order to provide good chest expansion. In two-rescuer CPR, one ventilation is performed after every 5 compressions during a 1.5- to 2.0-second pause. Once the patient is endotracheally intubated, the

rescuer need not stop compressions for the ventilatory pause. Rather, ventilation should be performed asynchronously at a rate of 12 to 15 per minute.

In children, compressions are performed with the heel

of one hand placed 2 fingerwidths above the xiphoid process. The chest is compressed to a depth of 2.5 to 3.8 cm at a rate of 100 per minute. At the end of every fifth compression, a 1- to 1.5-second pause is allowed for ventilation in both one- and two-rescuer CPR.

Chest compressions in infants currently are performed 1 fingerwidth below the intramammary line. The chest is compressed with 2 to 3 fingers to a depth of 1.3 to 2.5 cm. The recommended compression rate in infants is at least 100 per minute. A 5:1 compression-to-ventilation ratio is maintained for both one- and two-rescuer CPR.

The preferred technique for performing chest compressions in the neonate involves encircling the chest of the neonate with the rescuer's hands and compressing the sternum just below the nipple line with the two apposed thumbs. If the rescuer's hands are too small to encircle the

| TABLE 16-1 -- Standard CPR Guidelines | | | | |
|---|---|---|---|---|
| | Adult | Child | Infant | Neonate |

| Rate of compression (per min) | 80-100 | 100 | 100 | 120 |
|---|---|---|---|---|
| Compression depth | 1.5-2 in. | 1-1.5 in. | 0.5-1 in. | 0.5-0.75 in. |
| Compression duration | 50% of cycle | 50% of cycle | 50% of cycle | 50% of cycle |
| Compression mode | Both hands | Heel of one hand | 2-3 fingers | Apposed thumbs |
| Compression-to-ventilation ratio | 15:2 (1 rescuer) | 5:1 (1 rescuer) | 5:1 (1 rescuer) | 5:1 (1 rescuer) |
| | 5:1 (2 rescuers) | 5:1 (2 rescuers) | 5:1 (2 rescuers) | 5:1 (2 rescuers) |

chest, compressions may be performed with the ring and middle fingers 1 fingerwidth below the intramammary line. The sternum of a neonate is compressed 1.3 to 2.0 cm at the rate of 120 per minute. Chest compressions are interposed with pauses for ventilation in a 3:1 ratio.

## MECHANICAL DEVICES FOR STANDARD CPR

Mechanical resuscitators have been developed that can provide standard chest compressions and ventilations during CPR. Clinical studies have demonstrated that mechanical CPR devices are comparable to standard manual CPR. [8] [9] [10] The advantages of mechanical devices include controlled, constant chest compressions; the elimination of operator fatigue; and the freeing up of personnel to perform other functions. The device currently in most common use is the Thumper Cardiopulmonary Resuscitator (Michigan Instruments, Inc., Grand Rapids, MI).

**The Thumper**

The Thumper is a gas-powered mechanical device that is in relatively broad use. This device consists of a compressed gas-powered plunger mounted on a backboard and a

time-pressure cycled ventilator (Fig. 16-3 *A* and *B*).

The Thumper delivers chest compressions at rates consistent with American Heart Association (AHA) guidelines with a compression duration that is 50% of the cycle length. Every fifth compression is followed by a ventilation at an adjustable airway pressure. The Thumper can be driven by wall oxygen at 50 psi or by standard portable oxygen tanks.

**Setup of the Thumper**

The Thumper can be positioned from either side of the patient. Care should be taken to ensure that the base plate is positioned horizontally under the patient's posterior thorax with the patient lying near the center of the base plate. After the cylindric column and piston are fitted on the base plate, the piston pad position should be adjusted so that the pad lies over the lower one third of the sternum. The compressor piston is positioned after the oxygen hose has been connected; therefore, CPR is not interrupted during assembly of the Thumper. The piston column is calibrated with rings; each ring indicates 1.25 cm of piston excursion. Before the device is started, the piston height should be adjusted so that 1 ring is just visible on the piston column. With the Thumper operational, chest compressions should be adjusted to 5 to 6.25 cm ("4 rings" of sternal displacement) or 20 to 25% of the patient's anteroposterior chest diameter, once the machine is in operation. The device should not be set to function at a predetermined chest compression force.

The ventilation hose can be connected to an endotracheal tube, an esophageal obturator, or a face mask. Airway pressure should be adjusted to 40 to 50 cm $H_2O$ during ventilation as displayed on the airway pressure gauge when the patient is intubated. Otherwise the airway pressure limit should be set at 30 cm $H_2O$ to avoid gastric distention. Airway pressure is maintained for 1.5 to 1.8 seconds.

Use of the Thumper requires operation of 3 key switches: the main on/off switch, the ventilation on/off switch, and the piston activator switch. When a pause in compressions is required for other procedures, the operator can easily stop compressions by turning off the third switch. Subclavian or jugular central lines can be placed with the Thumper in position, although it is recommended that the device be turned off during needle advancement to avoid arterial or lung injury. Defibrillation should be performed during the compression phase, when thoracic impedance is minimized.

## ALTERNATIVE CPR TECHNIQUES

Modifications of the standard CPR technique have been proposed since the 1980s. Although each technique has initially been promising when investigated in one or two laboratories, and although some have demonstrated potential advantages in small clinical trials, insufficient clinical evidence exists for widespread implementation of these techniques over standard CPR at this time.

## Simultaneous Compression

and Ventilation CPR

The SCV method of CPR was designed to take advantage of the thoracic pump mechanism of forward blood flow. It maximizes intrathoracic pressure by generating a high airway pressure during the compression phase of chest massage. During the relaxation phase, the airway pressure is released. SCV has been shown to improve arterial pressure and carotid blood flow in some animal studies. [11] Because SCV CPR has not been demonstrated to have a beneficial effect on clinical outcome, the technique should be regarded as experimental. [12]

## Interposed Abdominal Compression CPR

The technique of interposed abdominal compression (IAC) CPR interposes abdominal compressions between the chest compressions of CPR (Fig. 16-4) (Figure Not Available) . [13] [14] [15] CPR is performed using American Heart Association guidelines; however, during

**Figure 16-3** The Thumper is an oxygen-powered device which is capable of providing the chest compressions and ventilations required during CPR. *A,* The device consists of a back plate with a cylindric column. On the column fits the sliding arm, with the compressor piston to be positioned over the lower sternum. The force of chest compression is adjustable, and defibrillation can be easily performed with the device in place. *(With permission of Dixie, USA.)B,* To apply the Thumper to the cardiac arrest patient, position the base assembly under the patient's back and snap the column and arm onto the base. Attach the oxygen supply and breathing circuit to the Thumper. Provided there are adequate personnel, manual CPR can be continued from one side of the patient while the Thumper is set up on the opposite side.During a pause in manual chest compressions the massager pad of the Thumper is swung into position over the patient's sternum. For positioning the massager onto the patient's chest, numbering on the back of the column prescribes a sternal deflection of 20% of the anterior-posterior chest diameter of the patient being treated. Depth of compression is easily monitored through a clear, graduated, plastic dome which houses the compression piston. Ventilations are delivered via a time-cycled, pressure-regulated ventilator which is synchronized to deliver one breath following each fifth chest compression. Maximum ventilation pressure is adjustable from 0 to 60 cm $H_2O$. The breathing circuit on the unit can be attached to a mask, endotracheal tube, or other airway management devices. Compressions and/or ventilations can be easily paused and restarted.The Thumper is factory set to deliver 80 compressions per minute with a 1:1 compression-to-relaxation ratio. The ventilator is set to deliver a 1.6-second inspiratory time and deliver a breath with each fifth chest compression. The compression rate and ventilation time can be adjusted should standards or local directives change.The Thumper is easily operated using the 5 controls on the unit, which are clearly marked with numbers 1 through 5. The five controls have the following functions.

> #1 opens the master valve and begins pneumatic cycling of the unit. A clicking sound is heard at the pre-set chest compression rate.

> #2 allows gas flow to the dome and compression piston.

> #3 is used to adjust the depth of compression.

the relaxation phase, the abdomen is compressed. Some investigators recommend using a partially inflated, folded blood pressure cuff connected to a transducer to monitor abdominally applied pressure (goal, 120 to 150 mm Hg). Babbs notes that the method of abdominal compression may impact the effectiveness of this technique; he recommends administering abdominal compressions from the victim's left side with the heel of the rescuer's right hand placed just above the umbilicus and centered 3 cm to the left of the abdominal midline. [16]

Several animal studies have demonstrated that IAC CPR increases arterial systolic and diastolic pressures, cardiac output, and the arteriovenous diastolic pressure difference (i.e., coronary perfusion pressure [CPP]) when compared with standard CPR. [13] [14] [15] Despite these results, IAC CPR has not been shown to improve *survival* in animal studies. [17] In humans, IAC CPR inconsistently improves the diastolic CPP. [18] One early study of IAC CPR vs standard CPR on patients treated out of hospital by paramedics following endotracheal intubation found no difference in resuscitation rates. [19]

More recent clinical studies have demonstrated promise for the use of IAC CPR for patients in cardiac arrest. Ward and colleagues showed significantly higher end-tidal $CO_2$ levels (an indirect measure of cardiac output during CPR)

**Figure 16-4** (Figure Not Available) An artist's conception of basic rescuers performing interposed abdominal compression CPR. For clarity, both rescuers are shown on the same side of the victim. With two rescuers, the first compresses the chest and performs ventilation while the second compresses the abdomen. With three rescuers, ventilation, chest compression, and abdominal compression are performed by separate individuals. Ideally, the rescuer performing chest compressions is on the victim's right side and the rescuer performing abdominal compressions is on the victim's left side. *(From Voorhees WD, Niebauer MJ, Babbs CF: Improved oxygen delivery during cardiopulmonary resuscitation with interposed abdominal compressions. Ann Emerg Med 12:128, 1983. Reproduced by permission.)*

when IAC CPR was compared to standard CPR. [20] Sack and coworkers evaluated IAC CPR in a randomized controlled trial of in-hospital cardiac arrest; patients who received IAC CPR had a significantly greater rate of return of spontaneous circulation (51% vs 27%) and hospital discharge (25% vs 7%) compared to patients receiving standard CPR. [21] In another 143 in-hospital patients with asystole and electromechanical dissociation, those patients receiving IAC CPR had significantly greater rates of return of spontaneous circulation (49% vs 28%) and 24-hour survival (33% vs 13%). [22]

**High-Frequency (Rapid Manual) CPR**

Rapid manual CPR uses standard CPR techniques, but chest compressions are performed at a rate of 120 per minute. Maier and colleagues demonstrated that increasing the compression rate to 120 compressions per minute in an animal model

resulted in increased cardiac output, aortic pressure, and coronary blood flow. [5]

Canine studies by Feneley and associates demonstrated that a compression rate of 120 per minute resulted in improved resuscitation and higher 24-hour survival rate compared with a compression rate of 60 per minute. [23] Swart and coworkers demonstrated that at a compression rate of 100 per minute, shorter compression duration (i.e., duty cycles less than 50% or "high-impulse" CPR) also improved resuscitation hemodynamics. [24] The relative merits of high-frequency versus high-impulse techniques remain to be determined, although considered intuitively, a shorter compression duration (high-impulse CPR) would permit greater cardiac filling during high-frequency CPR.

Kern and colleagues studied rapid manual CPR in 23 patients in cardiac arrest; they found that patients who had CPR with chest compression rates of 120 per minute had slightly higher levels of end-tidal $CO_2$ excretion (15 ± 2 vs 13 ± 2 mm Hg) compared to patients with chest compression rates of 80 per minute. [25]

**Vest CPR**

Mechanical devices have been developed to simulate "cough CPR" in the animal model. The vest or binder device produces pulsatile simultaneous increases in intrathoracic, intra-abdominal, and airway pressure through simultaneous inflation of a pneumatic vest, abdominal binder, and endotracheal tube. Niemann and coworkers demonstrated improved hemodynamics and 24-hour survival for those animals receiving vest/binder CPR compared with standard CPR. [26] In contrast, Kern and colleagues could find no improvement in resuscitation or 24-hour survival when vest CPR was compared with standard mechanical CPR. [27] Similarly, lack of benefit was demonstrated in one out-of-hospital cardiac arrest study. [28]

Recently a new mechanical device (vest CPR) has been developed to provide intermittent circumferential compression of the chest through a pneumatic system (Fig. 16-5) . [29] In arrest patients, vest CPR delivered with this device significantly increased peak aortic pressure and CPP when compared with standard CPR. After prolonged unsuccessful standard CPR, despite improved return of spontaneous circulation with the vest, there were no long-term survivors. [29]

**Active Compression-Decompression**

The concept of active compression-decompression (ACD) CPR was based on a case report in which a patient was resuscitated from cardiac arrest with the aid of a plumber's helper. [30] The application of a suction device to the chest wall of a patient in cardiac arrest converts the relaxation phase of standard CPR to one of active decompression. A device based on the principle of active decompression following compression has been developed (Fig. 16-6) and has shown improved hemodynamics in experimental models. [31] The application of this device to humans in cardiac arrest has shown promise in some settings but no improvement in other settings. [32] [33] [34] [34A]

**Chest Compression-Only CPR**

The role of ventilation in bystander CPR is being reassessed. Many people, including health care professionals, have concerns about performing mouth-to-mouth ventilation. [35] [36] [37] While no authority advocates that health care professionals not perform ventilation with CPR, some believe that more laypersons would learn and perform CPR if it involved only chest compressions.

In an animal model of cardiac arrest, Berg and colleagues found no difference in resuscitation, 24-hour survival, or neurologic outcome when comparing 12 minutes of chest compressions alone vs treatment with chest compressions and ventilations. [38] Studies on chest compression-only CPR in humans are limited.

Bossaert and colleagues found that when good-quality chest compression-only CPR was performed on out-of-hospital cardiac arrest patients, there was no difference in long-term survival when compared to chest compression and ventilation CPR (15% vs 12%). [39] Similarly, Van Hoeyweghen and coworkers retrospectively found that only 10% of out-of-hospital arrest patients who received chest compression-only CPR survived long term. This compares with a 16% survival rate for patients receiving good-quality chest compressions

**Figure 16-5** A comparison of vest CPR and standard manual CPR. With vest CPR, a pneumatic system inflates and deflates a bladder surrounding the chest. The compression phase results in circumferential compression as opposed to point compression during manual CPR. The vest CPR system is equipped with defibrillator electrodes that monitor the patient's electrocardiogram as well as allow defibrillation without removal of the vest.

and ventilations, and a 7% survival rate when no bystander CPR was performed.

## ASSESSMENT OF ONGOING CPR

**Quality of CPR**

The assessment of ongoing CPR efforts is crucial in efforts to resuscitate patients in cardiac arrest. Studies have called into question the quality of CPR as currently performed. Kern and colleagues noted in a study comparing two compression rates that the most improvement in end-tidal $CO_2$ excretion occurred before and after audiotones were used to guide compressions. Mean end-tidal $CO_2$ level significantly increased after audiotone guidance was begun (9 ± 1 vs 14 ± 1 mm Hg). [25] Milander and coworkers found that few in-hospital cardiac arrest patients had chest compression performed at rates within AHA guidelines. [41] In a laboratory study assessing compliance with AHA guidelines, CPR-certified health care professionals showed dramatic improvement in adherence to CPR guidelines and in subject end-tidal $CO_2$ levels with use of audiotones. [41] These studies imply that CPR is often not performed according to AHA

guidelines.

The importance of the quality of CPR efforts was emphasized in 2 European studies and 1 U.S. study. Bossaert and colleagues demonstrated that bystander CPR with correct technique and good effect was more effective in producing long-term survival than CPR with bad technique or weak effect. [39] Van Hoeyweghen and coworkers also demonstrated that patients who received correct CPR had significant improvement in long-term survival (16% vs 4%) compared to patients with incorrect CPR and patients with no bystander CPR (16% vs 7%). [40] Gallagher and colleagues found an odds ratio of about 4:1 in favor of hospital survival when out-of-hospital CPR by bystanders was judged as having both effective compressions and ventilations (independent of initial rhythm and intervals from resuscitation). [42]

## Assessment of Ongoing CPR

The prognosis for resuscitation of patients in cardiac arrest depends on the no-flow interval before CPR is initiated, the time interval from collapse to defibrillation (if the rhythm is ventricular fibrillation), and the initial cardiac rhythm. Once the resuscitation efforts are initiated, there is no ideal criterion to judge the efficacy of CPR. In most animal and clinical studies, outcome (i.e., resuscitation or death) is the only

**Figure 16-6** The active compression-decompression (ACD) device uses a suction cup positioned at mid-chest at the level of the nipples. The device is pushed downward into the chest during the compression phase of CPR. The force of compression can be approximated by the gauge within the handle. During the decompression phase, the handle is actively pushed up and away from the chest while the suction keeps the device attached. This active withdrawal characterizes the decompression phase of CPR. The value of this adjunct is uncertain.

criterion used. Recent studies in the literature have focused attention on the problem of assessing effectiveness of ongoing resuscitation efforts.

### Hemodynamics

Studies in experimental arrest models have demonstrated the importance of the aortic diastolic pressure and CPP for successful resuscitation. [43] Redding, in a series of classic experiments, showed that the aortic diastolic pressure must be raised >40 mm Hg to successfully resuscitate animals in cardiac arrest. [44] He suggested that alpha-catecholamine agents were crucial for resuscitation, because they help raise the aortic diastolic pressure. [45] Animals without vascular disease can be resuscitated even after 30 minutes of ventricular fibrillation, providing the aortic diastolic pressure and CPP are maintained at adequate levels. [46] Coronary blood flow has been shown to correlate well with the diastolic CPP. [47]

Although data confirming these observations are limited for humans undergoing CPR, one can extrapolate that CPR will be most effective when it provides adequate myocardial perfusion. In the setting of cardiac arrest, coronary blood flow and myocardial perfusion are directly related to the CPP. Paradis and colleagues measured

the CPP in 100 patients with cardiac arrest during CPR. Patients with return of spontaneous circulation had significantly higher maximal CPP (25.6 ± 7.7 vs 8.4 ± 10.0 mm Hg) compared to those who died. No patient with a maximal CPP <15 mm Hg had return of spontaneous circulation. [48] However, in patients with severe coronary artery obstruction, it may be impossible to generate adequate blood flow to some areas of the myocardium with CPR.

In summary, the aortic diastolic pressure and CPP appear to be the best available criteria for assessing perfusion during CPR and correlate with survival in animal models. Thus, the clinician should attempt to optimize these hemodynamic parameters during CPR. However, in most practice settings, the clinician will not have the opportunity to directly measure aortic and right atrial pressures during CPR. Therefore, simpler, less invasive parameters are needed to guide resuscitation efforts in most patients suffering cardiac arrest.

**Arterial Blood Gases**

Clinicians often use arterial blood gas values to confirm the adequacy of their CPR efforts. Studies in experimental models have not shown a correlation between arterial blood gas values and successful resuscitation. [49] Physiologically, one would not expect the arterial pH, $P_{O_2}$, and $P_{CO_2}$ to reflect tissue perfusion pressures. In fact, patients who suffer sudden ventricular fibrillation with no systemic perfusion will have normal aortic pH, $P_{O_2}$, and $P_{CO_2}$ for several minutes following the arrest.

More recently, attention in the literature has focused on mixed venous gases. In the setting of cardiac arrest, venous gases will frequently show severe hypercarbia and acidosis, while arterial blood gas values are relatively normal. [50] The extent of this venous-arterial $P_{CO_2}$ gap may be reflective of poor perfusion pressures. Evaluation of the venous-arterial $P_{CO_2}$ gap, however, must be considered an experimental technique.

**Pulses**

Another commonly used technique for assessing the adequacy of CPR efforts is the presence or absence of femoral or carotid pulses. There are no studies indicating the clinical utility of pulses during CPR. The pulse represents a pulse pressure or the systolic-to-diastolic pressure gradient and does not reflect the CPP. In addition, reliance on femoral pulses can be misleading. Since there are no valves in the inferior vena cava, blood frequently flows in a retrograde fashion to the lower half of the body during chest compression. Thus, a *palpable femoral pulse may represent venous rather than arterial blood flow*. The presence of carotid pulses may indicate some forward blood flow with chest compression. However, the extent of blood flow and, more important, tissue perfusion cannot be gauged by the presence or absence of a pulse.

**Capnometry**

Capnometry has been evaluated for monitoring ongoing CPR efforts. Capnometers use infrared spectroscopy to measure $CO_2$ in exhaled air. The sensor or sampling port is

attached to the endotracheal tube. Carbon dioxide concentration in exhaled air is dependent on (1) its production by systemic metabolism; (2) its circulation, specifically pulmonary blood flow or cardiac output; and (3) the ventilatory system for excretion. In the setting of cardiac arrest and CPR, in which systemic metabolism and ventilation are held relatively constant, exhaled $CO_2$ reflects primarily lung perfusion and cardiac output.

In the experimental model, end-tidal $CO_2$ concentration during CPR correlates well with cardiac output and CPP. [51] [52] Studies in animal models have shown that $CO_2$ monitoring during CPR can distinguish those animals who will be successfully resuscitated from those that die. [53] [54] There have been few studies evaluating the clinical use of capnometry

for monitoring patients in cardiac arrest. Kalenda reported three patients who suffered cardiac arrest while being monitored by capnometry. [55] He found that when a rescuer fatigued while performing CPR, $CO_2$ concentration fell; however, $CO_2$ concentration promptly increased again when a fresh rescuer took over. These observations suggest using capnometry as a prognostic guide to ongoing resuscitation.

**Indications.**

Although capnometry remains an experimental technique, it shows promise as a means of noninvasively monitoring perfusion during CPR. Animal [54] and clinical studies [56] [57] suggest that increased $CO_2$ concentration may be the first indicator of restoration of spontaneous circulation. Capnometry also may prove helpful for quantifying the probability of developing a perfusing rhythm as a result of resuscitative efforts. The technique is relatively contraindicated only when pharmacologic therapy may affect proper interpretation of the resuscitative efforts.

**Equipment and procedure.**

A variety of manufacturers supply capnometry equipment. Most capnometers use infrared absorption spectroscopy to measure $CO_2$ . Operation of each monitor and recorder should follow its manufacturer's instructions. Calibration of the monitor should be performed periodically with a known $CO_2$ source.

A sampling port slips between the endotracheal tube and a resuscitation bag. Some devices provide in-line continuous $CO_2$ readings, whereas others suction off samples of air from the endotracheal tube to a sensor in the body of the machine. Some capnometers are portable and battery-operated so that they may easily be used by prehospital care providers. [58] The clinician is interested in the end-tidal $CO_2$ (or peak) level, since this most closely approximates the alveolar $CO_2$ concentration.

Many manufacturers produce a multitude of capnometers for clinical use; many of these products are compared elsewhere. [59] Some capnometers are primarily designed for operating room or critical care unit use. Some systems incorporate 2 or 3 measurements, such as pulse oximetry and respiratory rate, along with capnometry.

However, pulse oximetry is not useful for patients in full cardiac arrest.

Two devices that seem appropriate for emergency department use are the Novametrix 1265 Capnogard and the Nellcor N-60 Stat Cap $CO_2$ Detector. The Novametrix 1265 Capnogard (Novametrix Medical Systems, Inc., Wallingford, Conn) weighs only 3.6 kg and is portable, with a 2-hour battery life (Fig. 16-7) (Figure Not Available) . It has a solid-state mainstream sensor and automatically adjusts to atmospheric pressure. The Nellcor N-60 Stat Cap $CO_2$ Detector (Nellcor, Inc., Hayward, Calif) weighs <1 kg and has a 16-hour battery life. It uses a mainstream sensor and requires no user calibration. Its portability makes it particularly useful for out-of-hospital cardiac arrest. Note that qualitative $CO_2$ detector devices are not recommended for assessing perfusion during CPR.

### Interpretation.

End-tidal $CO_2$ measurements are given either as a concentration in percent $CO_2$ (%$ET_{CO2}$ ) or as the partial pressure of $CO_2$ in mm Hg ($Pet_{CO2}$ ). Measurement of the $Pet_{CO2}$ varies slightly with the altitude at which the reading is taken. Animal studies have indicated that sequential end-tidal $CO_2$ measurements correlate with cardiac output, [57] myocardial perfusion pressure, [56] [60] and resuscitation from cardiac arrest. [60] [61]

Clinical studies assessing end-tidal $CO_2$ during CPR have been limited. In one series of 18 adults in late cardiac arrest, a %$ET_{CO2}$ of 1.7 to 1.8% was associated with a mean arterial

**Figure 16-7** (Figure Not Available) One commercial combination capnometer and pulse oximeter (Novametrix 1265 Capnogard). The mainstream sensor fits between the endotracheal tube and ventilation bag. Readouts of continuous $Pet_{CO2}$ readings permit the clinician to follow trends in $Pet_{CO2}$ levels and waveform analysis over time. *(Courtesy of Novametrix Medical Systems, Inc, Wallingford, Conn.)*

diastolic pressure of 22 to 25 mm Hg, and no patient was resuscitated. [60] Another study of 23 cardiac arrest victims, including 10 patients who attained spontaneous circulation, did not find a difference in %$ET_{CO2}$ between those resuscitated and those without return of spontaneous circulation. [56] Falk and associates also found no significant differences in the %$ET_{CO2}$ for resuscitated and nonresuscitated patients during CPR, although sequential changes in %$ET_{CO2}$ were seen with the onset of CPR and with return of spontaneous circulation. [57] However, Asplin and White found 1- and 2-minute $Pet_{CO2}$ levels monitored during out-of-hospital CPR to be higher in patients who had a return of spontaneous circulation. [58]

Several clinical studies have addressed differences in end-tidal $CO_2$ during ongoing CPR as a predictor of effectiveness of resuscitation. Callaham and Barton correlated initial end-tidal $CO_2$ with the return of a palpable pulse in 55 emergency department patients. The 14 patients who had a return of palpable pulse had a mean $Pet_{CO2}$ of 19 mm Hg, compared with 5 mm Hg for the 41 patients who did not develop pulses. [62] Another clinical study of 35 adult cardiac arrests found that the 9 patients who were successfully resuscitated also had a higher average $Pet_{CO2}$ during CPR than those who

could not be resuscitated (15 ± 4 vs 7 ± 5 mm Hg). [63] All 9 resuscitated patients had an average $Pet_{CO2}$ of 10 mm Hg or greater, and the 3 who survived to hospital discharge had an average $Pet_{CO2}$ of 17 mm Hg. One out-of-hospital cardiac arrest study found no survivors with a $Pet_{CO2}$ of 10 mm Hg after 20 minutes of arrest. [64]

Caution must be used in interpreting end-tidal $CO_2$ values. Experimentally, administration of sodium bicarbonate has been shown to increase excretion of $CO_2$ and hence adversely affect the correlation of end-tidal $CO_2$ with perfusion during CPR. Therefore, end-tidal $CO_2$ readings are not reflective of perfusion pressures during CPR for 5 minutes following the administration of bicarbonate. In addition, alterations in ventilations during CPR may affect end-tidal $CO_2$ readings. The effect of pressor agents during ongoing

CPR on end-tidal $CO_2$ is variable. [65] Experimentally, high doses of epinephrine lower end-tidal $CO_2$ and cardiac output during cardiac arrest while increasing myocardial perfusion pressures and coronary blood flow. [66] [67]

While explicit recommendations for use of capnometry to monitor patients during cardiac arrest are not warranted by the data available, the published data can be extrapolated to generate qualitative guidelines. If the $Pet_{CO2}$ value is 0, the ventilation system must be immediately reassessed. The endotracheal tube may have slipped into the esophagus, with resultant loss of ventilation. If tube placement cannot be ensured, the patient should be immediately reintubated. In a few cases, $Pet_{CO2}$ values of 0 may be due to cardiac arrest caused by a large pulmonary embolus obstructing pulmonary blood flow, with a resultant loss of $CO_2$ excretion. Finally, one must reevaluate the capnometer and ensure that it is working appropriately. The windows of the sensor cell may become clogged with mucus or blood, or the calibration may be inappropriate.

If the $Pet_{CO2}$ values are <10 mm Hg but not 0, one must reevaluate the artificial perfusion technique used during CPR. Readings below 10 mm Hg are associated with poor cardiac output during CPR and a poor prognosis for resuscitation. The force applied, the site of chest compression, and the rate of compressions should be assessed. A second rescuer or mechanical compressor may be needed.

If the $Pet_{CO2}$ values are >10 mm Hg, the resuscitators should try to optimize the $Pet_{CO2}$ values. Often the person doing chest compressions can directly observe the $Pet_{CO2}$ values produced. Subtle modifications in CPR technique using this feedback may then be used to improve the $Pet_{CO2}$ values.

The trend in $Pet_{CO2}$ values is important when monitoring patients in cardiac arrest. Decreasing values may mean that the resuscitator is tiring and a new person should do chest compressions. A consistent downward trend in $Pet_{CO2}$ values is a poor prognostic sign. On the other hand, a sudden upward trend in end-tidal $CO_2$ values may indicate a return of spontaneous circulation even before a pulse is felt. Such patients should be carefully assessed for spontaneous pulses, especially when the cardiac arrest is due to "pulseless electrical activity."

**Conclusions.**

Capnometry has potential as a noninvasive barometer of artificial perfusion during cardiac arrest. In general, optimal end-tidal $CO_2$ values are associated with optimal pulmonary perfusion. Hence, optimizing end-tidal $CO_2$ may improve the chance for resuscitation from cardiac arrest.

## SUMMARY

We have presented overviews of a number of proposed modifications of standard CPR. Limited evidence supports further evaluation of these techniques. Regardless of the closed-chest CPR technique used, assessment of ongoing CPR efforts is desirable for rational management of patients in cardiac arrest. Hemodynamic monitoring, especially of the aortic diastolic pressure and aorta-to-right atrium perfusion gradient (CPP), currently represents the best indicator of adequate cardiac perfusion during ongoing CPR. However, in most clinical cardiac arrest settings, it is impractical to measure these hemodynamic parameters. Therefore, indirect indices of perfusion such as capnometry may be useful in the overall evaluation of patients in cardiac arrest.

# Chapter 17 - Resuscitative Thoracotomy

**Robert L. Bartlett**

"If the etiology of the patient's deterioration is hemorrhage, surgery should not await resuscitation but is an integral part of resuscitation, as only when bleeding is controlled can the circulating blood volume be restored adequately." [1] As a multidisciplinary specialist, the emergency physician at times finds it necessary to use procedures previously considered to be the province of the surgical specialties. The emergency thoracotomy is the most invasive and controversial of these procedures. "The days when an individual specialty could restrict the use of a technical skill are long gone. Pertinent to technical skills are appropriate training, skills maintenance, and judgment as to when to apply them or when to withhold them." [2] The reality of emergency care is that few of the 6900 registered hospitals in the United States are classified as level I trauma centers with in-house surgical coverage. But almost all hospitals provide 24-hour emergency physician coverage. When death is imminent, the most qualified physician available should perform the indicated procedures without hesitation.

Since the early 1970s, the development of sophisticated emergency medical systems using well-trained paramedics, advanced life support, and rapid transport have increased the number of patients arriving at the emergency department (ED) in various stages of shock. With increasing frequency, emergency physicians are being given the opportunity to resuscitate patients who previously would have died at the scene. For some, survival is possible if an aggressive approach using emergency thoracotomy is taken. Therefore, knowing who may respond to thoracotomy becomes an important issue.

This chapter discusses the factors that influence the outcome of an emergency thoracotomy and the pathophysiology, diagnosis, and treatment of the injuries that require such an invasive procedure. The heart, lungs, and great vessels are the three vital structures in which injuries may require the use of a resuscitative thoracotomy. The mechanism of injury, prehospital vital signs, and systolic blood pressure following thoracotomy form the basic structure of a resuscitative algorithm.

Although this chapter provides considerable detail of the rationale for and the technique of resuscitative thoracotomy, the emergency physician should be aware of the *specific goals to be attained after opening the chest.* In the trauma patient receiving a resuscitative thoracotomy, the physician seeks to relieve any cardiac tamponade; to support cardiac function (with direct cardiac compression, cross-clamping of the aorta to improve coronary perfusion, and internal defibrillation when indicated); and to control hemorrhage from the heart, pulmonary vessels, thoracic wall, and great vessels. Although certain circumstances may require this sequence of resuscitation to be altered, the emergency physician should be cognizant of these goals on opening the chest.

Additionally, each institution should establish guidelines for the initiation of resuscitative thoracotomy and subsequent patient care in the ED. Ideally a preestablished plan of chest wound management and post-thoracotomy care should be established with the

service that will provide the emergency physician's surgical backup for those times when members of the surgical team cannot be on site at the time of the resuscitation. With such a plan, a team approach to resuscitation and optimal patient care is possible.

# INDICATIONS AND CONTRAINDICATIONS

## Penetrating Chest Injuries (General)

With penetrating thoracic trauma, the frequency of organ injury corresponds to the relative exposure of each organ. The lung is the most commonly injured organ, followed by the heart, the great vessels, the tracheobronchial tree, and the esophagus. Approximately 80% of penetrating chest injuries can be managed conservatively with tube thoracostomy if the only significant injury is a pneumothorax (see Chapter 9) .

The out-of-hospital mortality for penetrating chest wounds depends on the structures involved. For heart wounds in particular, the prehospital mortality is approximately 80%. Nonetheless, if any signs of life are present during the prehospital phase, these patients are *potentially* salvageable. The vital importance of speed and the use of the ED as the site for thoracotomy has been emphasized. [3] MacDonald and McDowell, commenting on their series of 28 emergency thoracotomies in a community hospital, observed that "resuscitation can be hampered by significant delays in assembling the necessary operating room staff. These delays sometimes mean that a trauma patient will be detained for a significant amount of time in the ED before a definitive procedure can be performed in the operating room." [4] "Delay and observation in the shocked, bleeding, or tamponading injury can only lead to prolongation of hypotension, acidosis, excessive requirement of blood and crystalloids, and on occasion sudden ventricular fibrillation or standstill." [5]

When the patient's vital signs suggest that cardiac arrest is imminent despite airway control and the initiation of volume replacement, a thoracotomy should be performed immediately. Death may result if the physician allows a cardiac arrest to occur while waiting for a possible response to ancillary therapy or while transporting the patient to the operating table. Beall and coworkers compared the mortality figures for emergency thoracotomies performed for penetrating cardiac injuries in patients with in-hospital cardiac arrest. [6] For the group receiving thoracotomies before cardiopulmonary arrest, the mortality was 15%. If the thoracotomy was performed after cardiopulmonary arrest, the mortality increased to 60%. The hardest part of any action is the decision to commence. It should be clear that every effort, including thoracotomy, should be made to avert, rather than treat, a cardiac arrest.

For patients in cardiac arrest, tracheal intubation and duration of out-of-hospital cardiopulmonary resuscitation (CPR) are prognostic factors that govern who will benefit from thoracotomy. Recent observations suggest that a time of 5 minutes of out-of-hospital CPR approaches the limits of viability of *nonintubated* trauma patients. The value of field

intubation is dramatic; the average time of CPR tolerated by intubated survivors is

double that of nonintubated survivors (9.4 vs 4.2 minutes). [7]

The type of electrical activity associated with the arrest forms a second tier of criteria. Although there are anecdotal responses to asystolic arrest with brief periods of resuscitation, these are extremely rare, and the presence of asystole should be considered a relative contraindication from a cost-benefit perspective. The presence of an agonal wide-complex idioventricular rhythm less than 40 beats/min and unresponsive to drug therapy also carries a grave prognosis. Ventricular fibrillation does not always indicate end-stage myocardial death and may be associated with a more favorable outcome to thoracotomy. [8] Finally, any patient with an organized rhythm faster than 40 beats/min is an *excellent* candidate for thoracotomy.

When the neurologic outcome of resuscitative thoracotomy is considered, a third decision factor is the presence of some form of neurologic activity, such as movement to painful stimuli or reactive pupils, during either the out-of-hospital phase or on arrival to the hospital. Patients with such recent neurologic activity are significantly more likely to survive without neurologic impairment. Although the absence of neurologic activity during the out-of-hospital phase may be associated with survival, in at least 1 series, all patients who left the hospital neurologically intact had some spontaneous movement present at some time in the field or ED. Of the 4 patients who survived in a persistent vegetative state, 3 had no evidence of movement (cerebral function) during their course in the field and in the ED. [9]

**Cardiac Injuries**

Eighty to 90% of stab wounds to the heart result in tamponade. The development of tamponade may be temporizing during the out-of-hospital phase. If tamponade does not occur, most patients with myocardial stab wounds will exsanguinate. [10] In contrast, when gunshot wounds involve the heart, the associated blast effect usually results in exsanguination rather than tamponade. [11]

Two additional factors that influence the development of tamponade are wound size and chamber involvement. Wounds of the myocardium less than 1 cm may spontaneously seal, depending on the location. Wounds larger than 1 cm usually continue to bleed regardless of the chamber involved. The low-pressure atrial wound usually forms a thrombus before tamponade develops. However, a wound of the higher-pressure right ventricle is often associated with tamponade. The thicker-walled left ventricle may spontaneously seal stab wounds up to 1 cm in length.

Cardiac tamponade is the *decompensated* phase of cardiac function resulting from increased intrapericardial pressure (see also Chapter 15) . Although one may suspect tamponade based on well-described signs, the clinical diagnosis of pericardial tamponade in the unstable trauma patient is notoriously difficult because of the combined effect of hemorrhagic and cardiogenic shock. The classic signs of Beck's triad (distended neck veins, hypotension, and decreased heart sounds) have *limited diagnostic value* for acute penetrating cardiac trauma. [12] Additional signs of tamponade include tachycardia; pulsus paradoxus; elevated central venous pressure; agitation and

confusion (reflecting decreased cerebral perfusion); air hunger; and cold, clammy skin.

Three mechanisms may partially compensate for the cardiac tamponade state. The first is reactive tachycardia. Reactive tachycardia during the hypotensive phase of tamponade is the usual finding, but for some patients, relative or absolute bradycardia may be present. [13] A second compensatory mechanism is the elevation of central venous pressure (CVP). However, many affected patients have marked volume depletion, and the CVP will not be elevated despite advanced tamponade. If hypotension persists and the CVP rises, the patient should be presumed to have cardiac tamponade; however, there may be a poor correlation between CVP and the extent of tamponade, even when the blood volume has been corrected. [14]

The third compensatory mechanism is increased peripheral vascular resistance that preserves arterial pressure in the presence of falling cardiac output. When hypotension does appear, it is an ominous sign, and the emergency physician must respond quickly.

**Pulmonary Injuries**

Pulmonary injuries can be divided into three types: parenchymal, tracheobronchial, and large-vessel. Parenchymal and tracheobronchial injuries rarely create a situation requiring thoracotomy in the ED, and most of these injuries are either rapidly fatal or can be adequately treated initially by tube thoracostomy.

Tracheobronchial injuries may be incurred from penetrating trauma. Although these injuries are more common with blunt trauma, most patients with this injury die at the scene. [15] With this injury, the airway is usually maintained, even in the presence of a complete transection. The stiff tracheobronchial cartilage tends to hold the lumen open while the peritracheal and peribronchial fasciae preserve the relationship of proximal to distal bronchi. Ninety percent of tracheobronchial tears occur within 3 cm of the carina. [16]

These tears most commonly involve the main stem bronchi. Less frequently, vertical tears may occur along the membranous cartilage line of the trachea. Complete division of the trachea is extremely rare. Depending on the size and location of the injury, patients may present with one or more of the following: massive hemoptysis when bronchial vessels are involved, airway obstruction, and pneumomediastinum or pneumothorax, with or without tension. If hemorrhaging is profuse or if the site of injury can be determined, the use of a bifid endotracheal tube or the unilateral intubation of a main stem bronchus will secure the airway.

Lacerations of the parenchyma unaccompanied by major vessel injuries generally respond to a tube thoracostomy. Although the associated hemothorax may be significant, reexpansion of the lung often halts or reduces bleeding. Reduction of parenchymal bleeding by negative pressure coaptation of the pleural surfaces is successful in 72 to 98% of cases. [5] If the initial chest tube drainage is more than 800 mL, with continued drainage at a rate of 50 mL every 10 minutes, or if there is persistent hypotension, immediate thoracotomy should be considered. Such patients rarely have simple parenchymal injuries; major vascular structures are usually involved. A

complication of parenchymal injuries that requires immediate thoracotomy is the development of air embolism.

## Air Embolism

### Presentation

Suspicion of air embolism following trauma is considered to be an indication for emergency thoracotomy. Until recently, the occurrence of air embolism following penetrating injuries of the lung had not been widely recognized, although it may be a significant cause of morbidity and mortality. [17]

The preoperative and postmortem diagnosis of air embolism is difficult. Air embolism is confirmed at thoracotomy by needle aspiration of a foamy air-blood admixture from the left or right ventricle or by visualization of air within the coronary arteries. Preoperative demonstration of air by aspiration from a central venous catheter or the femoral artery is rare, but it has been reported. [18]

Air embolism may appear in either the right or the left side of the circulatory system. Involvement of the right side of the circulation is referred to as *venous* or *pulmonary* air embolism. Generally, venous air is well tolerated, but death may occur when the volume of air reaches 5 to 8 mL/kg. The rate at which air moves into the circulation and the body's position are important determinants of the volume that can be tolerated. Death usually results from obstruction of the right ventricle or the pulmonary circulation. If the mean pulmonary arterial pressure exceeds 22 mm Hg, air may pass into the systemic circulation. Paradoxical air embolism may also occur in the 15 to 25% of patients who have a potentially patent foramen ovale.

The most common cause of venous air embolism is management error with IV therapy. Air embolism fatalities have been reported with subclavian venipuncture. [19] A pressure difference of 5 cm $H_2O$ across a 14-ga needle will allow the introduction of 100 mL of air per second. Injuries of the vena cava or the right ventricle also create obvious portals of entry into the right circulatory system.

Air embolism involving the left side of the circulatory system is referred to as *arterial* or *systemic* air embolism. The lethal volume depends on the organs to which it is distributed. As little as 0.5 mL of air in the left anterior descending coronary artery has led to ventricular fibrillation. Arterial air traverses systemic capillaries more readily than those in the pulmonary system. [20] Clinical manifestations of arterial air embolism are related to the involvement of the coronary or cerebral circulation. The distribution of arterial air is partly a function of body position.

Systemic air embolism following injury of the lung has only recently been described. The formation of traumatic bronchovenous fistulas creates potential entry points for air to move into the left side of the circulatory system. The only requirement is the formation of an air-blood gradient conducive to the inward movement of air. Although a lowered intravascular pressure from hemorrhage is a risk factor, the most important element in

all reports of air embolism has been the use of positive-pressure ventilation. [21]

In a review of 447 cases of major thoracic trauma, Yee and colleagues found adequate chart data to diagnose air embolism in 61 patients. [17] This incidence of 14% is remarkable in light of the small number of reported cases before 1973. [20] A mechanism of blunt injury should not preclude a consideration of this diagnosis, because 25% of patients with air embolism reported by Yee and coworkers had blunt trauma with associated lung injury secondary to multiple rib fractures or hilar disruption. The overall mortality was 56% (34 of 61 patients). Refractory cardiac arrest accounted for 63% of the operative deaths, with exsanguination or severe brain trauma as the cause in the remaining 37%.

The diagnosis of air embolism is easily overlooked because of the similarity of the signs and symptoms to those of hypovolemic shock. Two valuable signs that were present in 36% of patients were hemoptysis and the occurrence of cardiac arrest *after* intubation and ventilation. The diagnosis of air embolism also should be considered when unconsciousness develops suddenly and is followed by seizures in a patient with lung injury on positive-pressure ventilation. [22]

**Management**

A high index of suspicion with rapid control of the source of air embolism is vital. The patient should immediately be placed in the Trendelenburg (head-down) position to minimize cerebral involvement by directing the air emboli to less critical organs. This step should be followed by a left anterolateral thoracotomy. The exposed thorax should be flooded with sterile saline. Peripheral bronchovenous fistulas can be identified by the bloody froth created during positive-pressure ventilation. A quick search for hilar injuries should be carried out in the patient with blunt trauma. If the source of air embolism is not readily apparent, a contralateral thoracotomy should be performed. Once the bronchovenous communication is controlled, needle aspiration of the residual air that commonly remains in the left ventricle and the aorta should be performed. If the patient is hypotensive, the aorta may now be cross-clamped or compressed with the hand. "Reflex" cross-clamping of the aorta before control of bronchovenous fistulas and removal of residual air will result in further dissemination of air to the heart and the brain.

**Adjunctive Therapy**

As mentioned earlier, air emboli traverse capillary beds if the blood pressure is high enough. After control of the bronchovenous fistula, a brief period of proximal aortic hypertension can be produced by cross-clamping of the descending aorta. Systemic arterial pressure should be maintained with adequate fluid resuscitation. If vasopressors are required, metaraminol (Aramine) appears to be the drug of choice. Other pressor agents such as dopamine, epinephrine, or norepinephrine increase systemic pressure but do not necessarily facilitate the left-to-right passage of bubbles as well as metaraminol does. [23]

Left atrial pressure should be maintained at a high level. The ventilator inspiratory

pressures should be kept as low as possible, and 100% oxygen should be used to facilitate diffusion of nitrogen from emboli. Pharmacotherapy may include steroids, mannitol, aspirin, and barbiturates in conjunction with hypothermia. The most important adjunctive therapy is the use of a hyperbaric chamber.

Hyperbaric oxygen therapy is beneficial because it (1) compresses air bubbles; (2) establishes a high diffusion gradient, which greatly speeds the dissolution of the bubbles; (3) improves the oxygenation of ischemic tissues and lowers intracranial pressure; and (4) reduces the reperfusion injury that invariably follows the passage of bubbles. When it is available and logistically practical, hyperbaric oxygen therapy should be considered, *even though it may be many hours before it can be initiated*. The effectiveness of hyperbaric oxygen therapy is illustrated by cases of success and improvement

even when as many as 36 hours elapsed before pressurization. [24]

## Major Vascular Injuries

Major vascular injury resulting in rapid deterioration following blunt or penetrating trauma requires the use of an emergency thoracotomy for diagnosis, resuscitation, and control of hemorrhage. Mavroudis and others reviewed 76 patients with thoracic vascular injury from mixed trauma who received an ED thoracotomy because they were moribund or an immediate thoracotomy in the operating suite for hemodynamic instability. The three most common sites of vascular injury are the pulmonary artery (28% of cases), intercostal artery (23% of cases), and pulmonary vein (20% of cases). Aortic injuries account for only 12% of the injuries requiring immediate surgical intervention. However, even with immediate intervention, survival rates are low (14 to 29%). Air embolism was the cause of death in 18% of cases. [25]

The clinical approach to patients with suspected major vascular injury depends on hemodynamic status, the mechanism of injury, and the presence of associated injuries. If the patient's condition is sufficiently stable, angiography is a valuable diagnostic measure, although the risk of sudden deterioration necessitates constant monitoring. If the patient's condition is deteriorating rapidly and vascular injury is suspected, emergency thoracotomy plays a dual role as a diagnostic and a resuscitative procedure. It must be emphasized that patients with seemingly trivial penetrating wounds may appear stable and yet may precipitously exsanguinate and suffer an unexpected arrest.

## Blunt Trauma and Penetrating Abdominal Injury

Emergency resuscitative thoracotomy for *blunt* trauma has limited ability to resuscitate patients who arrive with agonal signs. [26] *Therefore, this procedure is relatively contraindicated for the management of blunt trauma in both adult and pediatric populations*. [27] The exception to this rule is the blunt trauma patient who arrives in the ED with a measurable blood pressure, but whose condition deteriorates precipitously, leading to suspicion of an intrathoracic injury. [28]

In the setting of penetrating abdominal injury, thoracotomy with cross-clamping of the thoracic aorta to control hemorrhage from the injury has been advocated, but survival rates have been poor for those undergoing this procedure, and its routine use is not supported. The collective survival rate for 194 cases described in the literature is only 5%. [29] Three factors contribute to this low figure. First, aortic occlusion does not substantially affect the rate and volume of bleeding from major venous injuries. Second, all vital signs had been lost in most patients by the time of thoracotomy. Third, multiple collateral pathways around the cross-clamped aorta diminish the effectiveness of this procedure. [30]

Aortic cross-clamping for massive hemoperitoneum was originally conceived as a preoperative "prophylactic" procedure to prevent sudden hypotension following abdominal decompression. Applied in the operating room in this role, it is of clear benefit when systolic pressure cannot be raised above 80 mm Hg prior to laparotomy. [31]

## Open-Chest Resuscitation for Nontraumatic Arrest

Failure to resuscitate patients from cardiac arrest is a result of (1) a delay in the onset of CPR, (2) the use of less than optimal resuscitative techniques, or (3) the intractability of the underlying disease process. The development of closed-chest resuscitation, which is quickly and easily applied, coupled with the development of more advanced out-of-hospital care has dramatically reduced the number of failures caused by a delay in the onset of CPR. Techniques to improve closed-chest resuscitation continue to be sought (see Chapter 16 , because after the first 5 minutes of CPR in adults, closed-chest compression may provide inadequate cerebral blood flow and little myocardial perfusion. [32]

The mean coronary perfusion pressure with conventional CPR is approximately 7 mm Hg, whereas open-chest cardiac massage produces a coronary perfusion pressure of 32 mm Hg. [33] The cardiac index with conventional CPR is 0.6 L/min/m [2] , and with direct cardiac massage the index is doubled. [34] Similarly, cortical blood flow cannot be maintained at more than 10% of normal during conventional CPR. [35] [36]

At present, the precise role of open-chest resuscitation for nontraumatic arrests is poorly defined, and the procedure is not considered a standard of care. Although several indications have been suggested, only 2 theoretically can be readily accepted. The first indication is for resuscitation of patients with hypothermic arrest (see Chapter 71) . Certainly, in this setting, cardiopulmonary bypass is the most rapid method of core rewarming, but it is rarely available on an immediate basis, or bypass is relatively contraindicated because anticoagulation would be used in the setting of known or suspected trauma. Open thoracotomy with mediastinal irrigation has been used successfully for severe hypothermia with cardiac arrest. With severe hypothermia, ventricular fibrillation is resistant to chemical or electrical conversion, and prolonged periods of closed-chest compression must be provided during the long process of rewarming.

Patients surviving neurologically intact in previously published case reports of

open-chest resuscitation with direct cardiac rewarming had 30 to 180 minutes of internal massage. [37] Core rewarming with this technique can be as fast as 8 °C/hour, and it preferentially rewarms the heart and lungs first. Such a rewarming sequence prevents "rewarming shock," which may occur if the extremities or abdomen rewarm first, producing metabolic and circulatory demands that exceed the capacity of the heart. Tap water at a temperature of 40 °C can be poured slowly over the heart and into the thorax. Sterile saline heated in a microwave oven to the same temperature is equally effective and somewhat more hygienic. Performing a thoracotomy for hypothermic arrest does not preclude the subsequent use of cardiac bypass. If there are no major contraindications to bypass, such as head trauma, open-chest resuscitation with mediastinal rewarming can be performed while arrangements for cardiac bypass are made.

The second more provocative theoretical indication is for use in normothermic arrests. Long-term survival is rare when the duration of closed-chest resuscitation is *continuous* for more than 30 minutes without cardiac response. The probability of long-term survival for witnessed medical arrests is approximately 86% during the first 10 minutes of

resuscitation. [38] This is reduced to 30% by 16 minutes and becomes less than 1% after 30 minutes. [39] [40] This temporal survival profile is predicated on basic life support beginning within 4 minutes and advanced life support being initiated within 8 minutes of cardiac arrest.

Investigation by Kern attempted to address the optimum timing of open-chest cardiac massage following closed-chest compression using a canine model. [41] It appears that if initiation of open-chest cardiac massage is delayed for more than 20 minutes from the onset of cardiac arrest, few or no successful outcomes can be expected despite the prompt prior institution of closed-chest compression. However, beginning direct cardiac massage after 15 minutes of closed-chest massage could still produce a 75% success rate vs a 12% success rate in the control group in which 15 minutes of closed-chest compression was not followed by direct cardiac massage.

In a comparative study of closed- and open-chest resuscitation using a canine model, Bartlett and coworkers demonstrated improved survival and neurologic outcome with the open-chest cardiac compression method. Only 10% of the control animals could be resuscitated after 50 minutes of closed-chest resuscitation, and all had fixed and dilated pupils. An experimental group received 10 minutes of closed-chest resuscitation followed by 40 minutes of open-chest resuscitation for a total arrest time of 50 minutes. *All* of these dogs were resuscitated; equally important was the preservation of the pupillary light reflex in 90% of these animals. [42] These animal studies should be considered "best case" scenarios in that the animals did not have underlying cardiac or neurologic disease, and closed-chest CPR was promptly instituted.

Based on these studies, the "best case" temporal window of efficacy for open-chest resuscitation is within 10 and 15 minutes of arrest and assumes performance of prior closed-chest CPR. For the vast majority of sudden death victims, it is unlikely that they will be at a facility with open thoracotomy capability within that time frame. Clinical studies comparing open- and closed-chest resuscitation suggest that the clinical utility of

open-chest resuscitation for out-of-hospital arrests may be extremely limited despite the impressive cardiac outputs that can be generated.

## EQUIPMENT

The physician must carefully consider the instruments to be included in a resuscitation thoracotomy tray. The inclusion of too many instruments makes the tray cumbersome and delays the procedure. Nonessential instruments are best kept available in the resuscitation room in case they are needed for specific repair (e.g., Foley catheter for stellate wound tamponade).

The following items are essential for a thoracotomy tray:

> Scalpel with attached No. 20 blade
>
> Mayo scissors (or long Metzenbaum scissors)
>
> Rib spreaders
>
> Liebsche knife (or sternal osteotome with hammer)
>
> 2 tissue forceps (10 in.)
>
> 2 Satinsky vascular clamps
>
> 3 Hegar needle holders (10 in.)
>
> 2-0 or larger silk sutures on large-curve needle
>
> Teflon patches
>
> Suture scissors
>
> Aortic tamponade instrument
>
> Skin stapler (6 mm staples)

The following items are optional for the tray and can be supplied as needed by an assistant:

> 6 towel clips
>
> 4 to 6 hemostats (curved and straight)

Metzenbaum scissors

Right-angled clamp

Foley catheter (20 Fr, 30-mL balloon)--sterile saline/ syringe

Chest tube (No. 30, Argyle)

12 lap sponges or gauze pads

6 towels

In addition, functioning suction and sterile suction tips, antiseptic solution, sterile gloves, a defibrillator with internal paddles, and overhead surgical lights are needed in the resuscitation room. In the unlikely event that the patient awakens during the procedure, sedative and analgesic agents should be available for sedation, amnesia, and pain control.

## PROCEDURE

### Preliminary Considerations

In the urban out-of-hospital setting in which a trauma center is less than 15 minutes away, patients with penetrating thoracic injuries should receive immediate transportation with the least possible field time. Two studies suggest that this "scoop-and-run" approach provides a better survival rate. [45] [46]

For all trauma victims presenting to the ED with hypotension, the initial working diagnosis must be one of volume depletion. Other possibilities should be rapidly excluded, such as tension pneumothorax, cardiac tamponade, air embolism, and neurogenic or cardiogenic shock. A useful algorithmic overview of the approach to chest trauma is shown in Figure 17-1 .

Because a large amount of blood may be lost into the chest, an autotransfusion system should be available (see Chapter 28) . The use of autotransfusion has several benefits. [47] The most important advantages are (1) immediate availability of compatible, warm blood; (2) significantly higher levels of 2,3-diphosphoglycerate (DPG) than in stored blood; (3) elimination of transfusion diseases; and (4) less risk of exhausting the banked supply of the patient's blood type. The fourth point can be crucial when there are blood bank shortages or crossmatch problems.

**Airway Control During Thoracotomy**

Patients undergoing resuscitative thoracotomy in the ED obviously require assisted ventilation. Airway control is best obtained with standard orotracheal intubation, but exposure of the thoracic organs and surgical repairs or procedures may be hampered by frequent inflations of the left lung. Selective one-lung ventilation using a specialized double-lumen endotracheal tube is an established technique in thoracic surgery, but the availability of and experience with these devices is limited. The right lung often can be selectively intubated by blindly advancing a standard single-lumen endotracheal tube to a depth of 30 cm (measured at the corner of the mouth) in adult patients. [48] Although the left lung and the right upper lobe are not ventilated with the

**Figure 17-1** An algorithmic approach to chest trauma. QRS, organized electrical activity; VFib, ventricular fibrillation; (+)TAP, pericardial tap yielding blood.

tracheal tube in this position, animal studies and preliminary data from humans suggest that selective right lung ventilation provides adequate oxygenation and ventilation for at least 60 minutes, thereby expediting the thoracotomy by minimizing the technical problems encountered by continual left lung inflation. [48]

**Anesthesia and Amnesia**

Comatose patients undergoing resuscitation may regain consciousness during a successful emergency thoracotomy, but the use of paralyzing agents may mask the return of awareness. The physician must be cognizant of this phenomenon and administer adequate analgesia and amnestic agents to the paralyzed and ventilated patient. Due to the profound shock that accompanies this procedure, ketamine (2 mg/kg IV) and midazolam (0.10 to 0.2 mg/kg IV) have been recommended. [49] It would be prudent to administer these agents routinely if the paralyzed patient demonstrates viable perfusion during resuscitation.

**Subxiphoid Pericardial Window**

Most patients who demonstrate viable perfusion with a suspected cardiac injury are candidates for a subxiphoid pericardial window for diagnosis and decompression of a suspected evolving pericardial fluid collection (see also Chapter 15) . This procedure is best performed in the operating suite.

**Anterolateral Thoracotomy Incision**

The patient should be intubated. A nasogastric tube can be passed to help differentiate the esophagus from the aorta (Fig. 17-2 *A*). When the site of injury is unknown and the patient's status requires immediate intervention for possible intrathoracic injuries, a left anterolateral incision over the fifth rib with dissection into the fourth intercostal space provides the best access to the heart and the great vessels. In the setting of cardiac arrest, time should not be taken to count the rib spaces. An incision just beneath the nipple in the male or along the inframammary fold in the female will approximate the

fourth intercostal space (Fig. 17-2 *B* and *C*). Closed-chest compressions are continued during the initial incision. The first sweep of the scalpel (No. 20 blade) should separate skin, subcutaneous fat, and the superficial portions of the pectoralis and serratus muscles. It is important to establish wide exposure from the outset by extending the skin incision past the posterior axillary line. To facilitate this

**Figure 17-2** Left anterolateral thoracotomy. *A,* Several towels or sandbags are placed under the left scapula and the arm is raised above the head. The patient should be intubated. A nasogastric tube can be inserted to facilitate differentiation of the esophagus from the aorta. *B,* The left anterolateral submammary incision is the suggested initial approach. Ideally the incision is made between the fourth and fifth ribs. Generally the incision is just inferior to the nipple (male) or along the inframammary fold (female). The incision begins on the sternum and extends to the posterior axillary line, where it should be deep enough to partially transect the latissimus dorsi muscle. *C, Dashes* indicate the incision site of the inframammary fold in women.

action, quickly wedge towels or sheets under the left posterior chest and place the patient's left arm above the head (see Fig. 17-2 *A*). Inadequate exposure, rib fractures, and additional delays occur when the skin incision is too limited.

To enter the pleural space, a small incision is made in the intercostal muscles and one blade of the scissors is inserted. The intercostal muscles are then cut with the scissors to expose the thoracic cavity (Fig. 17-3 *A*). When dividing the intercostal muscles with a scalpel or Mayo or Metzenbaum scissors, be careful not to lacerate the lung. With the first opening of the pleura, ventilations should be stopped momentarily. This will allow the lung to collapse away from the chest wall. The intercostal incision is placed just over the top of the fifth rib so as to avoid the intercostal artery. Some surgeons prefer to begin the thoracotomy incision over the sternum, whereas others begin 2 cm lateral to the edge of the sternum, hoping to avoid the internal mammary artery. Should the internal mammary artery be transected during the procedure, hemorrhage is generally minimal until after perfusion is reestablished. At that time, the patient can hemorrhage actively from a lacerated internal mammary artery. Therefore, all internal mammary artery lacerations should be ligated once perfusion is established.

The intercostal space is spread and a chest wall retractor (rib spreader) is placed with the handle and ratchet bar down (Fig. 17-3 *B*). If the retractor is placed with the handle up, the ratchet bar will prevent extension of the incision into the right chest. When the site of injury is to the right of the heart and cannot be reached, a transsternal extension into the right chest is performed with a Liebsche knife or a sternal osteotome. In patients with suspected left subclavian vessel injuries or aortic arch injuries, better exposure and control are obtained when the third intercostal space is used. If access is still difficult, the superior ribs may be separated at the costochondral junction. Ribs may be broken during spreading, so care must be taken to avoid being cut on the sharp bone edges. If a massive hemothorax is encountered, clots should be removed manually and towels used to soak up blood.

## Pericardiotomy

If cardiac arrest has occurred, the question of whether to open the pericardial sac arises. If the myocardium cannot be visualized, the pericardium should be opened. However, in some cases, the myocardium can be evaluated through the intact pericardium. Nonetheless, if there is no other obvious injury in the chest and a cardiac injury is possible, the pericardium should be opened routinely, because *it may be difficult to definitively rule out pericardial tamponade by visual inspection alone*. The author has had experience with two cases in which a substantial amount of clotted blood was hidden in the posterior, dependent aspect of the pericardium. The hemopericardium and associated injury were discovered during a continued search for possible injuries.

If the physician is confident that tamponade is not present, it is usually best to leave the pericardial sac closed while other life-threatening injuries are addressed. Opening the pericardium increases the risk of complications. For example, any delay in beginning cardiac compressions will add to the risk of cerebral damage. The myocardium or a coronary vessel may be injured. The left phrenic nerve may

**Figure 17-3** A, When entering the pleural cavity, it is important to make the incision on top of the rib to avoid the intercostal vessels. Once a hole has been made into the pleural space, the incision is widened with blunt scissors by cutting the intercostal muscles. The 4th and 5th fingers of the operator's free hand are inserted into the pleural space to fend off the lung as the scissors divide the intercostal muscle. Momentary cessation of ventilation will collapse the lung. Alternatively, the right mainstem bronchus can be intubated, which permits continuous ventilation and oxygenation without inflating the left lung into the operating field. *B,* The incision must always be carried to the posterior axillary line to maximize exposure. The rib spreader should be placed with the handle laterally. Because it can be difficult to determine if tamponade has occurred using visual inspection alone, the pericardium must be opened to definitively determine if tamponade is present. Using tissue pick ups with teeth, the operator must press hard against the pericardium to engage it within the tissue pick ups. The incision is started near the diaphragm and anterior to the phrenic nerve, which is easily identified as a thick tendon-like structure. Using blunt scissors, the incision is carried to the root of the aorta.

be cut by mistake, and if there has been previous pericardial disease, adhesions may be present. If attempts are made to separate these adhesions rapidly, tears of the atrial or right ventricular wall can occur. The incidence of traumatic rupture of the atria or the right ventricle during massage is greater when the pericardium is open. With an intact pericardium, pressure is distributed over a larger area and the pericardial fluid seldom allows the compressing fingers to remain in one spot for a prolonged period.

Patients with tamponade require pericardiotomy. This is performed in a location anterior and parallel to the left phrenic nerve. The incision should start near the diaphragm to avoid possible injury of the coronary arteries. The pericardial sac is lifted with forceps, and scissors are used to make a small hole in the sac; the scissors are then used to further open the anterior pericardium from the apex of the heart to the root of the aorta. When the pericardium is under tension, it may be difficult to grasp the pericardium with forceps. In that case, sharp, straight Mayo scissors are used to divide the pericardium

by layers. If the heart is in arrest, speed is important, and sharp scissors should be used to "catch" the pericardium and to start the pericardiotomy. To do this, the point of the scissors is held almost parallel to the surface of the heart with enough pressure to create a wrinkle in the pericardium that can be punctured as the scissors are moved forward. Moderate pressure must be used to puncture the fibrous pericardium. The sudden "give" that occurs when the pericardium opens may result in a laceration of the myocardium if the point of the scissors is unnecessarily angled toward the heart. Clots of blood are removed from the pericardial sac by the sweeping motion of a gloved hand or with sterile lap sponges or gauze pads.

## Direct Cardiac Compressions

Three techniques for cardiac compression have been advocated: one-handed compression, one-handed with sternal compression, and two-handed (bimanual) compression (Fig. 17-4) . The *one-handed compression* method is performed with the thumb placed over the left ventricle, the opposing fingers over the right ventricle, and the apex of the heart resting in the palm of the hand. The *one-handed with sternal compression* method also is performed with the fingers flat. The fingers of the hand are held tightly together to form a flat surface over the left ventricle while compressing the heart up against the sternum. To perform the *two-handed compression* method, the left hand is cupped and placed over the right ventricle. The fingers of the right hand are held tightly together to form a flat surface supporting the left ventricle. This flat surface compresses the heart against the cupped surface of

**Figure 17-4** Two-handed method of cardiac massage. The ventricles are compressed toward the interventricular septum. Note how the hands flank the left anterior descending artery, which overlies the septum. Avoid using excessive finger *tip* pressure or lifting the heart, which slows ventricular filling by distorting the soft atrial caval junction.

the left hand. Of these three, the bimanual technique is consistently superior, and whenever possible, it is the preferred method. [50]

A difference of opinion exists regarding the optimal rate at which the heart should be compressed. Most of the literature has recommended a rate of 50 to 60 compressions per minute; however, no physiologic data support such a recommendation. Johnson and Kirby studied the relationship of compression rate to cardiac output and blood pressure and found these parameters to be directly related. [51]

It is important to remember the following points while performing cardiac compression:

1. Finger *tip* pressure should be avoided at all times. Compression is performed using the entire palmar surface of the fingers.
2. Whichever technique is used, the force of compression should be perpendicular to the plane of the septum. The anterior descending coronary artery is located over the interventricular septum and is a helpful landmark to orient proper hand placement. It is clearly seen, with or without the pericardium open.
3. The fingers should be positioned so that the coronary arteries will not be occluded.

4. Venous filling of the heart is especially sensitive to changes in position. It is important to maintain a relatively normal anatomic position of the heart to *prevent* kinking of the vena cava and pulmonary veins. The heart should not be angled more than 30 degrees into the left chest.
5. It is also essential to completely relax the heart between compressions. If present, intra-arterial pressure monitoring is of tremendous value for assessing the consistency and effectiveness of compressions.

**Control of Hemorrhagic Cardiac Wounds**

One may partially control active bleeding from ventricular wounds by placing the finger of one hand over the wound while using the other hand to stabilize the beating heart. This maneuver buys time while the physician prepares to repair the injury. The use of surgical staples for ventricular wound closure is an extremely rapid method for controlling hemorrhage (Fig. 17-5) . [52] This technique may be particularly useful with large or multiple lacerations. Another advantage is that stapling does not expose the operator to the risk of a needle stick. Macho and coworkers reported a 93% success rate in temporarily controlling hemorrhage in 28 patients (33 lacerations) with penetrating injuries to both atria and ventricles using a standard skin stapler with wide (6 mm) staples placed at 5-mm intervals (Auto-Suture 35W, U.S. Surgical Corp, Norwalk, Conn). [53] The rotating long neck of the Ethicon Proximate Quantum Skin Stapler (Model PQW-35, Ethicon, Inc, Somerville, NJ) also is advantageous for obtaining proper orientation of the staples during placement. The staples may be left in place and reinforced or replaced on further wound exploration in the operating room.

Alternatively, the wound can be repaired by placement of several horizontal mattress sutures under the tamponading finger (Fig. 17-6) . Nonabsorbable 2-0 silk sutures are customarily used. Smaller sutures should *not* be used, and nylon sutures should be avoided. Some physicians prefer to use even larger silk sutures, such as No. 1 or 2 (note that this is *not the same as* 0 [1-0] or 0-0 [2-0] sutures). When multiple sutures are needed, they should all be in place before they are tied. This allows for a rapid and equal distribution of wound tension, which prevents tearing of the myocardium. [54] Passing the suture through Teflon pledgets also prevents the suture from cutting through the myocardium. It is especially important to use Teflon pledgets for reinforcement when the myocardium has been weakened by the blast effect of a bullet. [55]

With large wounds that cannot be palpably controlled, an incomplete horizontal mattress suture should be placed on either side of the wound (Fig. 17-7) . The free ends are then crossed to stop the bleeding. The actual reparative sutures can then be accurately placed. It must be stressed that suturing the myocardium requires good technique. Excessive tension may tear the myocardium and aggravate the situation. Keys to success include the use of an appropriate-sized suture, a generous "bite" with the needle, and the application of only enough tension necessary to control bleeding.

If exsanguinating hemorrhage is not controlled by the aforementioned methods, temporary inflow occlusion can be used. Inflow occlusion may be applied intermittently for 60 to 90 seconds. During occlusion the heart shrinks, hemorrhage is controlled, and

sutures can be placed in a decompressed injury. Two techniques that are useful are vascular clamping of the superior and inferior vena cava for partial inflow occlusion 56 and the Sauerbruch grip (Fig. 17-8) for occlusion of the vena cava between the ring and the middle finger of the left hand for partial inflow occlusion. [57] Use of the Sauerbruch grip has the utility of speed and the added advantage of cradling and stabilizing the heart during repair

**Figure 17-5** Technique of cardiac stapling to temporarily control hemorrhage. An assistant can approximate tissues with fingertip pressure or, as illustrated, 2 half-horizontal sutures can be used to approximate the wound edges and reduce bleeding. A skin stapler with wide (6 mm) staples is used to place staples 5 mm apart. This technique may be used for atrial and ventricular lacerations. Following stabilization of the patient's condition, the wound is revised in the operating room.

of wounds over either ventricle or the left atrium. The Sauerbruch grip will only interfere with the repair of wounds involving the right atrium.

Insertion of a Foley catheter (20 Fr with a 30-mL balloon) through a wound is another technique for temporarily controlling hemorrhage. [58] Following insertion of the catheter, the balloon is inflated, the catheter is clamped to prevent air embolism, and gentle traction is applied (Fig. 17-9) . Enough traction is applied to slow the bleeding to an acceptable level for visualization and repair. Attempts to achieve complete hemostasis with excessive traction may pull the catheter out and potentially enlarge the wound. The balloon will effectively occlude the wound internally. When repairing the wound, the operator must be careful with the suture needle to prevent rupturing the balloon. A pursestring suture is

**Figure 17-6** A, Technique of repair. Multiple horizontal mattress sutures are placed 6 mm from the wound edge before tying. The wound is closed just enough to stop the bleeding. Teflon pledgets are used for reinforcement. Closure without pledgets incurs the risk of sutures ripping through the contracting myocardium. Similarly, the use of simple vertical sutures should be discouraged because of the risk of suture dissection through the myocardium. *B,* For repairs near a coronary artery, care is taken to pass the suture under the artery.

commonly used. Temporarily pushing the balloon into the ventricular lumen during needle passage is important to prevent rupture. Alternatively, the balloon can be temporarily deflated during suture placement. It is important to use normal saline when inflating the balloon. Use of air will result in air embolism if the balloon is ruptured by the suture needle.

**Figure 17-7** Hemorrhage control using 2 widely placed incomplete mattress sutures. An assistant then crosses the two "half-horizontal" sutures to bring the wound edges into apposition. By controlling the hemorrhage in this manner, the assistant's hands are outside of the operative field, fully exposing the wound edges. This facilitates a more orderly closure of the wound. Following repair of the wound, the sutures may either be removed or tied to each other.

**Figure 17-8** Sauerbruch maneuver: the *method of choice* for reducing heavy bleeding from cardiac wounds. Venous inflow occlusion is achieved by using the first and second or second and third fingers as

a clamp.

Foley catheters have several advantages over other methods of controlling cardiac wounds. With the digital method, the fingertip will often slip if there is a strong heartbeat, the wound cannot be visualized during repair, and digital pressure significantly interferes with cardiac massage. Intermittent total venous inflow occlusion is an effective method of controlling bleeding and decompressing the heart, but such control will be at the expense of a poor cardiac output. Comparatively, the Foley catheter causes less cardiovascular interference, although inflation near the base of the ventricle may obstruct blood flow. Attempts to elevate the heart for control and repair of posterior cardiac wounds will often result in cardiac arrest by reduction of both venous and arterial flow. With posterior injuries, one cannot continuously view the wound for digital control of bleeding. Use of a catheter does not require continued viewing after initial placement. If bleeding can be controlled, repairs in this location should await full volume expansion or cardiopulmonary

**Figure 17-9** Serial illustration. Gentle traction on an inflated Foley catheter will control hemorrhage and allow easy repair. The balloon is inflated with saline, and care is taken to avoid rupturing the balloon with the suture needle. This technique is particularly useful with injuries of the inferior cavoatrial junction, with posterior wounds, and during cardiac massage. Volume loading can be obtained by infusion of blood or crystalloid solutions through the lumen of the catheter. Care should be taken to avoid an air embolus through the lumen of the catheter during placement.

**Figure 17-10** Technique for elective fibrillation: 20 J (watt-sec) is delivered through internal defibrillating paddles placed perpendicular to the epicardium. Coronary vessels should be avoided during paddle placement.

bypass. [59] Regardless of location, the most valuable feature of Foley catheter use is the ability to control hemorrhage without interfering with cardiac compression.

Deliberate *fibrillation* (to halt myocardial contractions) should be considered as a last resort for repair of difficult wounds of the ventricle or the proximal aorta. Elective cardiac arrest is best tolerated if there is adequate blood volume and oxygenation before fibrillation. To induce fibrillation, the internal cardiac paddles are placed perpendicular ("on-edge") to the surface of the heart and are discharged at 20 J (Fig. 17-10) . [56] This produces a local area of depolarization. The resulting disparity in relative refractory periods sets up a circus movement, which produces ventricular fibrillation. The heart should be massaged intermittently during repair, and the duration of fibrillation should not exceed 3 to 4 minutes.

*Defibrillation* is accomplished while the internal paddles are firmly pressed *tangentially* over the right and left ventricles. Following repair, the epicardium is often dry and should be moistened with saline to improve electrical conduction. An energy level of 20 J is used. If the initial attempt is unsuccessful, repeated shocks at the same setting should be used. Higher energy levels can cause myocardial necrosis. [60]

Defibrillation through an intact pericardium also should begin with 20 J. If unsuccessful,

the shock should be repeated once and then increased to between 40 to 60 J.

Management of the wounded heart that has spontaneously arrested is controversial. Some physicians have recommended a rapid repair of ventricular wounds while the heart is arrested. Others consider immediate cardiac massage and reversal of cardiac arrest to be more important. Immediate cardiac massage to maintain blood flow is probably the best approach. When cardiac arrest occurs, physiologic reserves have been depleted, and a delay for repair during arrest would only diminish the chance of a successful resuscitation.

Wounds of the atria are initially managed with partial occlusion clamps (Fig. 17-11) . Because of the thin structure and instability of the atrial wall, digital pressure will not effectively stop bleeding. Injuries near the caval-atrial junction are not amenable to clamping. In this location a Foley catheter should be used to tamponade the wound (Fig. 17-12) . [61] Care must be exercised to avoid obstruction of atrial filling with the inflated balloon. During wound closure, the catheter should be pushed away from the ventricular wall or the balloon temporarily deflated to avoid rupture of the balloon. Skin staples also have been used for closure of atrial wounds. [53]

Wounds of the septa, valves, and coronary arteries require definitive repair in the operating suite. Hemorrhage from a coronary artery can generally be controlled with digital pressure. Ligation of a coronary artery should be avoided when possible.

**Control of Hemorrhagic Great Vessel Wounds**

Wounds of the great vessels can be controlled with digital pressure or partial occlusion clamps. Exsanguinating hemorrhage

**Figure 17-11** Use of a partial occluding clamp in different locations for control of bleeding and subsequent repair.

**Figure 17-12** Wounds of the inferior cavoatrial junction are difficult to manage with simple vascular clamping. *A-D,* Use of a Foley catheter provides satisfactory control.

from the left subclavian artery can be prevented by cross-clamping of the intrathoracic portion of the artery. Cross-clamping of the right subclavian artery is very difficult. For injuries of this vessel, compression with laparotomy pads in the apex of the pleura from below, and the supraclavicular fossa from above (Fig. 17-13) , will prevent further bleeding as the patient is stabilized and moved to the operating suite. [52]

Large or difficult vena caval injuries may be controlled with a temporary intravascular shunt to maintain venous return while providing vascular isolation of the injured segment. [62] This is a difficult and time-consuming procedure that is best done in the

operating room, if the patient survives long enough.

Occasionally, fluid resuscitation can be accomplished by infusing fluid directly into the right atrium. The usual technique for placing atrial catheters using a pursestring suture in the right atrium has several disadvantages. First, it is relatively time-consuming for a patient in cardiac arrest, and cardiac massage is often interrupted to allow suture placement. In addition, the suture may tear the atrial appendage, the suture holes are frequently associated with fluid leaks through the thin atrial wall, and the catheter can slide out of place. Samuelson described an innovative technique utilizing an umbilical cord clamp with a center hole (Fig. 17-14 A). A standard umbilical cord clamp (Hollister, Inc., Libertyville, Ill) is modified by drilling a hole through the center to match the size of standard IV tubing or whatever catheter is to be used. [63] The diameter of the hole should hold the catheter firmly enough to prevent slippage. These clamps, by necessity, must be prepared in advance and gas sterilized.

To place a catheter using this technique, the right atrial

Figure 17-13 Cross-clamping for control of subclavian bleeding is difficult and time consuming. Compression with laparotomy pads in the apical pleura from below and the supraclavicular fossa from above will control hemorrhage while the patient's condition is stabilized and the patient is transported to the operating room.

Figure 17-14 A, Modified umbilical cord clamp. B, Modified umbilical cord clamp securing a catheter in the right atrial appendage.

Figure 17-15 Manual cardiac massage and cross-clamping of the aorta to increase coronary and cerebral perfusion selectively.

appendage is gently grasped between the left thumb and index finger. Scissors are used to create a small opening in the atrial appendage, and a previously flushed catheter is inserted into the right atrium. The umbilical cord clamp is placed over the catheter at the edge of the appendage and snapped shut (Fig. 17-14 B). The cord clamp holds the catheter securely, provides good hemostasis, and does not interfere with the operative field.

## Aortic Cross-clamping

When the systolic pressure cannot be raised above 70 mm Hg, temporary occlusion of the descending thoracic aorta will maintain myocardial and cerebral perfusion (Fig. 17-15) . Dunn and colleagues have demonstrated, using a canine shock model, that aortic cross-clamping is more beneficial to an ischemic heart because of enhanced coronary perfusion than it is detrimental because of increased afterload. [64] Selective clamping is necessary when the aorta has been injured with blunt trauma (Fig. 17-16) . Aortic occlusion has a limited role in controlling hemorrhage below the diaphragm. When there is a tense abdomen with massive hemoperitoneum, aortic cross-clamping is

clearly beneficial when applied just before laparotomy. This has been referred to as *prophylactic cross-clamping* to prevent a sudden drop in blood

pressure when the abdomen is decompressed. [65] As a preoperative procedure, cross-clamping should be applied when the systolic pressure is less than 80 mm Hg in the setting of a tense abdomen.

The aorta lies most immediately anterior to the vertebrae, actually lying on the vertebral bodies themselves. The esophagus lies anterior and slightly medial to the aorta (Fig. 17-17 *A*). To expose the descending aorta, the left lung is retracted in a superomedial direction by an assistant. To achieve adequate exposure, it is sometimes necessary to divide the inferior pulmonary ligament (Fig. 17-17 *B*). The aorta can be quickly identified by advancement of the fingers of the left hand along the thoracic cage toward the vertebral column. On some occasions, the operator may choose to have an assistant simply occlude the aorta with digital pressure. Because both the aorta and esophagus are covered on their anterolateral surface by mediastinal pleura, the pleura must be opened and the aorta bluntly dissected away from the esophagus prior to clamping. To locate the aorta, one uses a DeBakey aortic clamp or a curved Kelly clamp for blunt dissection and spreads open the pleura above and below the aorta (Fig. 17-18) . The esophagus, which lies medially and slightly anteriorly, is separated from the vessel. It may be difficult to separate the esophagus from the aorta by feel in a hypotensive or cardiac arrest situation. A nasogastric tube passed from above may help identify the esophagus. When the aorta is completely isolated, the index finger of the left hand is flexed around the vessel and a vascular clamp is applied with the right hand. The brachial blood pressure should be checked immediately after the occlusion. If the systolic pressure is more than 120 mm Hg, the clamp should be slowly released and adjusted to maintain a systolic pressure of 120 mm Hg. [31]

Given the need for speedy intervention, the simplest and most desirable approach to aortic occlusion is to have an assistant digitally compress it or use the aortic tamponade instrument (Fig. 17-19) (Figure Not Available) . The application of vascular clamps is fraught with complications, which include inadvertent

dislodgment or inadequate occlusion as a result of improper application. The aortic tamponade instrument, however, may be applied blindly to the vertebral column, permitting safe, quick, and complete aortic occlusion. [66] This technique may be the most prudent when isolation of the aorta is difficult. The instrument's unique shape allows it to remain in place and to provide atraumatic occlusion with little interference in the operative field compared to digital compression. The degree of occlusion can be

varied by the amount of pressure exerted by the operator.

Potential complications of aortic cross-clamping are multiple: ischemia of the spinal cord, liver, bowel, and kidneys as well as iatrogenic injury of the aorta and the esophagus may occur. Failure to monitor blood pressure every 60 seconds during aortic occlusion may result in cerebral hemorrhage or left ventricular failure if pressure elevation is excessive. Fortunately, these complications are infrequent. In a report of 12 patients surviving ED thoracotomy with cross-clamping for as long as 60 minutes, no lasting impairments of renal, myocardial, or neurologic function were noted. [67] Whenever possible, the aorta was unclamped for 30 to 60 seconds every 10 minutes to increase renal perfusion. Final release of the aorta is always performed gradually.

## INTERPRETATION AND HEMODYNAMIC MONITORING

Following emergency thoracotomy, the systolic blood pressure after the first 30 minutes of resuscitation may be used as a decision point for further treatment. A report of ED thoracotomies for blunt and penetrating trauma from Denver General Hospital related the hemodynamic response to thoracotomy to patient outcome. [68] Of the 146 cases reviewed, 45 patients (31%) were transferred to the operating room following initial resuscitation and aortic cross-clamping when necessary. For those patients who survived with full neurologic recovery, the average systolic blood pressure after the first 30 minutes of resuscitation was 110 mm Hg. In those who were long-term survivors but had significant brain damage, the average systolic blood pressure was 85 mm Hg. No survivals were recorded when the mean systolic blood pressure was less than 70 mm Hg. Thus, the blood pressure response to emergency thoracotomy is predictive of survival. For patients who remain lifeless with systolic blood pressures below 70 mm Hg despite control of hemorrhage, volume replacement, and cross-clamping for 30 minutes, Moore and colleagues recommend that heroic measures be discontinued. [68] Transfer of these patients to the operating suite for definitive repair would be nonproductive.

## COMPLICATIONS

A variety of postoperative complications may occur in patients surviving emergency thoracotomy. Most of these complications stem from the particular injuries of each patient and must be considered on an individual basis. The complications of open-chest resuscitation are relatively insignificant when compared with a fatal outcome. Two post-thoracotomy complications are frequently discussed. One is thoracic sepsis. This is a rare complication, and excessive concern with

**Figure 17-18** A, Identification of the aorta: in the posterior mediastinum the aorta lies directly anterior to the vertebral bodies. The esophagus is anterior and slightly medial to the aorta. In the lower thorax, both are covered on the anterolateral surface by mediastinal pleura, which must be dissected prior to isolating the aorta for cross-clamping. *B,* Aortic cross-clamping: using blunt dissection, one spreads the pleura above and below the aorta. The vessel should be fully mobilized and clearly separated from the

esophagus before clamping. It may be difficult to differentiate the aorta from the esophagus. The aorta is the more posterior structure and is in contact with the vertebral bodies. Passage of a nasogastric tube from above will often aid the rapid identification of the esophagus.

antiseptic skin preparation may unnecessarily delay a thoracotomy. [69] In a combined series of 142 ED thoracotomies, there were no reports of wound infections. It should be noted that most patients received antibiotics just before or

**Figure 17-19** (Figure Not Available) The use of a Conn aortic compressor is the method of choice for aortic occlusion, because it is fast, does not interfere with the operative field, and is associated with minimal risk of injury. Alternatively, the more awkward technique of direct digital occlusion can be employed. *(Courtesy of Pilling Company, Ft Washington, Pa.)*

during the procedure. Thus, the concern for infection as a complication of a thoracotomy performed with less than sterile technique is unwarranted, although a short course of antibiotics should be administered as soon as possible.

A second complication that is frequently feared and may unfortunately deter a life-saving thoracotomy is the imagined threat that the patient will survive but will be in a vegetative neurologic state. This complication is also rare, and such apprehension appears unjustified. Likewise, the concern of "tying up intensive care unit beds" with patients who have "fatal" injuries has been overemphasized. The first 24 hours following injury will rapidly demonstrate which patients will become long-term survivors. The San Francisco experience with 168 emergency thoracotomies for mixed trauma illustrates that most patients with fatal injuries die within 24 hours. [48] Of patients surviving the first 24 hours, 80% (33 of 41) recovered and left the hospital. Full neurologic recovery occurred for 90% of these survivors. Overall, only 2.4% (4 of 168) remained severely disabled or in a persistent vegetative state. Of these 4 patients, only 1 (0.6%) lived beyond 2 months; he eventually died at 14 months from sepsis.

Moore and colleagues in Denver, describing 146 emergency thoracotomies for mixed trauma, reported 15 patients who survived resuscitation in the operating suite. Eighty percent (12 of 15) of these patients went on to become long-term survivors; 75% had full neurologic recovery. In the Denver study, 2 valuable observations were made regarding the presence or absence of various signs. First, all survivors

with full neurologic recovery had respiratory efforts at the scene; in 75% of these patients, respiratory efforts were still present on arrival in the emergency care unit. Second, the presence or absence of a palpable pulse is not an absolute prognostic indicator. Sixty-six percent of long-term survivors (11 patients with penetrating trauma and 1 with blunt trauma) had no detectable pulse on arrival in the emergency care unit. [68]

A final complication is injury to the resuscitative team. In an emotionally charged environment in which many physicians are attempting to perform life-saving surgery under the harshest of conditions, it is easy to suffer a needle stick or scalpel or scissors injury. Because some victims of penetrating injuries are HIV positive, the need for extreme caution is obvious. Because thoracotomy exposes personnel to a large amount

of blood, universal precautions should be rigorously followed.

## CONCLUSION

The growing incidence of penetrating trauma coupled with advances in out-of-hospital care have increased the number of patients who require advanced resuscitation. For selected patients, an emergency thoracotomy will substantially reduce morbidity and mortality. The effectiveness of this procedure for resuscitation has been well documented. The mechanism of injury and the status of "vital signs at the scene" and on arrival at the hospital should be considered before an emergency thoracotomy is performed. Victims of penetrating trauma who had any vital signs at the scene are candidates for resuscitative thoracotomy and have a chance of survival. This procedure is most effective when it is used to prevent, rather than to treat, a cardiac arrest. In contrast, the survival rates for victims of blunt trauma are negligible. With blunt trauma, resuscitative thoracotomy generally should be considered only when vital signs are still present on arrival at the emergency care unit.

Five factors are closely associated with patient outcome following resuscitative thoracotomy: (1) mechanism of injury; (2) ECG activity; (3) presence or absence of pulse and respiratory effort at the scene and on admission; (4) presence of neurologic function at the scene and on admission; and (5) systolic blood pressure after aortic cross-clamping. The following guidelines should be considered:

1. Blunt trauma victims who lose their vital signs en route to the ED rarely survive despite thoracotomy, and they generally should not be candidates for resuscitative thoracotomy.
2. Penetrating trauma victims who lose their vital signs en route to the ED may still survive and should receive an immediate thoracotomy.
3. Pericardiocentesis (or possibly a subxiphoid pericardial window) should be considered when the systolic blood pressure cannot be maintained above 60 mm Hg with aggressive management and while preparations are made for resuscitative thoracotomy (see Chapter 15) .
4. Further efforts generally should be discontinued following thoracotomy when traumatic arrest patients do not exhibit cardiac activity or tamponade.
5. Consider discontinuing further efforts following thoracotomy if the systolic pressure cannot be raised above 70 mm Hg after 30 minutes of maximum management.

Debate about who should perform an emergency thoracotomy is not necessary. It stands to reason that whoever uses this resuscitative procedure must be prepared to manage the patient. Emergency physicians must possess the knowledge and skills necessary to enable the optimal survival of as many patients as possible. However, it is currently *not* a standard of care that all EDs have the capability of performing an emergency thoracotomy in the absence of surgical backup. Because aortic cross-clamping is only temporizing and should not exceed 30 minutes, rapid transfer of the thoracotomy patient to the operating room is desirable. Whenever possible, a preestablished plan of chest wound management and post-thoracotomy care should be established with the emergency physician's surgical backup. With such a plan, a team

approach to resuscitation and, hence, optimal patient care is possible.

# Chapter 18 - Pediatric Vascular Access and Blood Sampling Techniques

**Frank J. Cunningham Jr.** , **William A. Engle,  Frederick J. Rescorla**

The tasks of sampling blood and obtaining vascular access in an infant or child can challenge and frustrate even the most skilled emergency physician. Unsuccessful attempts on tiny or seemingly nonexistent veins or arteries can consume valuable time, especially when dealing with a critically ill or injured child. The use of invasive monitoring techniques with arterial and central venous catheters is commonplace in contemporary pediatric emergency and intensive care.

This chapter reviews the basic principles and techniques of blood sampling, as well as selection and placement of intravenous (IV) and intra-arterial catheters in infants and children. The use of umbilical catheters in newborns is also reviewed. Although rarely required, emergency cutdown is occasionally life saving, and a section of the chapter is devoted to this technique. Many patients require long-term parenteral nutrition delivered through a centrally placed venous catheter; the techniques for placement of these catheters from peripheral and central venous insertion sites are also described.

## PATIENT PREPARATION AND RESTRAINT

The fear of pain associated with painful procedures and injections makes the hospital experience a traumatic one for children. Therefore, prior to beginning any painful procedure in a child, the procedure itself and the reasons for it should be explained to the parents. In children capable of understanding, the procedure should be explained in developmentally appropriate language before starting and prior to each successive step. The use of deceptive phrases such as "This won't hurt" should be avoided. A simple and honest explanation that the procedure will hurt a bit and "it is okay to cry, but not to move" will provide realistic expectations for the child and set limits as well. Depending on the procedure and the preferences of the physician and the family, the parents may be permitted and willing to remain in the room with the child during the procedure. [1] They should have this option. If they remain during the procedure, their role should be solely to provide comfort and solace to the child and not to assist in any potentially painful procedure. Distracting the child with simple conversation regarding school, friends, hobbies, pets, or television shows can also decrease the level of the child's anxiety. Parents can become patients themselves, and despite their seeming braveness or composure, the potential for parents to faint at the site of blood or needles should always be addressed. Parental injury under such circumstances can be a source of litigation against the physician.

The success of painful procedures such as blood sampling or obtaining vascular access is dependent on proper positioning and restraint of the patient. In most cases this requires the assistance of at least 1 other person. For most procedures this entails restraint of the extremity a joint above and below the intended insertion site. The topical use of lidocaine-prilocaine (Emla) cream has proven particularly useful in relieving pain

associated with needle puncture. [2] [3] However, optimal analgesic results require application 1 hour prior to the procedure, which may not be satisfactory in some clinical settings or in busy emergency departments (see Chapter 31) .

# BLOOD SAMPLING TECHNIQUES

Blood samples for biochemical and hematologic analyses and blood gas analyses may be obtained from indwelling vascular lines. However, capillary, arterial, and venous blood sampling are the principal methods for obtaining blood samples (especially blood cultures) from patients presenting with acute medical or surgical problems. In the sick newborn and young infant, procuring blood samples may be difficult, because many clinicians are not experienced in the techniques of blood sampling in this age group. [4] This section reviews these techniques as they pertain to the newborn and young infant.

## Capillary Blood Sampling

### Indications and Contraindications

Capillary blood sampling, or heel stick puncture, is a frequently used technique to obtain blood samples in young infants. In older children and adults, this technique may be used to obtain blood samples from the finger, toes, and ear lobe. It is most appropriate for patients who require repeated sampling, because the number of times small arteries and veins can be entered successfully is limited, as is the total number of vessels available in small infants. Capillary blood sampling is most often indicated whenever an adequate sample of blood can be obtained by the heel stick puncture technique and when an alternative technique (i.e., indwelling catheter) is not more readily available. It is especially useful for obtaining "arterialized" blood for blood gas analysis when arterial access is unavailable, as in many chronically ill neonates and young infants. Noninvasive monitoring techniques such as pulse oximetry and transcutaneous oxygen monitoring (see Chapter 6) have reduced the frequency with which these samples must be taken.

Sampling from an area of local inflammation or hematoma should be avoided. Also, repetitive sampling from the same site may induce inflammation and subsequent scarring and hence should be avoided. In general, heel stick sampling is not ideal for blood gas analysis (1) when the infant is hypotensive, (2) when the heel is markedly bruised, or (3) when there is evidence of peripheral vasoconstriction. Capillary blood does not always produce an accurate analysis of arterial $P_{O2}$ . When the capillary $P_{O2}$ is >60 mm Hg, the arterial $P_{O2}$ may be considerably higher, with possibly dangerous consequences to infants receiving supplemental oxygen. In this situation the use of either a transcutaneous oxygen saturation or a transcutaneous $P_{O2}$ monitor may allow adjustment of the inspired oxygen concentration until either an arterial $P_{O2}$ or a repeat capillary $P_{O2}$ can be obtained. [5] [6]

### Equipment and Setup

The necessary equipment for capillary blood sampling is shown in Table 18-1 . A 3-mm

lancet (Becton-Dickinson, Rutherford, NJ) should be used to perform this procedure; a scalpel blade should never be used. The use of a 3-mm lancet will prevent the puncture from penetrating more than the maximum safe distance. The full 3 mm of the lancet should be used; a more superficial incision will not bleed adequately. Blood collection is performed using either heparinized capillary tubes or 1-mL Microtainer tubes with a collector attachment (Becton-Dickinson, Rutherford, NJ). If capillary tubes are used, a clay sealer will be needed to close off one end.

| **TABLE 18-1** -- Equipment for Capillary Blood Sampling |
| --- |
| Warm wet towel or diaper |
| Alcohol pads |
| Lancets |
| Blood collection tubes (capillary tubes or Microtainers) |
| Clay sealer (used with capillary tubes) |
| Sterile bandage |
| Nonsterile examination gloves |

**Figure 18-1** *A,* The heel stick is performed on the lateral or medial aspect of the heel. *B,* The collector end of the Microtainer is touched to the drop of blood, and blood is allowed to flow down the wall of the tube to the bottom. Avoid squeezing the foot, and keep the proximal end of the Microtainer below the

**Technique**

Although capillary blood sampling may be performed by the finger stick or heel stick methods, the latter will be described. The recommended sites for heel stick puncture are the medial-most and lateral-most portions of the plantar surface of the heel. [7] This avoids penetration of the calcaneus and the risk of osteochondritis. Prewarming the foot in a warm towel or disposable diaper soaked with warm water for approximately 5 minutes will produce hyperemia and will enhance blood flow. The foot is immobilized in a dependent position with one hand. After the heel is cleansed with alcohol and allowed to dry, the skin is punctured with the lancet (Fig. 18-1) . Although it is tempting and common to do so, *squeezing of the foot should be avoided*, since this may inhibit capillary filling and may actually decrease blood flow. Furthermore, squeezing may dilute the sample with serum or tissue fluid and make analysis less accurate. If blood does not flow freely, another puncture may be required.

The first small drop of blood is wiped away with gauze, and another drop is allowed to form. A heparinized capillary tube is placed in the drop of blood, and the proximal end of the inverted tube is allowed to fill by capillary action. The tube (or tubes, if several tests will be needed) is sealed at one end by sticking the end into wax before the tube is sent to the laboratory. If 1-mL Microtainer tubes are used, the tube is held at an angle of 30° to 45° from the surface of the puncture site. The collector end is touched to the drop of blood, and blood is allowed to drain into the tube. Gentle tapping of the tube will facilitate flow to the bottom of the tube. For heparinized tubes, blood should be collected until the level reaches the demarcation on the tube. Over- or underfilling of the tube may result in clotting and/or erroneous test results. Once an adequate specimen is obtained, a dry dressing is applied to the puncture site.

When a heel stick is performed for arterialized blood samples, the technique used is similar to that discussed previously for routine blood sampling, with the following differences: The infant's foot must be wrapped with a warm cloth for a few minutes. The first drop of blood must be

discarded and the remaining blood allowed to flow freely into a heparinized capillary tube. The tip of the tube should be placed as near the puncture site as possible to minimize exposure of the blood to environmental oxygen. Collection of air in the tube as well as excessive squeezing of the foot should be avoided, because this may artificially lower the $P_{O_2}$ . Approximately 0.2 to 0.3 mL of blood should be collected in the heparinized capillary tube.

**Complications**

When properly performed, heel sticks are associated with a low incidence of complications. Lacerations should not occur when the procedure is performed with a lancet rather than a scalpel blade. Heel sticks may cause infection (local infection, bacteremia, or osteomyelitis), scarring, and calcified nodules. [8] [9] [10] When the heel stick technique is used for the procurement of "arterialized" blood for pH, $PCO_2$ , and

P$o_2$ analysis, the most important potential error is that false information (inaccurate P$o_2$) may result in exposure of the infant to improper amounts of supplemental oxygen.

### Interpretation

Numerous studies have compared the reliability of capillary blood with that of arterial blood for determination of pH, P$CO_2$, and P$o_2$. [11] [12] [13] [14] [15] Although the results have been quite variable, most investigations have documented a close correlation between arterial and capillary samples for pH and P$CO_2$ determinations (except when the patient is in shock or has an extremely high P$CO_2$). Unfortunately, the P$o_2$ determination has not been found to be as reliable when performed on blood obtained by capillary or "arterialized" sampling. Most studies indicate that the capillary (heel stick) P$o_2$ correlates poorly with the arterial P$o_2$, especially if the arterial P$o_2$ is >60 mm Hg. For example, a capillary P$o_2$ of 70 mm Hg may reflect an arterial P$o_2$ of 70 to 200 mm Hg. In nearly all situations, the capillary P$o_2$ is equal to or less than the arterial P$o_2$, but in any individual case, one does not know how closely the capillary value approximates the arterial level. Therefore, *reliance on a capillary sample of blood for P$o_2$ measurement in an acutely sick infant may be fraught with potential risks*. "Arterialized" blood samples obtained from finger and toe sticks might be more reliable for P$o_2$ determination than those obtained from heel sticks, but again, the data are controversial. [16] [17] [18]

## Venipuncture

### Indications and Contraindications

Although many laboratory tests for the small infant may be performed on blood obtained by heel sticks, larger volumes of blood may be required, making heel sticks impractical. Venipuncture is the usual method used for obtaining larger quantities of blood as well as samples for blood culture. Femoral venipuncture for blood samples is discouraged, especially in children <1 year of age, due to the risk of septic arthritis of the hip. [19] In an emergency setting or when few venous access sites exist, blood for laboratory analysis may be obtained from an arterial puncture.

### Equipment and Setup

The equipment required for venipuncture in an infant or child is listed in Table 18-2 . A small-gauge butterfly needle

| **TABLE 18-2** -- Equipment for Venous Blood Sampling in Infants and Children |
| --- |
| A 3-, 5-, or 10-mL syringe |

| |
|---|
| A 21- or 23-ga butterfly needle |
| Evacuated blood tubes |
| Tourniquet (small drainage tubing for infants and children; elastic band for newborns and small infants) |
| Alcohol pads |
| Povidone-iodine swabs (if blood cultures are needed) |
| Sterile 2 × 2-in. or 4 × 4-in. gauze pads |
| Nonsterile examination gloves |

is usually preferred over a needle and syringe for obtaining blood in infants. It is difficult to manipulate standard needles and syringes in tiny veins, and better control is obtained with the butterfly needle. Suction is also more controlled with the butterfly needle and syringe. The butterfly needle may also serve as an infusion line once adequate amounts of blood are obtained. If a straight needle and syringe are used, the technique is similar to that described later for percutaneous arterial puncture, except that a peripheral vein is punctured. The 23-ga butterfly needle will suffice in most venipunctures regardless of age group. In older children and adolescents, the Vacutainer system (Becton-Dickinson, Rutherford, NJ) can be used. However, the negative pressure within the evacuated blood tube, once connected, may be sufficient to collapse the punctured vein. Therefore, a sterile syringe with a 23-ga butterfly attached is preferred. Likewise, a 3- or 5-mL syringe is less likely than a 10-mL syringe to cause vein collapse in young infants. Appropriate restraints and assistance are needed with most pediatric procedures.

**Technique**

Like adults, the usual site for venipuncture in infants and children is the antecubital fossa. However, any reasonably accessible or easily visible peripheral vein (e.g., on the scalp, the hands, or the feet) may be used (Fig. 18-2) . Veins on the dorsum of the hand

can be used, providing they will not be needed for IV cannulation. The external jugular veins and femoral veins are rarely used. When venous access is unavailable, arterial blood may be used for most laboratory tests, including blood cultures.

All needed equipment should be assembled and ready for immediate use. Drawing blood from a small infant is usually a 2-person procedure. It should be emphasized that immobilization of the extremity is mandatory. An assistant should withdraw the blood while the physician concentrates on keeping the needle within the vein and immobilizing the arm. The butterfly needle and the syringe can be attached either before or after skin penetration. However, if done before, the assembly should take place out of sight of the child. To minimize the number of venipuncture attempts, the optimal site for needle insertion should be chosen after a survey of the most prominent peripheral veins. If an extremity vein is to be used, a tourniquet should be applied proximal to the selected vein; in small infants, a rubber band will serve as an adequate tourniquet, but one must be certain to remove the rubber band following venipuncture.

The tourniquet should not be so tight as to impede arterial filling. The area surrounding the chosen site of skin penetration is cleansed with alcohol and allowed to dry.

**Figure 18-2** Venous access sites in the neonate and young infant. If venous access is unavailable, arterial blood may be used for most laboratory tests, including blood cultures.

Slight distal traction is applied to the skin to immobilize the vein, and the needle is inserted quickly through the skin and slowly into the vein at an angle of approximately 30°, with the bevel up (Fig. 18-3 *A*). Successful venous penetration is heralded by a flashback, or flow of blood into the butterfly tubing. *Gentle suction is applied by slowly withdrawing the plunger of the syringe.* Alternatively, suction may be applied with a Vacutainer system in which the Vacutainer needle punctures the sealed end of the butterfly device (see Fig. 18-3 *B*). If the suction is excessive, the vein will collapse, and blood flow will stop. If the required amount of blood is more than the capacity of the attached syringe, the tubing is pinched off, the filled syringe is removed, the next syringe is attached, and gentle suction is once again applied after release of the pinched tubing. After the required amount of blood is withdrawn, the needle is removed, and a sterile dressing and direct pressure are applied to the puncture site.

Although peripheral venous sampling (antecubital, scalp, hand) and arterial sampling are preferable, the external jugular and femoral veins may be used in infants for the performance of a venipuncture during resuscitations or when peripheral sites are inadequate. The external jugular vein lies in a line from the angle of the jaw to the middle of the clavicle and is usually visible on the surface of the skin. The vein is more prominent when the infant is crying. An assistant is needed to restrain the infant in a supine position with the head and neck extended over the edge of the bed. Alternatively, a towel roll or pillow placed under the shoulders can be used. The head is turned approximately 40° to 70° from the midline (Fig. 18-4) , and the skin surrounding the area to be punctured is cleansed with alcohol. Lidocaine (0.1 mL of a 1% solution) may then be infiltrated into the skin. A finger may be placed just above the clavicle to distend the jugular vein. Using a 21- to 25-ga straight needle with a syringe or a 21- to 25-ga butterfly needle attached to a syringe, the clinician punctures the skin and advances the

needle slowly until the jugular vein is entered. The syringe is connected to the needle at all times to maintain a constant negative pressure and avoid an air embolism. After the appropriate amount of blood is obtained, the needle is withdrawn, and slight pressure is applied to the vessel. The infant should be placed in an upright position after the needle is removed, and slight pressure should be continued for 3 to 5 minutes. Close observation of the puncture site should follow.

The femoral vein lies medial to the femoral artery and inferolateral to the inguinal ligament (Fig. 18-5) (Figure Not Available) . The use of this vein for blood sampling is reserved for situations in which patients present in extremis and no other sampling sites are present. An assistant positions the hips in mild abduction and extension while the artery is palpated and its location identified by placing a mark on the abdomen just superior to the femoral triangle (see Fig. 18-5) (Figure Not Available) . The femoral

**Figure 18-3** *A,* Technique for obtaining blood by antecubital venipuncture with a butterfly needle and a syringe. Once blood is obtained, the butterfly needle may serve as an infusion site. Note that this procedure often requires 2 persons to carry it out--one to hold the arm and insert the needle and the other to aspirate the blood. *B,* As an alternative to a syringe, a Vacutainer system may be used to apply suction. The Vacutainer needle punctures the sealed end of the butterfly set. Use of this method helps to prevent the premature clotting of blood that may occur if there is a delay in filling the collection tubes.

**Figure 18-4** External jugular venipuncture. A syringe or a butterfly needle may be used. Venous distention is aided when an assistant's finger occludes the vein, or when the infant cries. The neck is extended, either over the side of the bed or by placing a rolled towel under the shoulders. This procedure requires 2 persons to perform it. Gloves should be worn.

triangle is then prepared with alcohol; a povidone-iodine scrub is also recommended when obtaining blood cultures. The skin puncture site may then be infiltrated with 0.1 mL of 1% lidocaine. The technique of needle insertion is similar to that for external jugular venipuncture (see Fig. 18-4) . The clinician punctures the skin and then directs the needle or catheter toward the umbilicus at a 30° to 45° angle to the skin, remaining medial to the femoral artery pulsation. A slight constant negative pressure is applied throughout insertion. After the needle enters the femoral vein, the desired blood samples are withdrawn, and the needle or catheter is removed (unless venous access with an IV catheter is desired). Pressure is applied to the femoral triangle for a minimum of 5 minutes, and the site is observed closely for recurrent bleeding.

**Complications**

Complications of venipuncture include hematoma formation, local infection, injury to structures adjacent to vessels, and phlebitis. All of these complications are uncommon. Special care should be used when puncture of the external jugular vein or femoral vein is attempted. Inadvertent deep puncture in the neck can produce injury to the carotid artery, the vagus or phrenic nerve, or the apex of the lung. In the femoral triangle, injury to the femoral artery, femoral nerve, and hip capsule may occur. [19] [20] [21] However, such structures are unlikely to be injured when proper technique is practiced.

## Blood Cultures

Although the heel stick capillary tube procedure has been used in some centers for the procurement of blood from infants for cultures, [22] [23] there is a significant incidence of false-positive results with the technique, and therefore it is not recommended if venous blood is available. Venipunctures continue to be the main source of blood for culture in small infants. In the newborn infant, blood may be obtained for culture from an umbilical arterial or venous catheter, if it is obtained immediately after sterile insertion; even then, there is considerable controversy concerning the incidence of false-positive cultures. Arterial blood may also be used to obtain blood cultures.

**Figure 18-5** (Figure Not Available) *A,* Anatomy of the femoral triangle. The vein is always medial to the artery. *B,* The needle insertion site is located 1 finger width below the inguinal ligament, just medial to the artery. Use the index and middle finger to identify the course of the femoral artery. The needle is pointed medially toward the umbilicus at a 45° angle from the skin surface. *(From Pediatric Advanced Life Support Drug Lecture Slides, 1990. Reproduced with permission. Copyright American Heart Association.)*

### Technique

The technique of venipuncture for a blood culture is similar to that described previously for general blood sampling, with the following differences: The puncture site should be doubly prepared, first with a povidone-iodine solution (allowed to dry completely) or with alcohol applied vigorously for 30 seconds (see Chapter 74) . Following completion of the procedure, all of the iodine solution should be removed from the infant's skin to prevent irritation. The volume of blood required for a blood culture depends on the size of the infant. In the neonate with bacteremia, there is a greater number of organisms per milliliter of blood [24] ; a sample size of 0.5 to 1 mL is probably sufficient. In older infants, 2 to 3 mL of blood is ideal. After the appropriate volume of blood is withdrawn, the needle that was used to penetrate the skin is removed, and a sterile needle is attached to the syringe. Half of the specimen should be placed in an anaerobic culture bottle and half in an aerobic bottle.

## Arterial Blood Sampling

### Indications and Contraindications

Arterial blood gas evaluation provides useful information that is essentially unavailable by other means (see Chapter 19) . The arterial blood gas determination is an important laboratory test for evaluation of respiratory status and acid-base equilibrium in infants or children in respiratory distress or shock or with possible medication overdosage. Arterial blood may also be used for routine laboratory analysis if venous blood is difficult to obtain. In fact, if venous access is unavailable, an artery may be used to obtain all laboratory specimens. Possible sites for arterial blood sampling include (1) radial, brachial, temporal, dorsalis pedis, and posterior tibial arteries; (2) umbilical arteries in the newborn infant; and (3) capillaries ("arterialized"). The ulnar artery should not be used for arterial puncture to preserve the collateral circulation to the hand, although some clinicians advocate performing punctures and catheterization of the ulnar artery.

No vein or nerve is immediately adjacent to the radial artery, which minimizes the risk of obtaining venous blood or damaging a nerve. This is not the case with the brachial artery, and the risk of both complications appears to be greater when this artery is used. [25]

The temporal artery is also adjacent to a vein, and if the patient's head is in an oxygen hood, it is nearly impossible to obtain a sample in a steady state. Femoral arteries should not be used for obtaining routine blood samples from the infant or child. [26] Transcutaneous monitoring of $P_{O2}$, $P_{CO2}$, and oxygen saturation may provide useful adjuncts to arterial sampling in many patients. Nonetheless, they do not replace intermittent arterial sampling, which remains necessary for the stabilization of infants and children and for verification of the accuracy of these noninvasive methods. One should avoid puncture of an artery when infection, burn, or other damage to cutaneous defenses exists in the overlying skin.

**Equipment and Setup**

The equipment required for arterial puncture in an infant or child is listed in Table 18-3 . A small-gauge butterfly needle is usually preferred over a needle and syringe for arterial puncture in infants and children. As in venipuncture, a 23-ga butterfly needle is most often used, although in newborns, use of a 25-ga butterfly may be beneficial. Some clinicians prefer to use a 25-ga needle connected to a syringe, but use of a butterfly allows for better control of the needle while an assistant aspirates the syringe and may also permit a larger volume of blood to be withdrawn.

| **TABLE 18-3** -- Equipment for Arterial Blood Sampling in Infants and Children |
| --- |
| Heparin flush solution (10 units/mL) |
| A 1- or 3-mL Tuberculin syringe |
| A 23- or 25-ga butterfly needle |
| Povidone-iodine solution |

| |
|---|
| Alcohol pads |
| Ice-filled container (bag or cup) |
| Nonsterile examination gloves |
| Sterile 2 × 2-in. or 4 × 4-in. gauze pads |

Since the radial artery (Fig. 18-6) is most frequently used to obtain intermittent arterial samples from infants and children, the technique for it will be described. Although the merit of the Allen test (see Chapter 19) has been challenged, it is prudent to first assess the adequacy of collateral circulation in the hand. Both radial and ulnar arteries are compressed. After a short time, compression of the ulnar artery is released while the radial artery remains compressed. Adequacy of collateral flow to the hand by the ulnar artery is marked by return of color to the hand. One should then heparinize a tuberculin syringe. All heparin should be ejected from the syringe; a 23- or 25-ga butterfly needle should then be attached to the syringe. The amount of heparin coating the barrel of the syringe is adequate to anticoagulate the sample; excess heparin may result in inaccurate $P_{CO2}$ determinations because of dilution of the blood sample. [27] [28]

The clinician should hold the infant's wrist and hand in the left hand (if the clinician is right-handed). The child's hand is held fully supinated with the wrist dorsiflexed. Some clinicians prefer to immobilize the wrist by taping it to a sandbag or another restraint. [29] The pulsations of the radial artery should be palpable just proximal to the transverse wrist creases. A small indentation can be made in the skin with a fingernail to mark the insertion site. The area is cleansed with alcohol. Some practitioners advocate the use of an intradermal wheal of lidocaine. The skin is penetrated at a 30° to 45° angle (Fig. 18-7) , and while the plunger of the syringe is withdrawn, the needle is advanced slowly until the radial artery is punctured or until resistance is met (Fig. 18-8) . In contrast to the procedure in adults, it is necessary in infants to provide continuous suction on the plunger of the syringe. One can be sure that the radial artery is punctured when blood appears in the hub of the needle. Other clinicians prefer to attach the syringe to the butterfly needle only after blood return is noted; suction is thereafter applied. One may place a transilluminator on the underside (dorsum) of the neonate's wrist to visualize the radial artery. [29]

**Figure 18-7** For arterial blood sampling, the needle should be inserted under the skin at a 30° to 45° angle. A butterfly needle and syringe are used if larger volumes of blood are required. The wrist is held dorsiflexed by the nondominant hand.

If resistance is met while pushing the needle deeper, the needle is slowly withdrawn to the point at which only the distal needle tip remains beneath the skin, and then the procedure is repeated. After 0.3 mL of blood is obtained, the needle is removed, and light pressure is applied for 5 minutes or longer to prevent any bleeding. [30]

A simulator (Medical Plastics Laboratory, Gatesville, Texas) may be used to teach and practice the technique of radial artery puncture in infants. [30]

### Complications

The complications of radial artery puncture include infection, hematoma formation, scar formation, tendon injury, arteriovenous fistula formation, and nerve damage. [31] [32] With the use of proper technique, however, the complication rate is extremely low. The most common concern with puncture of a radial artery (or any peripheral artery) in infants is that the infant may start to cry before blood is obtained, thus changing the $P_{O_2}$ and $P_{CO_2}$ from the values of the quiet state. [33] [34]

Another potential problem is the dilutional effect of heparin on the $P_{CO_2}$. The heparin in the dead space of the

**Figure 18-8** Resistance met during passage of the blood gas needle usually indicates contact with bone. The needle should be withdrawn slowly. If the needle has traversed both walls of the artery, blood will be obtained as the needle is slowly withdrawn into the arterial lumen.

tuberculin syringe may decrease the $P_{CO_2}$ by 15% to 25% when 0.2 mL of blood is obtained and by approximately 10% with 0.4 mL of blood. This emphasizes the need for all heparin to be ejected from the dead space of the syringe *before the needle is applied*. The use of a syringe (e.g., Becton-Dickinson 1-mL U-100 insulin syringe) with minimal dead space or the use of lyophilized heparin eliminates this problem (see Chapter 19)

## VASCULAR LINE PLACEMENT: VENOUS AND ARTERIAL

Intravascular lines are indicated when access to the venous or arterial circulations is necessary. An IV line may be positioned in peripheral veins (scalp, hand, forearm, foot, ankle, axilla, thigh) or central veins (superior vena cava via the internal jugular, axillary, superficial temporal, posterior auricular, or subclavian venous approach [36] [37] and the inferior vena cava via the umbilical or femoral venous approach) (Fig. 18-9) . Likewise, intra-arterial lines may be positioned peripherally (radial, posterior tibial, dorsalis pedis, or superficial temporal arteries) or centrally (abdominal or thoracic aorta via an umbilical or femoral artery approach). Techniques to secure access to these intravascular spaces are discussed in the following sections (see Chapters 19 and 20) . Note that 50% nitrous oxide in oxygen administration to the patient (see Chapter 35) may facilitate these vascular procedures by reducing pain and anxiety. [38] The use of lidocaine-prilocaine (Emla) cream has been shown to decrease the pain associated with

venipuncture and venous cannulation in children as well. [39] [40]

## Peripheral Venous Catheterization: Percutaneous

### Indications and Contraindications

In general, peripheral IV lines are indicated when the patient is unable to attain medical and nutritional goals with enteral therapy. These lines provide maintenance fluids to support adequate hydration and serve as a route for administering medications. In the acute setting, peripheral IV lines provide a route for administering resuscitative medications and fluids as well as antibiotics.

### Equipment and Setup

Materials needed for placement of a peripheral IV line in an infant are listed in Table 18-4 . The 2 devices commonly used for peripheral IV insertion are the butterfly needle and the plastic over-the-needle catheter. The former ranges in size from 21 to 27 ga. Butterfly needles are used primarily for infusions of short duration and are removed after completion. Examples of such use include certain chemotherapeutic agents as well as single-dose antibiotic administration. Due to their rigid nature, they tend to infiltrate very easily in the active child. Placement in a vein close to a flexor surface is contraindicated. For the most part, over-the-needle catheters, such as Angiocath, Medicut, or Quikcath, have become the mainstay of peripheral venous catheterization. These thin-walled, flexible catheters range in size from 14 to 24 ga. For infants, a 22- or 24-ga catheter will suffice in most cases. The selection of catheter size is dependent on the catheter's intended purpose. In general, the smallest gauge appropriate for the clinical situation should be used. Larger diameter catheters allow for rapid administration of fluids in emergency situations. The use of T-connector extension tubing connected to the catheter after insertion facilitates withdrawal of blood for specimen collection, makes flushing the catheter and maintaining patency easier (especially while taping and securing the IV), and allows for dressing changes without disturbing the IV dressing. [41]

The clear plastic wrapper of the extension tubing package can be taped over the hub of the catheter as a protective covering to prevent accidental dislodgment when snagged on clothing or bed linen or subjected to the wandering

| TABLE 18-4 -- Equipment for Peripheral IV Insertion in Infants and Children |
| --- |
| 22- or 24-ga venous catheters |
| Tourniquet (rubber band for infants) |

| |
|---|
| IV solution and tubing |
| T-connector extension set |
| Pretorn tape (½-, 1-, and 2-inch) |
| Alcohol pads |
| Povidone-iodine swabs (for blood culture) |
| Arm or leg board |
| Non-sterile examination gloves |
| Sterile 2 × 2-in. or 4 × 4-in. gauze pads |
| Protective covering (container from IV catheter or T-extension set) |
| IV fluid chamber with microdrip |
| A continuous infusion pump |
| Saline flush solution |

| 3- or 5-mL syringes |
| --- |

fingers of an inquisitive child. An arm or leg board appropriate for the size of the child should be handy to provide for stabilization of the extremity after insertion. In newborns or small infants, fashioning an arm board from two tongue depressors taped together and covered with 4 × 4-inch gauze will provide the appropriate length needed. One should have primed and ready an IV fluid chamber with microdrip and a continuous infusion pump. Fluid administration in an infant must be carefully monitored. *Macrodrip tubing and liter bottles should not be used*; inadvertent infusion of large amounts of fluids in an infant may be disastrous. An infusion pump is an ideal way of limiting fluid infusion while keeping the vein open.

**Technique**

A number of IV sites are available for placement of a peripheral IV needle or catheter in the infant (see Fig. 18-2) . [36] [37] The most common sites chosen for IV insertion in infants and children are the superficial veins of the dorsum of the hand; the antecubital fossa; the dorsum of the foot; and, in newborns and small infants, the scalp. The veins of the dorsum of the hand are the most often used. These vessels are relatively straight and lay flat on the metacarpals and therefore are stabilized without undue difficulty. If the hand is chosen, one should take into consideration the age and hand preference of the patient. Veins in the antecubital fossa (cephalic and basilic veins) are easily accessible; however, their angulation across the fossa may make advancement of the catheter difficult. These veins may not be easily visible and yet may be palpable. It is recommended to select the most distal vein that is large enough to accommodate the catheter and leave the larger, more proximal veins in case (1) initial attempts are unsuccessful or (2) prolonged IV therapy may be needed and percutaneous central venous catheter placement is contemplated. Tributaries of the dorsal venous arch on the dorsum of the foot, like those on the dorsum of the hand, are relatively straight, and the extremity is easily immobilized after insertion. Because indwelling catheters in this location will prevent mobility, this site should be considered only in preambulatory patients or after attempts at other sites have been unsuccessful. The scalp veins are probably the easiest to cannulate, but their use is primarily limited to infants <1 year of age. If a peripheral vein on the hands, feet, or antecubital fossa is being used, the extremity can first be immobilized by taping it to an arm board, a padded splint, a full plastic IV fluid bag, or a sandbag. The particular site is a matter of preference, and the physician should choose the vein that appears to be the easiest to cannulate.

With few exceptions, the same techniques used for IV insertion in adults may be used in infants and children, especially in the veins of distal extremities. If a peripheral extremity is used, a tourniquet may be placed proximal to the planned site of entry. Although not in widespread use, nitroglycerin ointment (0.4-0.8 mg) is advocated by some physicians to induce local vasodilation, thereby aiding venous cannulation. [42] The tubing of the butterfly infusion set or the T-extension set should be flushed before venipuncture with a sterile IV solution, such as normal saline, to prevent air embolism. If a plastic catheter is

used, the catheter with stylet in place is directed through the skin at a 10° to 20° angle (see Fig. 18-9 A). [43] The catheter with stylet is slowly advanced until blood return is noted. One then advances

the catheter over the stylet into the vein. The stylet is removed, and the IV line is connected to the hub of the catheter by means of a T-extension set. After 1 mL of solution is flushed through the line, the site is inspected for signs of infiltration, such as hematoma or local swelling.

The catheter is fixed to the skin with a piece of 0.5 in. tape passed around and over the catheter hub and fixed to the skin. A second piece of tape is placed adhesive side uppermost under the catheter hub and crossed over the catheter in a V shape (see Fig. 18-9 B). The tubing of the T-extension set is looped back, and a piece of tape is placed midway over the tubing and secured to skin. This ensures against accidental dislodgment if the IV tubing is suddenly pulled. The hand and forearm are securely taped to the arm board for immobilization (see Fig. 18-9 C). The clear plastic wrapper of the extension tubing package is then taped over the hub of the catheter as a protective covering (see Fig. 18-9 D). Occasionally the flow rate of the infusion may be positional, especially if the catheter spans a joint or abuts a venous valve. Careful repositioning or adjustment of the hand position or catheter with strategically placed sterile gauze or slight withdrawal of the catheter may be all that is required to remedy the problem.

If blood specimens and blood cultures are needed, one can obtain these simultaneously during IV insertion and spare the child an additional needlestick. The procedure is similar to venipuncture, with the following exceptions. The T-extension tubing and attached syringe should not contain any flush solution. After appropriate preparation of the insertion site and successful placement of the catheter into the vein, the T-extension tubing and syringe are connected to the hub of the catheter, and the blood is aspirated into the syringe. After the desired quantity is obtained, the syringe is removed, and the T-extension tubing is connected to the IV infusion tubing, and the infusion pump is set at the desired rate.

If the scalp veins are used, the area surrounding the planned site of insertion should be shaved and cleansed with an iodine solution. Arteries and veins can usually be differentiated on the scalp by the fact that arteries are more tortuous than veins. In addition, the flow of blood is away from the heart in arteries and toward the heart in veins. If an artery is entered during placement of the needle and fluid is infused, blanching will occur in the area. If this happens, the catheter or needle should be removed, light pressure should be maintained for several minutes, and the procedure should be repeated at another site. A rubber band may be used as a tourniquet around the scalp *(never the neck)* to produce venous dilation. One should always ensure that the rubber band is removed after venous cannulation. When removing this rubber band, it should be carefully slipped over the catheter or butterfly needle or cut with a pair of scissors. Although cutting the rubber band with scissors is often the easiest technique, the clinician must take care to hold both ends of the cut rubber band to avoid having the infant "snapped" by one or both ends of the rubber band. Placing a piece of tape on the rubber band before placement on the scalp will facilitate lifting the rubber band away

from the scalp.

If a scalp vein butterfly infusion set is used, the wings of the butterfly are grasped between the thumb and forefinger, and the needle is introduced beneath the skin approximately 0.5 cm distal to the anticipated site of vein entrance (Fig. 18-10) . The needle is advanced slowly toward the

**Figure 18-10** Using a rubber band as a tourniquet to distend the scalp veins, the needle is introduced approximately 0.5 cm distal to the anticipated site of the vessel puncture. Gloves should be worn.

vessel until blood appears in the tubing, indicating that the vessel has been entered. The tourniquet should then be removed. The needle should be flushed with 0.5 to 2 mL of IV fluid, such as normal saline, to ensure that the needle is properly in place within the vein. If infiltration occurs, as noted by a subcutaneous (SQ) bump, the IV line should be removed and the process repeated at another site.

After the wings are secured with tape, the tubing of the butterfly set should be taped in a loop on the scalp so that it is not inadvertently pulled. A wisp of cotton may be placed under the wings of the butterfly if the infusion is positional. A small plastic medicine cup or half of a paper cup may be taped over the wings and the needle to protect the IV line (Fig. 18-11) . The catheter of the butterfly set should then be connected to the tubing from the IV system, and the IV pump should be started.

**Complications**

Complications of IV fluid therapy include infection [44] ; injection of sclerosing agents into the SQ space, with resultant necrosis and sloughing of the skin (especially in small infants) [45] ; air embolism [46] ; and administration of inappropriate volumes of fluid. The incidence of infection secondary to peripheral IV therapy may be decreased by routine periodic replacement of the needles. [47] Because the life span of an IV needle or catheter is usually fairly short (<72 hours) in the small infant, the decision concerning elective removal and replacement of the IV system is not usually a problem. Of course, it is important to pay meticulous attention to sterility during insertion and maintenance of the IV system to decrease the risk of infection.

A simulator (Medical Plastics Laboratory, Gatesville, TX) is available to demonstrate and practice the proper technique for placement of peripheral IV needles in infants. [48]

## Peripheral Venous Catheterization: Venous Cutdown

**Indications and Contraindications**

With the development of small IV catheters and butterfly needles and the rapidity and safety of intraosseous (IO)

**Figure 18-11** Protecting the IV line with a plastic medicine cup.

cannulation (see Chapter 26) , peripheral venous cutdowns are rarely used in infants. Even in experienced hands, a saphenous vein cutdown may take >10 minutes, may last no more than percutaneous catheterization, and is associated with a higher rate of infection than other routes of vascular access. [49] [50] Nonetheless, if peripheral venous, central venous, and IO cannulation cannot be performed, venous cutdowns may provide an alternative means of emergency venous access. For the purpose of illustration, the exposure and cannulation of the saphenous vein are discussed (Fig. 18-12) . The same principles apply when a cutdown is performed on an arm vein.

### Equipment and Setup

Successful venous catheterization in the small infant requires sterile instruments, an assistant, good lighting, and a selection of catheters. The use of self-retaining retractors is a personal preference. Because of temperature instability, a warming light or an overhead radiant warmer is frequently useful. Silastic catheters, which can be obtained in 2, 3, and 4 Fr sizes (Dow-Corning Company), seem to remain patent longer, and can be sterilized with the instruments to make a "cutdown tray." Standard 18- to 22-ga IV catheters (Angiocath, Deseret Medical, Inc., Sandy, Utah) are also useful.

### Technique

The clinician should begin with complete immobilization of the thigh, leg, ankle, and foot by taping them to a padded arm board, which in turn is attached to the table or bed where the procedure is being performed (see Fig. 18-12 *A*). The area around the medial malleolus is prepared with a povidone-iodine solution and draped with sterile towels. Local anesthesia is accomplished by superficial infiltration with 0.25 to 1% lidocaine in an area proximal and anterior to the superior portion of the medial malleolus. Fortunately, there are no major nerves or tendons that accompany the vein in this location (see also Chapter 22) .

A tourniquet is placed in the midleg, and a transverse skin incision is made; a small mosquito hemostat is inserted into the wound, with the concavity of the clamp upward. The tip of the hemostat is advanced to the bone in one corner of the wound, and all tissues lying against the bone and in the SQ region are "scooped up" with the hemostat (see Fig. 18-12 *B*). This will invariably lift the vein out of the wound along with surrounding tissues. A fine forceps or a mosquito hemostat is used to separate and remove all nonvenous structures, leaving only the saphenous vein tented over the hemostat (see Fig. 18-12 *C*). To avoid injury to the vein during dissection, one spreads the ends of the hemostat parallel to the direction of the vein, *never* transversely.

Two 4-0 silk sutures are passed under the vein; one silk suture is pulled distally to stabilize the vein, and the other suture is pulled proximal to the site of venipuncture. The distal suture may be tied, but if left untied, it can still be used for stabilization of the vein. Removal of the untied distal suture following vein cannulation may allow for subsequent vein recannulation following eventual catheter removal. If the distal suture is left untied, longitudinal traction on it permits hemostasis and continued exposure of the vein above

the wound. Fine scissors or a scalpel blade may be used to make an oblique or V-shaped incision in the anterior (superficial) vein wall between the sutures (see Fig. 18-12 *D*). The catheter (beveled at its tip) is then filled with saline.

The catheter is grasped with forceps and is advanced into the vein for a distance of 2 to 3 cm (see Fig. 18-12 *E* and *F*). This is usually the most difficult and time-consuming portion of the procedure. A vein dilator or forceps may be used to hold open the incision in the vein (see Fig. 18-12 *G*). Downward pull on the distal tie will give countertraction and will stabilize the vein during catheter advancement. The tourniquet is then removed. One ties the proximal suture around the vein with the catheter inside, taking care not to occlude the catheter by tying the suture too tight. If the distal suture was tied, the free ends of the suture can be tied around the catheter, providing additional stability to the catheter. If the distal suture was not tied, it is now removed. When the distal suture is left untied, the proximal suture is still tied to secure the catheter, but the ends are left long so that the suture can be pulled out of the incision and removed to allow recannulation once the infusion catheter is removed.

Continued infusion of saline through the catheter from an attached syringe will ensure patency. The catheter is oriented into either corner of the incision, and the incision is closed with interrupted 4-0 nylon sutures. The skin suture nearest the catheter is wrapped around the catheter and tied to hold the catheter in place. Bleeding can be controlled with direct pressure. Antibiotic ointment is placed over the wound, and a sterile occlusive dressing is applied. The IV tubing is connected and taped securely to the foot board to prevent inadvertent removal of the catheter (see Fig. 18-12 *H*).

One should change the dressing carefully every day, using sterile technique with reapplication of antibiotic ointment. When cared for properly, catheters can remain in place for as long as 7 to 10 days. Generally, though, a line is replaced, using another site, after 3 to 4 days. Obviously, at the first sign of infiltration or infection the catheter must be removed. Unfortunately, once the vein has been used for a cutdown, it is usually rendered useless for future venous cannulation.

**Figure 18-13** The mini-cutdown procedure using a standard IV catheter over-the-needle system is technically easier than the full cutdown and may be preferred in an emergency.

**Mini-cutdown.**

The cannulation of a small vein with a catheter or tube may be difficult and very time consuming if one is not experienced in the technique. As an alternative, the mini-cutdown procedure may be used. Once the vein is exposed through a skin incision and SQ dissection, it is cannulated directly with a standard IV catheter (Medicut, Angiocath) rather than nicked with a scalpel (Fig. 18-13) . A silk suture or hemostat may be placed under the vein to immobilize it during puncture, but with the mini-cutdown technique, the vein is not tied off after being cannulated. The catheter will not be as secure with this modification, but the technique is useful when time is critical. The vein is not destroyed with this technique. In essence, the mini-cutdown uses the percutaneous technique of cannulation, except that venipuncture is performed through a skin incision

under direct visualization (see Chapter 22) .

**Suggestions**

In an emergency situation, if a percutaneous peripheral or central venous access is not available within several minutes, a saphenous cutdown should be performed (see previous discussion). The technique requires practice and may consume 5 to 15 minutes of resuscitation time. One common error is making an improper skin incision. The incision must be made through all layers of the skin without severing the vein. SQ fat should be visible through the incision. The SQ incision should be carried to the end of the skin incision so that the clinician can take full advantage of the skin incision. A 2-cm incision is usually required, and one should not try to work through a skin incision that is too small.

One should perform dissection only with a blunt technique, spreading the hemostat *parallel to the course of the vein.* Inadvertent severance of the vein may occur during dissection, and one can best control bleeding by pulling the silk ties taut. The incision in the vessel may be a source of frustration. One must incise completely into the lumen of the vessel; a superficial nick, although it will bleed, will not allow for catheter passage. If the vein is severed completely, it will retract from view and will be difficult to find. Generally an incision should include one third of the vessel diameter. Placing the catheter into the vessel lumen is usually the most difficult part of the procedure, and it is easy to create a false lumen. Small plastic vein dilators are available to facilitate entering into the lumen. If a valve is encountered during passage, one should increase the rate of fluid administration while gently advancing the catheter.

**Complications**

In addition to the problems discussed previously, venous cutdowns can result in wound infections and phlebitis. Adjacent structures may be injured during the incision and subsequent blunt dissection. When the mini-cutdown technique without ligatures is used, extravasation of infusate may result. Light pressure on the closed wound will generally prevent continued extravasation.

## Central Venous Catheterization: Percutaneous

Percutaneous placement of central venous lines (CVLs) has become the technique of choice of many clinicians for securing central venous access in neonates and young infants (see Chapter 24) . [49] [51] [52] This technique has largely supplanted the conventional technique of venous cutdown catheterization. Both percutaneous and venous cutdown catheterization require central venous catheters, which can be purchased separately or within self-contained kits (Arrow International, Inc., Reading, PA; Gesco International, San Antonio, TX). However, CVL placement requires little additional equipment, whereas venous cutdown catheterization requires a set of sterile instruments. Percutaneous CVL placement also preserves the access veins for repeated use, whereas the venous cutdown technique has traditionally included ligation of the access vein. Finally, the percutaneous approach avoids the tissue injury associated with surgical incision and dissection. [53] It may be used for rapid venous

access in emergency situations, as well as to secure central venous access when peripheral venous access is limited (e.g., in low-birth-weight infants). [49] [54] [55]

**Indications and Contraindications**

Percutaneous central venous cannulation is indicated to secure vascular access (1) when peripheral venous access is limited or impossible, (2) for emergency drug and fluid administration during cardiac arrest and shock, (3) when hyperalimentation and IV infusions are required for days to weeks, (4) when low-birth-weight neonates and young infants require central venous access, and (5) when precise hemodynamic monitoring is needed in a critically ill or injured child. Contraindications to percutaneous placement of central venous catheters include an uncorrected coagulopathy; local infections or burns at insertion sites; malformations or deformations that may distort vascular anatomy; vascular insufficiency of an extremity; obstruction or compression of the access veins by tumor, abnormal vessels, hematoma, thrombus, abscess, or malformation; absence of access veins; or a planned future transfemoral cardiac catheterization. [55] Bacterial septicemia is a relative contraindication, and delaying placement of central venous access until cultures have been sterile for 48 hours is generally recommended.

**Equipment and Setup**

Percutaneous central venous catheterization in infants and children can be performed using any of a number of sterile over-the-needle catheters ranging in size from 22 to 16 ga

(choice depends on the age of the patient) and equipment similar to that used for percutaneous peripheral venous catheterization. If insertion of a larger indwelling catheter is desired, commercially available kits are convenient (Gesco International, Inc., San Antonio, TX; Arrow International, Inc., Reading, PA; Cook, Inc., Bloomington, IN). The catheters are typically made of a silicone elastomer, polyvinyl chloride, or polyethylene. Catheter length is variable, and 1- to 3-lumen catheters are available. It is important to remember that the flow rate of the catheter is directly proportional to the gauge of the catheter and indirectly related to the length. Rapid volume replacement, as in the case of severe dehydration or acute blood loss from trauma, is best achieved by inserting a short, large-bore catheter for the initial resuscitation and stabilization. If the patient requires hemodynamic monitoring or multiple medication infusions, the catheter can be replaced later with a larger indwelling catheter using the Seldinger technique.

Other necessary equipment includes sterile forceps and scissors, povidone-iodine solution, gauze pads, sterile drapes, gowns, gloves, caps and masks, syringes (3 mL, 5 mL, and 10 mL), Tegaderm (Medical Products, Inc., St. Paul, MN), Op-Site (Smith and Nephew Medical, Massilon, OH), or other sterile transparent skin coverings, Luer-Lok three-way stopcocks, 0.25 to 1.0% lidocaine, flush solution (1 to 2 U heparin per mL normal saline or $D_5W$), and IV tubing with a T-connector extension. Depending on the access vein to be used, restraint of the extremity, pelvis, or head may require a padded support, an assistant, or both.

**Techniques**

Percutaneous placement of central venous catheters can be accomplished using 2 methods that differ only in the use of a guide wire. The guide wire (Seldinger) technique is preferred when catheters are inserted into the femoral vein or subclavian vein. When using the basilic or cephalic vein of the forearm and antecubital space, axillary vein, or superficial temporal or posterior auricular scalp vein, we prefer to insert the catheter through an introducer needle. Some investigators may use the saphenous vein as an access vein. Details of the pediatric femoral, external and internal jugular, and subclavian and antecubital approaches follow.

**Femoral catheterization.**

Previously, central venous catheters were most commonly inserted by cutdown on the external or internal jugular vein or high saphenous vein or by percutaneous cannulation of the subclavian or internal jugular vein. [56]

The safety and efficacy of percutaneous femoral venous catheterization have been demonstrated. [21] [52] [57] Femoral venous catheterization is the central venous access route most commonly used in infants and children in emergency situations. Advantages of this technique are several. The femoral anatomy is easily learned, and the arterial pulse provides a landmark for catheter insertion. In case of inadvertent arterial puncture or venous laceration, hemostasis can be achieved by application of direct pressure. Also, femoral catheterization is less likely to interfere with emergency procedures in the region of the head, neck, and chest during medical or trauma resuscitations. [21] In addition, the specific risks associated with subclavian and internal jugular vein catheterization (pneumothorax and carotid or subclavian artery puncture) are avoided. Risks of the procedure include thrombosis and infection; these can occur with any type of venous catheters.

**Technique.**

The child must be adequately restrained to permit exposure of the inguinal region; sedation may be useful. It may be helpful to use an ultrasonic Doppler flow detector to locate the femoral artery and then place a heavy ink mark on the abdomen in the line of the femoral artery. This may be useful if edema makes palpation of the artery difficult or if the artery is difficult to locate when wearing gloves. Note that during CPR, palpable pulsations or Doppler tones in the femoral vein may be detected. [58] Hence, if the vein is not found medial to the pulsations, catheterization of the pulsating vessel during cardiopulmonary resuscitation (CPR) may be considered as a last resort when other options for vascular access or drug delivery are unavailable. Both groins are generally prepared with povidone-iodine in the event that the initial attempt is unsuccessful.

The introducer needle supplied with the kit can be used with or without a syringe to enter the femoral vein. The femoral artery is palpated with 1 finger, and the needle is placed in the skin just medial to the artery. One enters the skin at a 30° to 45° angle

approximately 1 cm below the inguinal ligament. The general course of the needle is in a line directed toward the umbilicus. When blood return is noted, the wire is gently passed through the needle into the proximal vein. An alternative method that may be useful when placing the 4 Fr double-lumen Arrow catheter is to remove the tubing from a 21-ga butterfly needle (Abbott Hospitals, Inc., North Chicago, Ill) and use the needle to enter the vein (Fig. 18-14 *A*). The butterfly needle is very easy to hold in a stable position and is also shorter than the needles supplied with the assembled kits. When blood return is obtained, the wire is passed through the butterfly needle into the proximal vein.

A small incision (1 to 2 mm) is then made along the wire to allow passage of the vein dilator (Fig. 18-14 *B*). The dilator is removed; the catheter, which has been flushed with saline, is advanced over the wire into the vein; and the wire is then removed (Fig. 18-14 *C*). [59] Occasionally it is useful to rotate and advance the catheter simultaneously as it enters the vein. Blood return is noted from the catheter ports, which are then flushed with a heparinized saline solution (10 U/mL). The catheter is subsequently secured with silk or nylon sutures (Fig. 18-14 *D*). A sterile transparent skin covering placed over the exit site may be used as an impermeable dressing.

This technique is useful in children as small as 1000 gm. When one is placing femoral venous catheters in children <1500 gm, a smaller single-lumen catheter (3 Fr or 24 ga) should be used, because a larger catheter may occlude blood flow in the femoral vein. [4]

**External jugular venous catheterization.**

The external jugular vein is superficial and easily visible. This site should be selected for catheterization only after catheterization of other, more peripheral sites has been unsuccessful. Also, the external jugular vein should not be chosen as a primary catheterization site during resuscitative efforts, since manipulation of the head and neck may compromise management of the airway. In young infants, use of the Seldinger technique is difficult due to the short length of the infant's neck, as well as the low success rate of central venous catheter placement resulting from the acute angle of entry of the external jugular vein into the subclavian vein. [60]

**Technique.**

The external jugular vein lies in a line from the angle of the jaw to the middle of the clavicle and is

**Figure 18-14** Technique for inserting a femoral venous catheter. *A,* A 21-ga butterfly catheter is used to enter the femoral vein, and the guide wire is passed through the butterfly needle into the proximal vein. Note that the tubing has been removed from a standard butterfly set. *B,* A small incision is made alongside the wire, and the dilator is advanced over the wire and into the vein. *C,* The catheter is advanced over the wire and into the vein. *D,* The wire is removed and the catheter secured. Note that

usually visible on the surface of the skin. The vein is more prominent when the infant is crying. An assistant is needed to restrain the infant in a supine position with the head and neck extended over the edge of the bed. Alternatively, a towel roll or pillow placed under the shoulders can be used. The head is turned approximately 40° to 70° from the midline (see Fig. 18-4) . The skin surrounding the area to be punctured is cleansed with alcohol. The area is covered with a sterile drape, and 1% lidocaine may then be infiltrated into the skin. A finger may be placed just above the clavicle to distend the jugular vein.

Using an 18- to 22-ga catheter with a syringe, the catheter is aligned parallel to the vein, and the skin is punctured approximately one half to two thirds of the distance from the angle of the jaw to the clavicle. The catheter is advanced slowly until the jugular vein is entered. The syringe is connected to the catheter at all times to maintain a constant negative pressure and avoid an air embolism. After the appropriate amount of blood is obtained, the catheter is advanced and secured in place. If the Seldinger technique is used, proceed as described for femoral catheterization. The catheter should be passed far enough to reach the superior vena cava-right atrium junction. The catheter is checked for blood return, and the line is secured with sutures and a sterile occlusive dressing applied. [61] A chest radiograph is warranted to assess the proper location of the catheter, as well as to rule out the possibility of an iatrogenically induced pneumothorax.

**Internal jugular venous catheterization.**

The internal jugular veins lie within the carotid sheath containing the carotid artery and vagus nerve. The lower part of the veins lies within the triangle formed by the sternal and clavicular heads of the sternocleidomastoid muscle and becomes more lateral and anterior to the artery as the veins join the subclavian

vein. The right internal jugular vein is preferred over the left, since the internal jugular and innominate vein and the superior vena cava form a nearly straight line into the right atrium. This lessens the chance for pneumothorax or injury to the thoracic duct (see Chapter 24) . Like the external jugular, this site should be chosen for catheterization only after catheterization of other, more peripheral sites has been unsuccessful.

**Technique.**

Three approaches (the anterior, medial or central, and posterior approaches as discussed in Chapter 24) to internal jugular catheterization are possible. The medial or central approach is recommended in pediatric patients and will be described here. The child is positioned in the same fashion as that described for external jugular venous catheterization. The medial or central approach uses the apex of the angle formed by the sternal and clavicular heads of the sternocleidomastoid muscle as the entry site. The skin surrounding the area to be punctured is cleansed with alcohol. The area is covered with a sterile drape, and 1% lidocaine may then be infiltrated into the skin.

Using an 18- to 22-ga needle with a syringe, the needle is introduced at the apex of the triangle at an angle of 30° downward relative to the coronal plane and directed caudally toward the ipsilateral nipple (Fig. 18-15) (Figure Not Available) . The needle is advanced slowly until the jugular vein is entered. The syringe is connected to the needle at all times to maintain a constant negative pressure and avoid an air embolism. After blood flow is obtained, the syringe is removed, and a finger is placed over the hub of the needle. A guide wire is then inserted during a positive-pressure breath or exhalation, the needle is removed, and a catheter is introduced using the Seldinger technique (see Chapter 21) . The catheter should be passed far enough to reach the superior vena cava-right atrium junction. The catheter is checked for blood return, the line is secured with sutures, and a sterile occlusive dressing is applied. [61] A chest radiograph is warranted to assess the proper location of the catheter, as well as to rule

**Figure 18-15** (Figure Not Available) Technique for internal jugular venous catheterization (medial or central approach). The needle is inserted at the apex of the triangle formed by the sternal and clavicular heads of the sternocleidomastoid muscle. The needle is angled 30° downward relative to the coronal plane and directed toward the ipsilateral nipple. *(From Textbook of Pediatric Advanced Life Support, 1994. Reproduced with permission. Copyright American Heart Association.)*

out the possibility of an iatrogenically induced pneumothorax.

**Subclavian venous catheterization.**

The subclavian vein is a popular route of central venous access in the adult patient but is used far less frequently in children. The technique is more difficult in the child because of the vessels' smaller size, as well as their more cephalad location under the clavicles. An infraclavicular approach to the subclavian vein has been used, but due to the high risks of pneumothorax and hemothorax, especially when performed during emergencies, this approach should be considered only if other peripheral or central venous access sites are unobtainable. [62] [63] The younger the patient, the higher the risks of these complications. Also, subclavian venous access may interfere with resuscitative efforts, and except in very small infants and obtunded children, heavy sedation and adequate restraint are often required.

**Technique.**

The technique for subclavian venous catheterization differs from that for the adult in that the approach to the vein is more lateral in children. The equipment needed is the same as that used for femoral catheterization. The patient is placed in the Trendelenburg position with the head turned away from the side to be punctured and a towel roll placed under the shoulders (Fig. 18-16) (Figure Not Available) . The right side is preferred, since the dome of the lung is more cephalad on the left side. The needle insertion site is at the distal one third of the clavicle in the depression created between the deltoid and pectoralis major muscles. If the patient is awake, sedation should be administered. The skin is prepped with antiseptic solution, the area covered with a sterile drape, and the skin infiltrated with 1% lidocaine.

An entry point is created at this location using a scalpel blade. The catheter needle is

introduced and advanced slowly while negative pressure is applied with the attached syringe. The syringe and catheter needle should be parallel to the frontal plane and directed medially and slightly cephalad, beneath the clavicle toward the posterior aspect of the sternal end of the clavicle (i.e., with the lower end of the fingertip placed in the sternal notch). [64] The needle is advanced until blood return is obtained. The catheter can be advanced a few more millimeters further to ensure its position in the vein. Catheter functioning can be assessed by the ability to withdraw blood and flush saline without difficulty. At this point, a large-bore catheter can be inserted using the Seldinger technique as previously described for femoral catheterization. Auscultation of bilateral breath sounds should be performed and a chest radiograph obtained to confirm the proper positioning of the catheter in the superior vena cava, as well as to rule out procedural complications such as pneumothorax or hemothorax. The catheter is then secured in place with sutures, and a sterile, occlusive dressing is applied.

**Antecubital access.**

Percutaneous insertion of central catheters by way of peripheral antecubital veins is used most frequently to obtain central venous access in patients with very small caliber vessels (e.g., low-birth-weight neonates and very young infants). These peripherally inserted central catheter (PICC) lines are small Silastic catheters ranging in size from 23 to 16 ga. [55] [65] [66] The catheter is inserted through a peripheral vein through a 19-ga butterfly needle or 20-ga angiocatheter. The most common sites include scalp, neck, and arm veins. The catheter is then threaded centrally. PICC lines offer several advantages over conventional peripheral

**Figure 18-16** (Figure Not Available) Technique for subclavian venous catheterization. The needle is inserted at the distal one third of the clavicle in the depression created between the deltoid and pectoralis major muscles. The needle should be parallel to the frontal plane and directed medially and slightly cephalad toward a fingertip placed in the sternal notch. The patient is shown in a 30° Trendelenburg position. *(From Textbook of Pediatric Advanced Life Support, 1994. Reproduced with permission. Copyright American Heart Association.)*

IV catheters and percutaneous central venous catheters. They can remain in place for up to 3 months, sparing vein wasting from multiple reinsertions with peripheral venous catheterizations; the long-arm catheter is simpler to insert than central venous catheters and poses no risk of producing a pneumothorax or hemothorax on insertion. [67]

**Technique.**

The vessel to be cannulated is initially stabilized using a support board or the help of an assistant. The remainder of the procedure requires sterile technique. Povidone-iodine is used to cleanse the skin overlying the vessel to be cannulated, and 0.25% to 1% lidocaine is infiltrated at the skin site to be punctured. This skin site is punctured with an 18-ga needle to ease insertion of the introducer through the skin. The catheter to be inserted is chosen based on the size of the access vessel.

Typically a 23-ga silicone elastomer catheter with other needed accessories is used, as in a kit prepared by Gesco International, Inc. (San Antonio, Texas). Advantages of this

catheter include (1) a double-wing silicone adapter, which precludes the need to make homemade blunt-end adapters to fit small cannulas and simplifies the taping procedure, and (2) a breakaway introducer needle that can be peeled off the catheter, thereby precluding the need for sliding the introducer off the catheter and placing an adapter. Because the length of this catheter (33.5 cm) is longer than needed in low-birth-weight neonates and young infants, the distance from the insertion site to the superior vena cava-right atrium junction is estimated (i.e., by measuring the distance between insertion site and the right nipple [Fig. 18-17 A]), and the catheter is cut 1 to 3 cm longer than the estimated distance to compensate for variability between the estimated and actual needed length of the catheter. The end of this catheter is then connected to a Luer-Lok stopcock and syringe and filled with flush solution; the catheter is then ready for use.

**Figure 18-17** Techniques for insertion of central catheters from peripheral veins. *A,* A tape measure is used to determine the catheter length. *B,* Placement of the catheter through the specialized breakaway butterfly-type introducer needle.

The 20-ga breakaway introducer needle (Gesco International, Inc.) is also filled with flush solution and then directed slowly through the insertion site and into the access vein. When blood return occurs, the catheter is picked up approximately 1 cm from its tip and guided into the introducer needle (Fig. 18-17 B). The catheter is advanced in 1-cm increments until the previously estimated distance is reached (i.e., the catheter tip is at the superior vena cava--right atrium junction). The breakaway introducer needle is then withdrawn several centimeters from the insertion site before peeling the introducer off the catheter to avoid inadvertent catheter laceration. If accidental laceration occurs, blunt-end adapters should be readily available.

Immediately after catheter placement and withdrawal of the introducer needle, the clinician will be able to manipulate the position of the catheter. Once clotting occurs around the catheter at the insertion site, however, the catheter becomes difficult to manipulate. In addition, after the sterile field is discontinued, the catheter should never be advanced. The function of the catheter is checked by withdrawing blood, by noting the presence of residual air bubbles within the catheter, or both. After the clinician is assured that no air bubbles remain in the line, the catheter is flushed. This catheter should be "easy" to flush; if it is not, the clinician should reposition the catheter and recheck its function. If the catheter remains difficult to flush, it should be considered clotted and should be removed. Alternatively, position may be confirmed by chest radiograph and fibrinolytic therapy used, if considered appropriate.

A transparent skin covering is placed and is removed only when the catheter is removed; it is not routinely removed as are coverings of some surgically placed central venous catheters. Stabilization sutures are not routinely placed during this procedure. Occasionally a small amount of bleeding occurs at the insertion site; this generally stops spontaneously or with gentle pressure. With the three-way stopcock in place, central venous pressure measurement and infusion of medications, IV fluids, and hyperalimentation solutions can be performed.

**Complications**

The incidence of complications from central venous catheterization ranges from 10% to 50%. [65] [68] [69] [70] Infection and thrombosis are the major risks associated with these catheters. [53] [71] [72] Other complications include accidental displacement, phlebitis, hemorrhage, hematoma, dysrhythmia, air embolus, vascular obstruction or perforation, right atrial perforation, and localized edema. Blood sampling from indwelling central venous lines must be performed with caution, because the risk of contamination increases each time the system is opened. Morbidity from complications can be minimized by removing catheters as soon as they are no longer needed. Occasionally infections associated with central venous lines can be successfully treated with antibiotics alone. In the majority of catheter-associated infections, however, the catheters must be removed to resolve the infection. Colonization of the catheter tip is related to in situ time, younger age, and inotropic support. [73] Central venous catheters are generally free of colonization if used for no more than 3 days in infants under 1 year and no more than 6 days in older children. Right atrial and major vessel thrombus formation can be monitored using echocardiography; treatment with fibrinolytic agents is occasionally successful in restoring catheter patency. [74] These types of catheters have been used for up to 80 days, with a mean duration as long as 34 days, in very-low-birth-weight neonates. [53] [55] Most clinicians find that the majority of these catheters are discontinued because they are no longer needed. [49]

**Emergency Vascular Access**

The first steps in managing pediatric resuscitations are to establish an adequate airway, ensure adequate ventilation, and enhance blood circulation. Maintaining or reestablishing adequate circulation often requires prompt access to the intravascular space for administration of fluids and/or medications. The difficulty of obtaining venous access during pediatric resuscitations can be considerable. [75] [76] In the review by Rossetti and colleagues, IV access required 10 or more minutes in 24% of the cases. Only 6% of cases had no IV line established before attempts at resuscitation were halted. The average time required for a cutdown was 24 minutes. Children who were successfully resuscitated had vascular access achieved significantly sooner than those who were not resuscitated. Emergency IV access was most prolonged in children <2 years of age. [75] This last finding is important, since the majority of cardiopulmonary arrests in children occur in this younger age group.

If no IV line is available, appropriate drugs can be given via the endotracheal tube (see Chapter 27) while attempts at venous access are initiated. *The initial attempts at venous access should consist of peripheral placement by percutaneous venipuncture*; however, if such placement is unsuccessful within the first 1 to 2 minutes, an IO line should be started in children <6 years of age, and percutaneous central venous cannulation or saphenous venous cutdown should be performed in children older than 6 years of age. [64] The femoral vein is often the central vein of choice in emergencies, because its consistent anatomic location and large size make it the safest and easiest central vein to catheterize. The femoral vein also can be accessed with minimal interference to resuscitative efforts. However, pulsations may be present in the vein during CPR, thus

making the distinction between femoral vein and femoral artery challenging. [58]

An IV protocol for resuscitations helps eliminate the common mistake of persisting with futile attempts at peripheral sites and wasting precious time (Fig. 18-18) (Figure Not Available) . [54] If experienced personnel are available, a simultaneous cutdown of the greater saphenous vein at the ankle or the groin can be performed. One advantage of a cutdown at the groin level is that the saphenous vein can be cannulated and a double-lumen catheter advanced into the femoral vein for rapid fluid infusions through one port and medication administration through the other port. Depending on the experience of the physicians involved in the resuscitation effort, a subclavian or internal jugular catheter insertion may be attempted; however, these are not easy procedures, and attempts may interfere with chest compressions and securing of the airway. One group reported a 92% success rate with percutaneous subclavian vein catheterization in critically ill infants using a Seldinger technique. [63]

## Umbilical Vein Catheterization

### Indications and Contraindications

The major indication for umbilical vein catheterization is access to the vascular system for emergency resuscitation

**Figure 18-18** *(Figure Not Available)* Priorities for emergency vascular access in infants and children. *(From Textbook of Pediatric Advanced Life Support, 1994. Reproduced with permission. Copyright American Heart Association.)*

and stabilization of the newborn. The umbilical vein may also be used for exchange transfusions and short-term central venous access in newborns. The umbilical vein may be cannulated up to the age of 5 to 7 days, but after 1 week of life, the technique is not generally used. The procedure is technically easier than umbilical artery cannulation. *Umbilical vein catheterization is not an acceptable alternative after the infant leaves the hospital*; for example, the procedure would not be used when a 2- to 4-week-old infant presents to the emergency department.

### Equipment and Setup

The supplies and equipment for catheterization are listed in Table 18-5 . Although unlikely to be needed for most emergency

**TABLE 18-5** -- Umbilical Vein and Artery Catheterization Equipment

Infusion solution (usually D5-10 W with electrolytes. Some physicians also add 1 unit heparin per milliliter of fluid to "prevent" clotting in the catheter) Fluid chamber, IV tubing, infusion pump, filter (0.22 mum), short length of IV tubing, three-way stopcock
Umbilical artery catheter (3.5 to 5 Fr)
3-0 silk suture on a curved needle
Curved iris forceps without teeth
Small clamps, forceps, scissors, needle holder
Sterile drapes
Light source
10 mL of heparinized solution for flush (1 to 2 units heparin per milliliter of fluid)
Surgical cap, mask, gown, and gloves

department resuscitations, double-lumen umbilical venous catheters (#C-DUCO 5.0-30, Cook Critical Care, Bloomington, IN) are available that minimize the need for multiple venous catheters in the infant. [77] The infant is placed beneath a radiant warmer, and the extremities are restrained. Oxygen is administered as needed, and the audible beep on the cardiac monitor is turned on. The operator should wear a surgical cap and mask and a sterile gown and gloves.

**Technique**

The infant's extremities are restrained using gauze wrapped around the ankles and wrist and pinned to the bed. An assistant is used to hold the umbilical stump up. The cord is scrubbed with a bactericidal solution. Pooling of liquid at the infant's side should be avoided, because this may be associated with blistering of the skin under a radiant warmer. The umbilical area is draped in a sterile fashion, with the infant's head left exposed for observation.

To provide hemostasis and to anchor the line after placement, a pursestring suture is placed at the junction of the skin and the cord (Fig. 18-19) . Alternatively, a constricting loop of umbilical tape in the same position may be used. The cord is cut about 1 cm from the skin, and the vessels are identified. The vein is usually located at 12 o'clock and has a thin wall and large lumen and may continue to bleed after cutting, whereas the two arteries have thicker walls and smaller lumina, and constriction prevents bleeding after being cut. Occasionally a persistent urachus may be mistaken for the umbilical vein, but the return of urine should identify the mistake.

The catheter (3.5 Fr [preterm infants] to 5.0 Fr [term infants]), which has been flushed with heparinized saline

**Figure 18-19** When placing an umbilical vein catheter, a pursestring suture or umbilical tape is passed around the base of the cord to provide hemostasis and to anchor the line.

and attached to a three-way stopcock, is placed in the lumen of the umbilical vein and advanced gently. The catheter is advanced only 1 to 2 cm beyond the point at which good blood return is obtained. This is usually only 4 to 5 cm in a term-sized infant. If the catheter is pushed farther than this, it will do one of two things: It may enter the ductus venosus and then move into the inferior vena cava, or it may enter a branch of the portal vein within the liver (evidenced by resistance at 5 to 10 cm).

The *inferior vena caval site* may be a desirable location in some newborn infants in whom peripheral vascular access is limited and for whom central venous access is desired for central venous pressure monitoring or infusion of medications, high concentrations of glucose (>10%), IV fluids, and hyperalimentation solutions. The catheter must be inserted approximately 10 to 12 cm in a term-sized infant to reach the inferior vena cava. The *portal vein site* can be verified by radiographs that document the placement of the catheter. Note that an umbilical venous catheter will proceed directly cephalad (without making a downward loop) until it passes through the ductus venosus (Fig. 18-20) (Figure Not Available) . Of course, in a resuscitation, radiographic documentation may not be possible. Therefore, it is generally recommended that the catheter be inserted approximately 4 to 5 cm to minimize the risk of injecting sclerosing solutions into the liver.

Air embolism may occur at the time of catheter removal if the infant generates sufficient negative intrathoracic pressure (as during crying) to cause air to be drawn into the patent umbilical vein. Therefore, caution must be used during catheter removal to ensure that the vein is promptly occluded (by tightening a pursestring suture or applying pressure on or just cephalad to the umbilicus).

### Complications

Complications of umbilical venous catheters include hemorrhage, infection, injection of sclerosing substances into the liver (resulting in hepatic necrosis), air embolism, and vessel perforation. It is most important that one follow careful technique in insertion and maintenance of catheters to minimize such complications.

## Umbilical Artery Catheterization

### Indications and Contraindications

Umbilical artery catheterization is a useful procedure in the care of newborn infants who require frequent monitoring of arterial blood gases and arterial blood pressure, fluid and medication administration, and exchange transfusions. It is imperative for the clinician to remain aware of potential complications. [78] [79] [80] One of the two umbilical arteries may be cannulated for resuscitation purposes, but an umbilical vein is generally technically easier to cannulate and may be preferred in an emergency.

### Equipment and Setup

The equipment required for umbilical artery catheterization is identical to that used for umbilical venous catheterization. Additional equipment needed for continuous arterial pressure monitoring and infusion should be readily available.

**Technique**

The technique of umbilical artery catheterization is similar to that described for umbilical vein catheterization in the preceding section. After the umbilical arteries have been located, the cord is grasped with a curved hemostat near the selected artery (Fig. 18-21) . This maneuver is important, because it provides clear visualization and stabilization of the vessel. Using the curved iris forceps without teeth, one gently dilates the artery. Umbilical artery spasm may make the procedure difficult. A 3.5 to 5 Fr catheter is attached to a three-way stopcock and is flushed with sterile heparinized solution. The catheter may then be introduced into the dilated artery. A 3.5 to 4 Fr catheter is recommended for infants weighing <2 kg and a 5 Fr catheter for those weighing 2 kg.

When the catheter is being inserted, tension should be placed cephalad on the cord, and the catheter should be advanced with slow, constant pressure toward the feet (Fig. 18-22) . Resistance is occasionally felt at 1 to 2 cm and should be overcome by gentle, sustained pressure. If the

**Figure 18-20** (Figure Not Available) An umbilical vein catheter is directed toward the head and remains anterior until it passes through the ductus venosus into the inferior vena cava. *(From Ludwig S, Fleisher GR: Textbook of Pediatric Emergency Medicine. Baltimore, Williams & Wilkins, 1994.)*

**Figure 18-21** The umbilical cord is grasped with a curved hemostat near the selected artery. The umbilical artery is then dilated with curved iris forceps.

catheter passes 4 to 5 cm and meets resistance, this generally indicates that a "false passage" through the vessel wall has occurred. Occasionally one may bypass the perforation by attempting catheterization with the larger 5 Fr catheter.

The optimal position of the catheter tip in the descending aorta remains the subject of much debate. [81] [82] [83] If a low (L3 to L4) position is desired, the catheter may be advanced 7 to 8 cm in a 1-kg premature infant or 12 to 13 cm in a full-term infant. Graphs are available to estimate the proper length of insertion for a high or low catheter location. [84] [85] Once sterile technique is broken, the line may not be advanced. It is therefore preferable to position the catheter too high and to withdraw as necessary according to the location on a radiograph. After it has been positioned appropriately, the catheter should be tied with the previously placed suture (Fig. 18-23) and taped to the abdominal wall. A radiograph should be obtained and the catheter repositioned, if necessary, with the tip at the lower border of the L3 vertebra. Some clinicians prefer to place the catheter high (T6 to T9 vertebrae). There are no unequivocal data to support either preference. [86] [87]

Radiographs of an arterial catheter (Fig. 18-24) will show the catheter proceeding from the umbilicus down toward the pelvis, making an acute turn into the internal iliac artery, continuing toward the head into the bifurcation of the aorta, and then moving up the aorta slightly to the left of the vertebral column. [88]

Most unsuccessful umbilical artery catheterization attempts fail because the catheter perforates the arterial wall approximately 1 cm below the umbilical stump, where the umbilical artery begins curving toward the feet. [89] In this instance, the catheter is advanced in the extraluminal space, and resistance is met at 4 to 6 cm. The following maneuvers make it possible to avoid perforating the umbilical arterial wall in most cases:

1. The catheter should be advanced slowly. When slight resistance is met at approximately 1 cm, the catheter should be advanced very gently with steady pressure. It should never be forced, because it will likely perforate the wall. A catheter or feeding tube with a molded tip should be used. A catheter tip that has been cut with scissors is more difficult to insert and advance.
2. Because the artery curves toward the feet, the umbilical stump should be held with a curved clamp and should be pulled toward the head so that the catheter is inserted toward the feet in as straight a direction as possible.

The use of a placenta or a commercially available simulator

**Figure 18-22** The catheter is introduced into the dilated artery and advanced toward the feet. The suture placed around the base of the cord is tied to the catheter.

**Figure 18-23** The tape is pleated above and below the catheter.

(Medical Plastics Laboratory, Gatesville, Tex) makes it relatively easy to demonstrate and practice the proper technique of umbilical artery catheterization. [90] [91]

**Complications**

If the catheter becomes plugged or fails to function properly or if there is blanching or discoloration of the buttocks, the heels, or the toes, then the catheter should be removed at once. Umbilical arteries are most easily cannulated in the first few hours of life but may provide a viable vascular route as late as 5 to 7 days of age.

Complications include hemorrhage, [92] infection, [93] [94] [95] thromboembolic phenomena (especially to the kidneys, the gastrointestinal tract, and the lower extremities), [96] [97] [98] aortic thrombosis, [99] aortic aneurysm, [100] vasospasm, air embolism, vessel perforation, peritoneal perforation, hypertension, [101] [102] and possible effects of plasticizers. [103]

## Percutaneous Arterial Catheterization

### Indications and Contraindications

Despite the growing use of noninvasive devices for monitoring transcutaneous oxygen and carbon dioxide, percutaneous

**Figure 18-24** The umbilical artery catheter makes a loop downward before heading cephalad (schematic drawing of a radiograph interpretation).

peripheral arterial catheterization is indicated when there is a need for frequent blood gas sampling, continuous arterial blood pressure monitoring, or both. Arteries used for peripheral catheters in infants include the radial, [104] ulnar, [105] femoral, [106] temporal, [107] [108] and posterior tibial arteries. [109]

Percutaneous radial artery catheterization has become widely accepted and has been shown to be a safe method in infants and children. [110] The catheter allows for preductal blood gas determinations if placed in the right radial artery. Only the procedure for radial artery catheterization is described here, but catheterization of other vessels is similar.

The following are contraindications to peripheral arterial catheterization:

> Situations in which adequate peripheral arterial samples can be obtained by percutaneous punctures

> Situations in which circulation of the extremity to be catheterized is compromised

> Situations in which occlusion of the vessel to be catheterized results in compromised perfusion of that extremity

> The presence of an ongoing bleeding diathesis

> The presence of localized infection or inflammation overlying the artery to be cannulated

> Situations in which intensive monitoring of line function is not available.

### Equipment and Setup

The equipment needed for arterial catheterization is essentially the same as that required for percutaneous peripheral venous catheterization (see Table 18-4) . The catheters used are usually 22- or 24-ga over-the-needle catheters, a T-piece connector, and a stopcock. One should connect the T piece and the stopcock and then fill them with normal saline solution. An infusion pump with heparinized saline (1 to 5 U/mL)

should be readied. [111]

**Technique**

The procedure should be performed with good lighting and an adequate work area, with the infant's heart and respiratory rates monitored closely. The radial artery may be palpated proximally to the transverse wrist crease on the palmar surface of the wrist, medial to the styloid process of the radius. The artery is then compressed, and the hand and

fingers are observed for color change. If blanching or cyanosis is noted (indicating poor collateral circulation), catheterization is not performed.

A fiberoptic transilluminator may be used to localize the artery. With the overhead lights off, the transilluminator head (with a rubber shield or filter to prevent overheating the skin) is positioned beneath the wrist, and the artery is visualized as a dark, pulsatile shadow. [112] [113]

The infant or child's hand and lower forearm are secured to an arm board with the wrist dorsiflexed 45° to 60° with the aid of a roll of gauze placed underneath. Care must be taken to leave the fingers exposed to assess the peripheral circulation. The radial artery is palpated at the point of maximal impulse and can be marked with a gentle indentation by one's fingernail. The area over the radial artery is prepared with a povidone-iodine solution and washed with alcohol. A local anesthetic such as 1% lidocaine without epinephrine may be used at the planned insertion site. The catheter with stylet is inserted through the skin just proximal to the transverse wrist crease at a 10° to 20° angle (Fig. 18-25) . The catheter with needle is advanced slowly until blood appears in the catheter hub, signifying puncture of the anterior arterial wall. The catheter is slowly advanced further until blood appears in the needle and then the needle angle is carefully lowered to approximately 10°. The catheter is slowly advanced over the needle into the lumen of the artery, and the needle is removed. The stopcock and T-piece connector are attached to the catheter hub. The stopcock is opened to the syringe to confirm pulsatile blood return. It is then flushed with 0.5 mL heparinized flush solution very gently to clear the catheter while the fingers and the hand are observed for evidence of blanching or cyanosis.

The puncture site is then covered with antibiotic ointment, and the catheter is fixed to the skin by a thin piece of tape placed adhesive side uppermost under the catheter hub and crossed over the catheter in a V shape. A second piece of tape is passed around and over the catheter hub and is fixed to the wrist (Fig. 18-26) . A small piece of tape is used to attach the T-piece connector to the wrist area or to the splint. The fingers should be easily visible.

Only heparinized normal or half-normal saline is used for infusion. Some clinicians prefer to add 1 to 5 U of

**Figure 18-25** The catheter assembly is introduced into the radial artery through skin at a 10° to 20° angle.

**Figure 18-26** One technique of taping the arterial catheter. The arm board should be well padded and secured.

heparin per milliliter of infusion solution infused at a rate of 1 to 2 mL/hour. Medications, blood or blood products, amino acid solutions, IV fat solutions, and hypertonic solutions are not infused through the catheter.

The catheter must be removed when there is evidence of blanching or cyanosis or when it is impossible to withdraw blood from the catheter or difficult to flush the catheter.

**Complications**

Complications, which have been reported with every type of arterial catheter, include hemorrhage, thrombosis, spasm, infection, scars, air embolism, retrograde blood flow, transient elevation in blood pressure with rapid (<1 second) infusion, and nerve damage. Thrombosis or spasm may result in blanching or cyanosis of the extremity or skin. [114] [115] [116] There is potential for loss of digits, an entire extremity, or large areas of skin, as well as cerebral infarction with temporal artery catheters. [117] [118] [119] Malposition of the catheter such that infusion of a glucose solution flows into the celiac artery may also precipitate hypoglycemia on discontinuation of the glucose infusion. Saladino and colleagues note that complications from emergency department-placed arterial lines are uncommon and generally minor. [120]

**Figure 18-27** Anatomy of the posterior tibial artery and surrounding structures.

**Figure 18-28** Posterior tibial artery cutdown technique. With the foot prepared and immobilized, a 5- to 7-mm incision is made in the skin posterior to and at the midline of the medial malleolus. Curved forceps and a silk suture are inserted beneath the posterior tibial artery, which courses just posterior to the medial malleolus.

## Arterial Cutdown Catheterization

### Indications and Contraindications

Arterial catheterization by cutdown on the posterior tibial artery, radial artery, and temporal artery may be indicated when the need exists for frequent monitoring of arterial blood gases or blood pressure and when percutaneous access is not possible. [109] [121] Arterial cutdowns are contraindicated when (1) adequate peripheral blood gas samples can be obtained by percutaneous punctures or catheterization, (2) circulation of the extremity to be catheterized is compromised, or (3) occlusion of the vessel to be catheterized results in compromised perfusion of that extremity.

**Equipment and Setup**

Successful arterial cutdown catheterization in the small infant requires sterile instruments, an assistant, good lighting, and a selection of catheters. Previous clinical experience is helpful. The use of self-retaining retractors is a personal preference. Because of temperature instability, a warming light or an overhead radiant warmer is frequently useful. The equipment required for performing an arterial cutdown catheterization can be found on a cutdown tray, available in most emergency departments. Also needed are a 22- or 24-ga over-the-needle catheter, T-extension connector tubing, stopcock, a 5- or 10-mL syringe filled with flush solution (normal saline with 1 to 5 U heparin/mL) and silk suture ties.

**Technique**

The anatomy and technique for posterior tibial arterial cutdown are described in detail (Fig. 18-27) . The same technique is applicable for the radial artery. The clinician stabilizes the foot in a neutral position by taping the externally rotated lower leg to a splint. The *posterior* tibial artery is then localized by Doppler ultrasound just posterior to the medial malleolus. The operator prepares for the procedure by scrubbing and donning a gown and gloves; the foot is prepared with a povidone-iodine solution.

Following SQ injection of 1% lidocaine, a 5- to 7-mm transverse incision is made in the skin over the artery posterior to and at the midlevel of the medial malleolus (Fig. 18-28) . Using blunt dissection in a vertical direction (parallel to the vessels), the tissue is separated with a small, curved forceps, and the artery is identified. The artery courses with the vein just anterior and superficial to the nerve and is usually pulsatile. One isolates the artery by sliding a small, curved forceps beneath it and gently elevating the vessel (see Fig. 18-28) . Excessive manipulation of the artery can cause spasm; if this occurs, a few drops of 1% lidocaine applied locally may result in dilation. A silk tie (without a needle) is then placed beneath the artery to stabilize it during cannulation.

**Figure 18-29** Technique of inserting the arterial catheter. A silk tie is used *only to stabilize the artery during cannulation*. It is never tied. The catheter is inserted under direct vision without making an incision in the vessel.

**Figure 18-30** The skin incision is closed, and the catheter and connector are secured to the heel with tape.

At a 10° angle, a 22-ga Angiocath with the catheter bevel down is inserted into the artery over the surface of the forceps. When blood return is seen, the catheter is advanced over the stylet to its full length (Fig. 18-29) . The needle stylet is then removed, and the catheter is connected to the T-connector tubing and a three-way stopcock that has been prefilled with heparinized flush solution. Patency is checked by observation of blood return with pulsations; the catheter is then flushed slowly and gently. The silk suture is removed, and the skin incision is sutured. The catheter is

sutured to the skin over the heel. The catheter and connector are then secured to the heel with tape (Fig. 18-30) . The stopcock is then connected to the infusion line.

**Complications**

The complications of arterial cutdown are similar to those of percutaneous arterial catheterization. They include hemorrhage, thrombosis, or spasm resulting in loss of tissue; infections; permanent scars; and nerve damage. Complications have been reported with all types of arterial cutdown. Follow-up data and computed tomography data suggest an association between temporal artery catheterization by the cutdown technique and cerebral infarct that may result in hemiparesis. [122] Therefore, temporal artery catheterization should be the last choice for arterial catheterization.

# Chapter 19 - Arterial Puncture and Cannulation

**William J. Barker**

Arterial blood gas evaluation provides useful information that is essentially unavailable by other means. The respiratory status and acid-base equilibrium of individuals with pulmonary disorders, drug overdoses, and metabolic diseases may be evaluated through this procedure. The current sophistication of critical care medicine would be impossible without arterial access, permitting continuous arterial pressure monitoring and frequent blood sampling for metabolic and hematologic indices.

Currently access to the arterial system can be easily obtained in most patients. Many improvements in arterial access methodology and equipment have occurred since Hales, in 1733, first used a technique similar to today's cutdown technique to attach a fluid column for measurement of the blood pressure of a horse. [1] J. L. M. Poiseuille first introduced the use of a mercury manometer for the measurement of blood pressure in 1828. In 1847, Karl Ludwig graphically recorded blood pressure fluctuations by placing a float with a pointer on the mercury column, permitting the pointer to scratch a smoked drum rotating adjacent to the column. Today's manometers, transducers, and recorders use electronics rather than simple mechanics, thus permitting mathematical waveform analysis in addition to visual analysis.

## INDICATIONS AND CONTRAINDICATIONS

Many patients who are seen in the emergency department benefit from arterial puncture or cannulation (Table 19-1) . Most commonly, arterial blood gases are used to evaluate individuals who have significant respiratory pathology. Although most patients with respiratory illness may be managed without arterial puncture, arterial blood gas determination is imperative when they are severely ill. Critically ill patients with nonrespiratory disease may need frequent metabolic and electrolyte monitoring. For instance, the patient with severe diabetic ketoacidosis cannot be managed properly without frequent pH, electrolyte, and glucose measurements.

| TABLE 19-1 -- Arterial Puncture and Cannulation | |
| --- | --- |
| **Indications** | **Relative Contraindications** |

| | |
|---|---|
| Blood gas sampling | Previous surgery in the area, especially cutdown |
| Continuous pressure monitoring | |
| Frequent blood sampling for any purpose | Anticoagulation |
| | Coagulopathy |
| Diagnostic angiography | Skin infection at site |
| Therapeutic embolization | Atherosclerosis |
| | Decreased collateral flow |

Although arterial blood gas analysis is the most frequent indication for arterial puncture, all blood chemistry tests can be performed from an arterial sample. Blood cultures may also be obtained through an indwelling arterial line, with sensitivity and specificity equal to those of cultures obtained from a venipuncture site. [2] [3] When frequent blood sampling is required, it is easier and certainly more humane to insert an indwelling arterial cannula. The nurse who is caring for the patient may then sample the blood as needed.

The most common indication for long-term arterial cannulation is continuous monitoring of arterial blood pressure. An electromechanical pressure transducer attached to an oscilloscope screen allows continuous observation of arterial systolic and diastolic pressures. This capability is most useful in a critical care unit but is also helpful when available in an emergency department. Many situations, such as the use of vasoactive drugs (e.g., nitroprusside and dopamine), require continuous arterial pressure monitoring. The response of trauma and cardiac patients to resuscitative efforts also may be more easily followed in this manner.

Another common use of arterial puncture is radiologic diagnostic imaging of the arterial system. Obviously this is an uncommon indication in the emergency department, but it

may be considered for suspected cases of peripheral arterial trauma or embolism. Although this procedure is fairly simple to perform, arterial access for diagnostic imaging is most often performed by a radiologist. Arterial puncture is also used by cardiologists and radiologists for coronary, cerebral, renal, and other central arterial angiography. Aortography can be very important for chest trauma and suspected cases of dissecting or leaking aneurysms.

Few contraindications to arterial puncture exist; none are absolute. Arterial puncture performed in patients who are anticoagulated or who have other coagulopathies should be undertaken with extreme care. Luce and associates reported 7 patients with compression neuropathies secondary to hematoma after arterial puncture, all of whom were anticoagulated at the time of puncture. [4] Repeated arterial sampling in these patients may necessitate insertion of an indwelling cannula to minimize the number of puncture sites in the arterial wall. Arterial cannulation or puncture should be used in a patient receiving thrombolytic therapy *only* if it will provide essential data that cannot be obtained in any other fashion.

The presence of severe atherosclerosis, with or without diminution of flow, is a relative contraindication to arterial puncture, especially when followed by cannulation. The existence of a bruit or a palpable decrease in the pulse should warn the physician of the presence of intravascular disease. If either is noted and the arterial puncture is imperative, an alternative site should be considered.

Evidence of decreased or absent collateral flow in areas in which flow normally exists should also lead one to consider an alternative site. A positive Allen test (discussed in Techniques) may possibly eliminate the radial and ulnar arteries as sites of cannulation. One should also avoid puncture of an artery when infection, burn, or other damage to cutaneous defenses exists in the overlying skin.

## EQUIPMENT

The equipment used for arterial cannulation has a great influence on the accuracy of pressure measurements. The frequency responses of tubing, transducers, and other components of the monitoring system influence the measurement accuracy of systolic and diastolic pressures.

The dynamic response characteristics of the monitoring system are of minimal importance if the clinician is interested primarily in the mean arterial pressure (MAP). [5] In the emergency department, trends in the MAP are more useful than absolute values for systolic and diastolic pressures.

The various catheter types have demonstrated similar frequency response characteristics, but studies have shown more variable effects on complication rates. Teflon catheters have been implicated as a factor associated with increased thrombosis in 1 study, [6] whereas the results of other studies deny any such effect. [7] [8] [9] The diameter of the indwelling cannula seems to have a more consistent effect; the incidence of thrombosis is inversely related to the ratio of vessel lumen to catheter diameter. [10] Thus, for a given vessel size, the incidence of thrombosis will decrease as

the catheter diameter decreases. Catheter choice should be influenced by availability and convenience of use of a particular brand. A short catheter is ideal for peripheral artery cannulation, whereas a longer over-the-needle catheter is preferable for the femoral artery (Fig. 19-1) . Downs and associates suggest that thrombosis is less likely with a nontapered catheter. [11] An 18- to 20-ga catheter should be used in adults. Small children and infants require a 22- to 24-ga catheter, which may need to be inserted through a cutdown technique.

The tubing that connects the catheter to the pressure transducer has a significant effect on monitoring systems. The higher the frequency response of the entire system, the more accurate the determination of systolic and diastolic pressure. [5] [12] A stiff, low-capacitance plastic tubing should be used, and the manometer should be placed as close as possible to the patient, because the frequency response of a tube is inversely related to its length. [12] [13] [14]

After the pressure wave is transmitted from the artery through the catheter and connecting tubing, a measuring device is required to obtain a numerical value for the arterial pressure. Currently the most commonly used device is an electromechanical pressure transducer that changes a mechanical pressure pulse, the fluid wave, into an electrical

**Figure 19-1** Catheters that may be used for arterial line insertion. Short, small-gauge over-the-needle catheters are ideal for peripheral artery cannulation *(top)*. The long over-the-needle catheter is used for femoral arterial lines *(bottom)*.

**Figure 19-2** (Figure Not Available) Assembly technique for anaeroid manometer system. The middle and proximal extension tubings contain heparinized saline. The middle extension tubing is arranged to form a fluid meniscus at the same level as the heart when the proximal stopcock is closed to the middle tubing (i.e., no pressure input). The distal extension tubing is filled with air and held vertically so that there is no saline contamination of the manometer at maximal pressures. Approximately 10 to 12 cm of air in the distal and middle tubings is optimal. The same system can be used with a mercury manometer in place of the anaeroid manometer. Sterility of the extension tubing and stopcocks is essential. (From Zorab JSM: Continuous display of the arterial pressure: A simple manometric technique. Anaesthesia 24:433, 1969. Copyright © 1969 by the Association of Anaesthetists of Great Britain and Ireland. Reproduced by permission.)

signal, which is then displayed on an oscilloscope. Additional circuitry can be added to display the systolic and diastolic pressure as numerical values. Various minicomputer systems that allow computation of mean arterial pressure, trend monitoring, and other capabilities in addition to displaying the systolic and diastolic pressures are available. So many transducer and oscilloscope combinations exist that a discussion of their relative merits is beyond the scope of this chapter.

Intravascular transducers are also available but have many potential disadvantages and are used infrequently. They are fragile, temperature sensitive, of variable quality, and much more difficult to place into a vessel than is a catheter. Fibrin deposition on the device is also a common finding. The greatest, and possibly only, advantage of these intravascular transducers is the elimination of potential error induced by catheters,

stopcocks, and connecting tubing.

Less expensive means of deriving a number representative of the arterial pressure are available, especially if one is interested in determining only the mean arterial pressure (MAP). Zorab describes the use of an anaeroid manometer connected to the arterial system by a catheter filled with heparinized saline. [15] The catheter is arranged to have a fluid meniscus at the same level as the heart when there is no pressure input. The meniscus is below an air column in a vertical tube that is long enough to avoid saline contamination of the manometer at maximal pressures (Fig. 19-2) (Figure Not Available) . A mercury manometer, especially a J tube, may also be used in place of an anaeroid manometer in the previously described system for measurement of MAP.

For continuous arterial pressure monitoring, some method of flushing the system is necessary to maintain patency of the catheter lumen. This may be as simple as a three-way stopcock through which the tubing is intermittently flushed with heparinized saline. There are continuous flush devices that are designed to push a set amount of fluid (usually 2 to 3 mL/hour) through the line. A typical monitoring system that includes this device is shown in Figure 19-3 (Figure Not Available) . The pressure transducer is usually mounted at the level of the patient's heart on a bedside pole.

**Figure 19-3** (Figure Not Available) Arterial pressure monitoring systems. *A,* System for continuous flush. Heparin (2 mL of a 1:1000 unit solution) is added to a 1-L bag of normal saline, and the bag is pressurized to 300 mm Hg using a metered blood pump (not shown). The continuous flush device is set to deliver 3 mL/hour of the heparinized saline. A mechanical pressure transducer (Pressurveil, Concept Co.) is depicted. The transducer device is a sterile, inexpensive, fully assembled monitor that can be used during patient transfer. Alternatively, the electronic transducer depicted in *B* may be used. *B,* System for manual flush. A heparinized saline flush solution can be injected manually through a syringe at the proximal or distal port. The transducer dome should be maintained at the level of the patient's heart. (From Beal JM [ed]: Critical Care for Surgical Patients. New York, Macmillan, 1982. Reproduced by permission.)

**Figure 19-4** Arterial blood gas kit. Contents include skin preparation pads, prefilled heparinized syringe, stopper for syringe, needles, gauze sponges, and plastic bag for crushed ice.

The equipment needed to percutaneously obtain a single sample for arterial blood gas analysis is simple and readily available in any hospital, often in the form of a prepackaged kit (Fig. 19-4) . The necessary items for arterial line placement are listed in Table 19-2 and referred to in the section that describes the procedures. Obtaining a sample from an indwelling arterial line requires only 2 syringes, 1 of which has been heparinized.

## SITE SELECTION

Selection of an arterial site is the first step in placing an indwelling cannula or obtaining a sample of arterial blood for a blood gas analysis. Successful cannulation of the radial, ulnar, brachial, axillary, dorsalis pedis, and femoral arteries in children and adults is possible. The temporal and umbilical arteries are often cannulated in infants and

neonates. However,

| TABLE 19-2 -- Equipment for Insertion and Maintenance of an Indwelling Arterial Cannula |
|---|
| Percutaneous insertion |
| Alcohol or iodophor solution |
| 1% lidocaine or other local anesthetic solution |
| 4-0 nylon or silk suture on skin needle |
| 10-cm × 10-cm dressing sponges |
| Adhesive tape |
| Iodophor ointment |
| Arm board for brachial, radial, or ulnar cannulations |
| Appropriate sized intravenous catheters |
| Syringes |
| Pressure tubing |

| |
|---|
| 2 three-way stopcocks |
| Pressure transducer |
| Connecting wire |
| Oscilloscope, thermal graph, or other output display |
| Heparinized saline |
| Pressure blood infuser, set up with continuous flush device |
| Additional equipment required for cutdown insertion technique |
| Scalpel blade (No. 10) |
| 0-0 silk sutures (2 or more) |
| Small hemostat |
| Forceps |

radial artery cannulation is also very safe for these patients [16] (see Chapter 18) . The radial, brachial, and femoral arteries are usually punctured for blood gas sampling in adults.

The potential consequence of total loss of blood flow through a vessel due to

intraluminal thrombosis is one of several variables that must be considered when choosing a site for arterial puncture. Arteries known to have good collateral blood flow, such as the radial and dorsalis pedis, are thus favored. Determining the effect of the site chosen on the ease of patient care will be appreciated by those who will subsequently be providing that care. An arterial line in the lower extremities may be preferred during a procedure on the upper body, whereas a femoral or axillary line may be poorly tolerated by patients who are capable of positioning themselves. Characteristics of the common arterial sites are discussed following general descriptions of the techniques of arterial puncture and cannulation.

## TECHNIQUES

### Percutaneous Technique for Single Arterial Puncture

To obtain a single sample of arterial blood by the percutaneous method, a small (5-mL) syringe is attached to a 20- to 22-ga needle. A smaller needle may be required for young children or individuals who have had many previous punctures. To prevent clotting of the sample, 1 or 2 mL of a heparinized saline solution (1000 IU/mL) are drawn into the syringe to coat the barrel and needle and are then ejected through the needle shortly before puncture. All of the visible heparin is ejected, so that all that remains is enough anticoagulant to coat the barrel and fill the dead space of the syringe and needle, minimizing heparin-related errors.

Because of the air-fluid boundary in the heparin storage bottle, heparin solution has a higher $P_{O2}$ and a lower $P_{CO2}$ than blood, and changes in these parameters reflect a dilutional effect. The addition of 0.4 mL of heparin solution to a 2-mL sample of blood (dilution of 20%) will lower the $P_{CO2}$ by 16%. A falsely low $P_{CO2}$ is the most clinically significant change caused by excess heparin. [17] [18] $P_{O2}$ levels are not significantly altered by the addition of heparin in most instances, although a slight increase in $P_{O2}$ has been reported. The pH is affected (lowered) only if high concentrations of heparin are used (25,000 IU/mL), but generally the tremendous buffering capacity of blood maintains a normal pH. The dead space of a 5-mL Luer-Lok glass syringe and a 22-ga, 3.8-cm needle is 0.2 mL. Therefore, to minimize heparin-related error, *at least 3 mL of blood should be collected*, even though blood gas analysis equipment requires only a 0.5-mL sample. Some packaged blood gas syringes contain a powdered heparin that mixes with the arterial blood. Although thrombosis of the needle may be more common with this system, smaller volumes (1 mL) of blood can be used.

The arterial pulse is palpated to ascertain the location of the vessel, and the overlying skin is prepared sterilely with an iodophor or other antiseptic solution. The patient's skin should then be anesthetized with a wheal of local anesthetic without epinephrine placed through a small-gauge (25- or 27-ga) needle. If the patient is unresponsive to pain in the

**Figure 19-5** Arterial puncture at the wrist. The index and middle fingers are used to isolate and outline the course of the pulsating artery before insertion of the needle. Contrary to the illustration, latex gloves should be worn during the procedure.

area, this step may be omitted. Care must be taken to use a small amount of local anesthetic, because a large wheal may obscure the pulse.

The arterial pulsation is then isolated between the index and middle fingers of the *gloved* nondominant hand (Fig. 19-5) . The skin should be punctured through the anesthetic wheal, and the needle should be advanced toward the pulsating vessel. The needle should form an angle of about 15° to 20° with the skin. A larger angle is required for femoral artery puncture. Once the needle has entered the arterial lumen, the syringe plunger should be allowed to rise with the arterial pressure to minimize the chance of venous sampling. If no blood flow is obtained, the needle should be withdrawn slowly, because both walls of the vessel may have been punctured. A sample may be obtained during withdrawal. Redirection of the needle should occur only when the needle has been retracted to a location just deep to the dermis.

Maher and Dougherty describe the use of a hand-held Doppler ultrasound probe to aid arterial puncture. [19] The probe is held over the artery proximal to the puncture site. Loss of audible pulsations suggests vessel compression with imminent arterial puncture.

After a sample of at least 3 mL of blood has been obtained, the needle is removed from the artery, and firm pressure is applied at the puncture site for a minimum of 5 minutes. Ten to 15 minutes of pressure is required if the patient is on anticoagulant therapy or has a coagulopathy.

Proper handling of the sample is very important. When the needle is withdrawn, it is imperative to expel any air bubbles that are present in the syringe to avoid false elevation of the $P_{O2}$ . [20] Air in the sample will significantly increase the $P_{O2}$ (mean increase, 11 mm Hg) after 20 minutes of storage, even if kept at 4 °C. The pH and $P_{CO2}$ are not significantly altered by air bubbles if the blood is stored at 4 °C for 20 minutes.

Removal of air is neatly and easily accomplished by placing an alcohol wipe or gauze sponge on the needle and tapping the inverted syringe to force any air to the top. Air is then pushed out of the needle, and any blood that is spilled will be caught by the sponge (Fig. 19-6) . The needle is then plugged or removed, and the syringe is capped to ensure anaerobic conditions. Blood gas analysis is ideally performed immediately, but if this is not feasible, the sample may be stored in ice water for 1 hour with limited deterioration. [20] [21] If the sample is stored anaerobically, regardless of the temperature, the $P_{O2}$ , $P_{CO2}$ , and pH are relatively stable for up to 20 minutes. If blood is stored at room temperature

**Figure 19-6** Removal of air bubbles from the syringe. *A,* Air bubbles are finger-tapped to the top of the syringe. *B,* An alcohol swab is placed over the top of the needle. *C,* The plunger is advanced to expel air while drops of blood are collected on the alcohol swab. After removal of the bubbles, the syringe is capped and sent to the laboratory.

for longer than 20 minutes, the $P_{CO2}$ will increase and the pH will decrease, probably as a result of leukocyte metabolism. In a stored sample, the $P_{O2}$ varies to such an extent

that the change is unpredictable for chemical interpretation at 30 minutes, regardless of storage method. High leukocyte or thrombocyte counts, such as those seen in leukemic patients, may shorten acceptable storage intervals. [22] [23] Local anesthesia will make the procedures of arterial puncture easier for both the patient and the physician; however, 1 study found no significant alterations in $P_{CO_2}$ or pH from the pain or anxiety of an unanesthetized arterial puncture (Table 19-3) . [24]

**Percutaneous Technique for Arterial Cannulation**

Percutaneous puncture is the preferred method for arterial cannulation. It is also the method of choice for obtaining an isolated blood gas sample (see preceding section) when an indwelling cannula has not been placed.

Once the site has been selected, the skin should be prepared with iodophor or other antiseptic solution, and sterile drapes should be placed around the area. Sterile technique should be meticulously maintained throughout the procedure. Levy and colleagues describe an alternative skin preparation method for central venous catheters that uses a 1-minute isopropyl alcohol scrub followed by catheter placement through an iodophor-impregnated adherent film (Ioban Z antimicrobial film, 3M Company, St. Paul). [25] Catheter tip and glove tip contamination were virtually eliminated. This technique may be considered for arterial cannulation as well.

The artery is entered as described in the preceding section, using a catheter-over-the needle apparatus. Once the artery has been entered, bright red blood should be visible in the flash chamber of the cannula. Advance the needle approximately 1 mm into the vessel lumen (Fig. 19-7) (Figure Not Available) , then fix the needle while threading the catheter further into the lumen. When the needle is withdrawn, blood will pulsate from the catheter hub. Inadvertent puncture of the back wall of the artery can occur, and indeed, a variation of the percutaneous technique is to puncture both walls of the

| **TABLE 19-3** -- Parameters that Affect Interpretation of Arterial Blood Gases | | | |
|---|---|---|---|
| **Parameter** | **Heparin** * | **Air Bubble in Sample** | **Delayed Analysis** |
| $P_{O_2}$ | No signficant change | Elevated | Variable ¶ |
| $P_{CO_2}$ | Lowered | No significant changes § | Elevated * |

| pH | Unchanged | No significant changes § | Lowered * |
|---|---|---|---|

* Use only 1000 IU/mL concentration; fill dead space of needle and syringe only, and collect 3 mL of blood.
Anaerobic storage at room temperature for 20 minutes results in no significant change.
There are reports of slight increases in $P_{O_2}$ with excessive heparin.

¶ Changes unpredictable at 30 minutes, regardless of storage method.
The falsely lowered $P_{CO_2}$ that occurs with added heparin is the most clinically significant change noted.
pH may be decreased if a large volume of concentrated heparin (25,000 IU/mL) is used.
§ If stored at 4° C for 20 minutes.
* *Minimal changes up to 2 hours, if stored at 4° C.

**Figure 19-7** (Figure Not Available) Percutaneous arterial cannulation at the wrist. The catheter unit is advanced 1 to 2 mm into the vessel lumen after blood first appears in the flash chamber. While the needle is fixed, the catheter is threaded over the needle. (From Beal JM [ed]: Critical Care for Surgical Patients. New York, Macmillan, 1982. Reproduced by permission.)

vessel with a single pass. If the back wall is punctured, the needle is withdrawn from the catheter, and the catheter is slowly pulled back until a steady stream of blood flows from its hub. The catheter is then advanced carefully further into the lumen. The double-puncture method is especially useful for cannulating small vessels. Jones and colleagues reported no increase in complications when both walls, rather than one, are punctured. [26]

Once the vessel has been entered, occasionally one will encounter difficulty advancing the catheter into the lumen. The "liquid stylet" method may aid further passage of the catheter. [27] A 10-mL syringe should be filled with about 5 mL of sterile normal saline. The syringe is then attached to the catheter hub, and 1 to 2 mL of blood should be easily aspirated to confirm intraluminal position. The fluid from the syringe is then slowly injected, and the catheter is advanced behind the fluid wave. Catheter sets are available with a wire stylet that permits a modified Seldinger technique for catheter placement; the over-the-needle catheter follows the self-contained guide wire during cannulation. One readily available device of this type is the Arrow arterial catheterization system (Arrow International, Inc., Reading, Pa) (Fig. 19-8) (Figure Not Available) . This device is available alone, in catheter exchange sets, and in complete single-use kits that are packaged in a container that can serve as a disposable wrist support.

Once the catheter has been placed successfully, it should be advanced until the hub is in contact with the skin. The catheter is then secured by fastening it to the skin with suture material. Silk (4-0) or nylon (5-0) sutures provide the best anchoring. To accomplish this, a moderate bite of skin is taken with the needle, and a knot is tied in the suture. Care should be taken to avoid pinching the skin too tightly. The loose ends of the suture are then tied around the catheter or its hub without occluding the lumen by constriction (Fig. 19-9) .

After tying the catheter in place, a drop of antibiotic ointment is applied to the puncture site, and a self-adhesive dressing is applied over the area. The catheter and its

connecting tubing are further secured with sterile sponges and adhesive tape. All tubing connections must be tight and secure. If the tubing becomes disconnected inadvertently, the patient may exsanguinate rapidly.

When successful arterial cannulation has been performed,

**Figure 19-8** (Figure Not Available) Step-by-step arterial cannulation, using the guide wire technique (Arrow arterial catheterization kit). (Courtesy of Arrow International, Inc., Reading, Pa.)

the catheter should be attached to a pressurized fluid-filled system. If the catheter has been placed for monitoring arterial blood pressure, it should be connected to a mechanical or electrical transducer by a short length of rigid plastic tubing filled with saline. A three-way stopcock is interposed between the patient and the transducer for blood gas sampling and to allow flushing of the system with heparinized saline (2 mL 1:1000 heparin/L of saline). Flushing

**Figure 19-9** Illustration of a technique for securing a vascular catheter to adjacent skin.

can be periodic or continuous at a rate of 3 to 4 mL/hr through a continuous flow device (see Fig. 19-3) (Figure Not Available) .

Procurement of a blood sample from this system is easily performed. A syringe is attached to the three-way stopcock, and blood is aspirated and discarded to clear the line. Studies examining the necessary discard volume of flush-blood solution have found considerable variation dependent on the volume of the system. [28] [29] Short lengths of tubing between the catheter and aspiration port minimize the discard volume. For a tubing length of 91 cm (36 in.), 4 to 5 mL should be aspirated [29] ; for a tubing length of 213 cm (84 in.), 8 mL should be aspirated. [28] A second syringe, which has been heparinized, is then attached, and 3 mL of blood is aspirated and sent for blood gas analysis. If the blood is to be used for other tests, the second syringe does not need to be heparinized. The stopcock and line should be flushed after sampling to avoid clotting.

**Seldinger Technique for Arterial Cannulation**

An alternative method of placing an indwelling cannula is the Seldinger technique, [30] which is described in detail for

**Figure 19-10** Placement of an arterial line using the cutdown technique. Note that the catheter enters the surgical wound percutaneously to minimize bacterial entry into the healing wound and permit better stabilization of the catheter. Catheter entry of the vessel is more parallel to the vessel than is illustrated. Ligatures are used only to *temporarily* isolate the artery and to control bleeding. *The artery should not be tied off.* The catheter is secured by suturing the hub to the skin (see Fig. 19-9) .

venipuncture in Chapter 21 . A needle is percutaneously placed into the arterial lumen, as described previously. A guide wire is then placed through the needle into the vessel lumen, and the needle is removed. A catheter is then threaded over the wire, and the

wire is pulled out. As mentioned before, one commercial catheter permits the Seldinger technique to be performed without separate guide wire manipulation.

**Cutdown Technique for Arterial Cannulation**

The cutdown technique is another common method of obtaining arterial access. Cannulation is performed after direct visualization of the vessel. A cutdown can be performed on any artery but is most commonly reserved for the brachial and other distal limb arteries. After a site has been selected, the overlying skin should be surgically prepared with an iodophor solution. The physician then puts on sterile gloves and drapes the extremity. Local anesthetic solution is injected subcutaneously (SQ) in a horizontal line 2 to 3 cm long and perpendicular to the artery. Local anesthesia is not necessary if the patient is unconscious or is otherwise anesthetic at the cutdown site.

Using a scalpel with a No. 10 or 15 blade, the skin is incised along the anesthetic wheal. Underlying tissues are spread parallel to the artery with a mosquito hemostat. The pulse is palpated repeatedly throughout the procedure to ensure proper positioning. Once the surrounding soft tissue has been removed, exposing the artery for a distance of approximately 1 cm, the artery should be isolated by passing 2 silk sutures underneath it, using the hemostat. Strip away only enough perivascular tissue to expose the artery. Perivascular tissue will help to limit bleeding at the time of catheter removal. A catheter-over-the-needle device, such as that used in the percutaneous method, is then introduced through the skin just distal to the incision and advanced into the surgical site (Fig. 19-10) . [31] The arterial wall is punctured with the needle tip, and the catheter is threaded into the vessel lumen. When this has been accomplished, the 2 silk sutures, which have been used only to control the vessel, are removed, and the skin incision is closed. *The artery is not tied off as the vessel would be during a venous cutdown.* Firm pressure, as used following arterial puncture, should be applied over the cutdown site. The separation of the soft tissue during the procedure may allow considerable hemorrhage into the tissue if pressure is not applied. The catheter is secured to the skin in the same manner used for the percutaneous method (see Fig. 19-9) .

# ARTERIES

**Radial and Ulnar**

The radial artery is the artery that has most frequently been used for prolonged cannulation. A widespread collateral flow exists in the wrist. There are two major palmar anastomoses, known as *arches* (Fig. 19-11) (Figure Not Available) . The superficial palmar arch lies between the aponeurosis palmaris and the tendons of the flexor digitorum sublimis. The arch is formed mainly by the terminal ulnar artery and the superficial palmar branch of the radial artery. The other major communication of these 2 vessels, the deep palmar arch, is formed by connections of the terminal radial artery with the deep palmar branches of the ulnar artery. [32] Some collateral flow is almost always present at the wrist, with the deep arch alone being complete in 97% of 650 hand dissections. [33] Despite the findings of Coleman and Anson at autopsy, Friedman noted the absence of palpable ulnar pulses in 10 of 290 (3.4%) healthy children and young adults. [34] Interestingly, this was always a bilateral finding. Radial pulses were

present in 100% of the subjects.

A simple test has been recommended to determine the presence of collateral flow in the hand. This procedure has seen many modifications [35] [36] since being described by Allen in 1929. [37] In a cooperative patient, the basic Allen test is performed as follows: The examiner occludes the radial and ulnar arteries with digital pressure, and the patient is asked to tightly clench the fist repetitively to exsanguinate the hand. The hand is then opened, and the examiner releases the occlusion of the ulnar artery (Fig. 19-12) (Figure Not Available) . After several

**Figure 19-11** (Figure Not Available) Arterial anatomy of the hand and wrist. (From Ramanathan S, Chalon J, Turndorf H: Determining patency of palmar arches by retrograde radial pulsation. Anesthesiology 42:758, 1975. Reproduced by permission.)

**Figure 19-12** (Figure Not Available) Allen test. Before puncturing the radial artery it is important to be sure that a competent ulnar artery is present. This can be done as follows: *1,* The examiner compresses both arteries, and the patient repeatedly makes a tight fist to squeeze all the blood out of the hand. *2,* The patient then extends the fingers, and the examiner observes the blanched hand. *3,* Compression of the ulnar artery is released, and the examiner observes the hand fill with blood. If filling does not occur within 5 to 10 seconds, radial artery puncture should not be done. If brisk filling occurs, the test is then repeated with release of the radial artery to assess radial artery patency. If both radial and ulnar arteries demonstrate patency, the wrist may be used for arterial puncture. (From Schwartz GR [ed]: Principles and Practice of Emergency Medicine. Philadelphia, WB Saunders, 1978, p. 354. Reproduced by permission.)

minutes, the test is repeated with release of the radial artery. Rubor should rapidly return to the hand with release of either vessel.

An abnormal (positive) Allen test, suggestive of inadequate collateralization, is defined as the continued presence of pallor 5 to 15 seconds after release of the artery. [10] [32] [38] [39] If the return of color takes >5 to 10 seconds, radial artery puncture should not be performed. One must be careful to avoid overextension of the hand with wide separation of the digits, because this may compress the palmar arches between fascial planes and give a false-positive result. [38] Barber and associates [35] reported a modified Allen test that is useful in unconscious or anesthetized patients who cannot clench their fists. An Esmarch bandage is used to exsanguinate the hand, and the test is performed as previously described. Time permitting, performance of some variation of the Allen test is desirable before ulnar or radial puncture for cannulation or blood gas sampling.

A study by McGregor has shown that positive Allen test results may be false positives when further evaluated by intra-arterial fluorescein angiography. [40] Slogoff and colleagues, in a study of 1700 radial artery cannulations for monitoring purposes, reported 16 cases of radial artery cannulation in patients with abnormal Allen tests, none of whom developed ischemia or abnormality of radial flow after cannulation. [41] Of note, the Allen test was performed on only 411 of their 1700 patients, giving an abnormal result 4% of the time. Cardiovascular surgical patients of the Texas Heart Institute have been monitored with radial artery cannulation for 20 years without routine performance of the Allen test. Nonetheless, when alternative sites exist, the results of an Allen test should be documented on the chart for medicolegal reasons, and an alternative site used. A normal Allen test is desirable, but it is not a guarantee against

digital ischemia following radial artery cannulation. [42]

Once adequate collateral flow has been ascertained, arterial puncture may be performed. At the wrist, the radial artery rests on the flexor digitorum superficialis, flexor pollicus longus, and pronator quadratus, and against the radius. [33] Just distal to the styloid process of the radius, the artery winds around the lateral aspect of the wrist to the dorsum of the hand. The pulsation of the artery should be isolated on the palmar surface of the wrist, where it is superficial. Dorsiflexing the wrist at about a 60° angle over a towel or sandbag, with or without fixing the wrist to an arm board, will help isolate and fix the artery (see Fig. 19-7) (Figure Not Available) . [10] [43] [44]

The ulnar artery may occasionally be used but is technically more difficult to puncture than the radial artery because of its smaller size. At the wrist, the ulnar artery runs along the palmar margin of the flexor carpi ulnaris in the space between it and the flexor digitorum sublimis. [33] In this area it is in intimate contact with the ulnar nerve. The ulnar nerve and artery pass into the hand just radial to the pisiform bone. The ulnar artery can often be made more accessible with dorsiflexion of the wrist.

**Brachial**

Barnes and colleagues [8] monitored 54 patients with an 18- or 20-ga Teflon catheter percutaneously placed in the left brachial artery at the antecubital fossa. None of these patients developed Doppler evidence of brachial artery obstruction; however, partial to complete obstruction of the radial artery was noted in 2 patients, and of the ulnar artery in 1 patient. None of these 3 patients exhibited ischemic symptoms. These researchers also noted no clinical evidence of ischemia in 1000 brachial catheterizations over a period of 3 years. Thus the brachial artery appears to be a safe site for arterial puncture, although collateral circulation in this area is not as great as in the hand.

The brachial artery begins as the continuation of the axillary artery and ends at the head of the radius, where it splits into the ulnar and radial arteries. The preferred site of puncture of the brachial artery is in or just proximal to the antecubital fossa. In this region the vessel lies on top of the brachialis muscle and enters the fossa underneath the bicipital aponeurosis (Fig. 19-13) (Figure Not Available) . The median nerve runs along the medial side of the artery. Owing to reduced collateral circulation and the necessity of maintaining the arm in extension

**Figure 19-13** (Figure Not Available) The right brachial artery and its branches. (From Christensen JB, Telford IR: Synopsis of Gross Anatomy. New York, Harper & Row, 1966. Reproduced by permission.)

for puncture or prolonged cannulation, more distal vessels are preferred when the upper extremity is chosen for cannulation.

**Axillary**

Axillary artery cannulation as described by Adler and coworkers [45] is also a safe means of monitoring arterial blood pressure for a long period of time. The left axillary artery is

preferred in order to decrease the possibility of cerebral embolization of flush solution or thrombus. The path from the left subclavian to the left carotid artery is less direct than on the right side, whereas the vertebral arteries are equally vulnerable.

To cannulate the axillary artery, the arm is held in 90° abduction. The axillary pulse is then palpated high in the axilla between the insertion of the pectoralis major and the deltoid muscles. The artery may then be cannulated percutaneously with or without a Seldinger guide wire. This site is technically more difficult and time consuming and probably should be avoided in the emergency department. No studies have been reported regarding large numbers of axillary punctures; therefore, the relative safety of this location cannot be determined.

## Dorsalis Pedis

The dorsalis pedis artery is a continuation of the anterior tibial artery. Anterior to the ankle joint, the dorsalis pedis runs from approximately midway between the malleoli to the posterior end of the first metatarsal space, where it forms the dorsal metatarsal and deep plantar arteries. The lateral plantar artery, which is a branch of the posterior tibial, passes obliquely across the foot to the base of the fifth metatarsal. The plantar arch is completed where the lateral plantar artery joins the deep plantar artery between the first and second metatarsals. On the dorsum of the foot, the dorsalis pedis artery lies in the SQ tissue parallel to the extensor hallucis longus tendon, between it and the extensor digitorum longus (Fig. 19-14) (Figure Not Available) . [5] The artery should be cannulated in the midfoot region. Although this vessel is amenable to cutdown, the vascular anatomy of the foot is quite variable. This is of no consequence if a pulse can be palpated, but Huber, in his dissection of 200 feet, noted the dorsalis pedis artery to be absent in 12%. [46] In 16% of patients, the dorsalis pedis artery provides the main blood supply to the toes. [47] Collateral flow can be determined with a modified Allen test using the posterior tibial and dorsalis pedis arteries, but this is not as easily performed in the foot as in the hand. The pressure wave obtained with an electronic transducer attached to the dorsalis pedis artery will be 5 to 20 mm Hg higher than that of the radial artery and, in addition, will be delayed by about one tenth of a second. [48]

## Femoral

Currently, the femoral artery is the second most commonly used vessel for prolonged arterial cannulation. Several studies have demonstrated the efficacy and safety of using this vessel, and indeed, several investigators suggest that it should be the vessel of choice. [49] [50] [51] [52] The femoral artery is the direct continuation of the iliac artery. The femoral artery enters the thigh after passing behind the inguinal ligament, where, in most patients, it may be easily palpated at a point midway between the pubic symphysis and the anterior superior iliac spine. When puncturing this vessel, care must be taken to avoid the femoral nerve and vein, which are in close proximity to the artery on the lateral and medial sides, respectively (Fig. 19-15) (Figure Not Available) .

A longer cannula is required for the femoral artery owing

**Figure 19-14** (Figure Not Available) A 20-ga catheter in the dorsalis pedis artery, illustrating the

relationship to surrounding tendons. The catheter is secured with Steri-Drape. Splinting is not needed. (From Johnstone RE, Greenhow DE: Catheterization of the dorsalis pedis artery. Anesthesiology 39:655, 1973. Reproduced by permission.)

**Figure 19-15** (Figure Not Available) The right femoral vessels and some of their branches. The femoral nerve (not shown) lies lateral to the artery and may be deep to the artery. (From Warwick R, Williams PL [eds]: Gray's Anatomy. 35th ed. Edinburgh, Churchill Livingstone, 1973, p 676. Reproduced by permission.)

to the relatively greater depth at which it lies. The Seldinger technique is especially useful for this site, enabling placement of a 15- to 20-cm plastic catheter for prolonged monitoring. Catheter-over-the-needle devices may also be used but should be at least 10 cm long. Use of catheter-through-the-needle devices has been reported [51] but should be avoided because of the possibility of leakage around the catheter, which may occur with high arterial pressures owing to the loose fit of the cannula in the hole in the vessel wall. Regardless of the device used, the needle should enter the skin at an angle of about 45° instead of the usual 15° to 20°.

The extremely large ratio of arterial diameter to catheter diameter is thought to beneficially reduce the incidence of thrombosis, particularly total occlusion. However, occlusions have been reported with femoral cannulation for monitoring purposes. [53]

A commonly postulated disadvantage of this site is the possibility of increased bacterial contamination because of its proximity to the warm, moist groin and perineum; however, no studies confirm this hypothesis. [54] The femoral area is inconvenient for the patient who is awake and mobile, especially if the patient is capable of sitting in a chair. In spite of these theoretic difficulties, some large hospitals use femoral arterial lines almost exclusively, and the intensive care nursing staff is often more comfortable caring for these lines than those at other sites.

### Umbilical and Temporal

In the neonate, arterial access can be accomplished through the umbilical artery for a short period of time. After this artery closes, the temporal artery provides a safe alternative. Prian described the use of the temporal artery, noting its accessibility and the lack of clinical sequelae if it undergoes

thrombosis. [55] The cutdown method should be used with a 22-ga catheter after the artery's course has been traced with an ultrasonic flow detector. Because of the increasing accuracy of ear oximeters and the use of capillary blood gases for pH determination, prolonged arterial cannulation will become less frequent during infant care. Further discussion of infant arterial cannulation is provided in Chapter 18 .

## COMPLICATIONS

Long-term arterial cannulation is safe if care is taken to avoid complications. Almost all the difficulties one may encounter can be avoided or their incidence markedly decreased if one adheres to a few simple principles. Reported clinical sequelae of

arterial puncture and cannulation range from simple hematomas to life-threatening infections and exsanguination. The incidence of complications varies with the site and method of cannulation and with the skill and concern of the patient's physicians and nurses. It is difficult to compare complication rates at various sites, because most published studies have primarily used the radial artery. Many studies also report complications of puncture for arteriography and other procedures unrelated to long-term cannulation.

A commonly encountered problem is hematoma formation at the puncture site. Zorab [15] reported this complication in 50% of catheterizations. The bruising was of minimal clinical significance in Zorab's study, but leakage, when it occurs around the catheter or from the puncture site after the catheter's removal, can be of danger to the patient. Compression neuropathy secondary to bleeding has been reported after brachial artery puncture in anticoagulated patients; in some cases, surgical decompression has been necessary. [4] The large amount of soft tissue surrounding the femoral artery makes bleeding in this area difficult to control. [56] Large hematomas are not uncommon after femoral artery catheterization; indeed, Soderstrom and associates [49] reported 2 cases of bleeding that required transfusion after femoral puncture. Another patient suffered a large hematoma that became infected and required incision and drainage.

Prevention of bleeding complications may be accomplished with frequent careful inspection of the puncture site and with the use of prolonged compression after removal of the catheter or needle. Firm pressure should be maintained for at least 10 minutes after removal of a peripheral artery catheter and for a longer period of time after femoral cannulation or if the patient is anticoagulated. Five minutes of pressure is sufficient after puncture for a blood gas sample in an individual with normal coagulation. Exsanguination, a related complication, may occur if the arterial line apparatus becomes disconnected. This is more common in the obtunded or combative patient, and restraints are often required for patients with indwelling arterial cannulas. Exsanguination should not occur if tight connections are maintained throughout the system and if frequent, careful inspections of both the circuit and the patient are made.

Serious infections rarely complicate arterial cannulation. However, the incidence of catheter-related infections increases with prolonged cannulation. [54] [57] Catheters placed with sterile technique have an extremely low rate of infection up to 96 hours. Catheters changed over a guide wire every 96 hours have an infection rate of about 10% at the radial and femoral sites. [54]

Most infections begin as local infections at the puncture site and remain localized, although systemic sepsis has been reported. [56] Radial and femoral sites have a similar incidence of complications, but axillary cannulations seem to have a much higher incidence of infection, although no large studies of cannulation at this site exist. [54] [57] Arterial cannulas are more prone to infectious complications than other vascular catheters. Many mechanisms have been proposed for this. [58] [59] [60] The arterial pressure monitoring system usually consists of a long column of fairly stagnant fluid and is subject to frequent manipulation. Stamm and colleagues [59] found that patients were at greater risk for systemic infection if they had an arterial line and required frequent blood gas determinations than if they had the cannula alone. The sampling stopcock is

a site of frequent bacterial contamination.

The risk of infection also increases as the duration of cannulation is prolonged. Catheters should be changed after 4 days if continued monitoring is necessary. [57] [61] In addition, Makai and Hassemer [60] recommend changing the entire fluid-filled system, including transducer chamber-domes and continuous flow devices, every 48 hours. Shinozaki and coworkers [62] demonstrated a marked reduction in equipment contamination when the continuous flush device was located just distal to the transducer, as opposed to the device being positioned closer to the three-way stopcock used for sampling. This setup reduces the length of the static column of fluid between the sampling stopcock and the transducer. As mentioned previously, a drop of iodophor or antibiotic ointment applied to the puncture site decreases the incidence of local wound infection. [61]

Thrombosis of the vessel in which the cannula is placed is another frequently encountered problem. The incidence with which this occurs varies with the method used to determine the presence of the clot. Bedford and Wollman [7] found a >40% occlusion rate when radial artery catheters were left in place for >20 hours. All of these occluded vessels eventually recanalized. Angiographic studies show deposition of fibrin on 100% of the catheters left in place for >1 day, although clinical evidence of ischemia secondary to occlusion with thrombus is present in <1% in most studies. [6] Most reports of nonangiographic catheterizations that mention thrombosis are reports of studies of the radial artery. Therefore, it is difficult to compare the incidence of thrombosis at other sites, although during the 176 femoral catheterizations of Soderstrom and coworkers and Ersoz and associates, [49] [51] dorsalis pedis pulses were decreased in only 2 patients, and no clinical signs of ischemia were noted. Larger catheter sizes, trauma during cannulation, and the presence of atherosclerosis have all been postulated to increase the incidence of thrombosis; however, conflicting studies abound. Downs and colleagues associated tapered catheters with an increased incidence of thrombosis. [11]

Arterial spasm after puncture can predispose to thrombus formation and can even lead to ischemic changes without fibrin deposition. Successful reversal of spasm after intra-arterial lidocaine, reserpine, and phentolamine has been reported, but no reliable studies of efficacy in this situation have been published. [63] Thrombosis can be minimized by decreasing the duration of catheterization, by proper flushing, and by using larger arteries. Surgical embolectomy or thrombectomy is rarely required, because the smaller vessels that are most likely to occlude usually have good collateral

circulation. A normal (negative) result on an Allen test or a similar test suggests but does not ensure adequate collateral flow. [42] The larger femoral artery, which has poor collateralization, rarely occludes with catheterization for monitoring purposes.

Another complication of thrombosis is occlusion of the catheter. Times until occlusion of radial and femoral artery catheters have been compared, and it was noted that radial cannulas became occluded at an average of 3.8 days, whereas femoral cannulas occluded after 7.3 days. [49] The importance of this comparison is minimal if the clinician follows infection prophylaxis guidelines and changes arterial catheters after 4 days.

A few less common complications are easily prevented. One such complication, which occurs only with the percutaneous catheter-through-the-needle method, is catheter embolization. Once the catheter has been placed through the needle, it should never be pulled back, because the end of the catheter may be sheared off by the sharp needle bevel. If this occurs, surgical removal of the catheter tip is necessary.

Skin necrosis is a complication of radial artery cannulation involving an area of the volar forearm proximal to the cannula. [64] [65] Wyatt and colleagues [64] believe this is secondary to the poor blood supply of this area and state that taking the precautionary steps described previously prevents or decreases the incidence of necrosis. One feared complication of indwelling radial and brachial arterial catheters is the occurrence of a cerebrovascular accident secondary to embolization from flushing of the catheters. [11] [66] As little as 3 to 12 mL of flush solution have been shown to reflux to the junction of subclavian and vertebral arteries. [66] A fatality due to air embolism from a radial artery catheter has been reported and was re-created in a macaque model. [67] Although these animals are much smaller (7 kg) than an adult human, as little as 2.5 mL of air introduced at a relatively low flush rate was found to embolize in a retrograde fashion to the brain. Cerebral embolization can be prevented with the use of continuous flush systems (3 mL/hour) and by ensuring the integrity of the tubing and transducer systems to prevent air entry. In addition, small volumes (<2 mL) of intermittent flush solution should be used.

Complication rates also vary according to the method of arterial cannulation. Mortensen [68] studied the 3 main techniques (discussed in Techniques), but unfortunately, most of his arterial cannulations were for angiographic purposes. The complications associated with prolonged cannulation time are therefore underrepresented. For Mortensen's series, cutdown arteriotomy exhibited the lowest incidence of complications (7.7%), whereas the Seldinger technique had a complications incidence of 17.7%. Complications of percutaneous cannulation were 11.3%. Apparently, false passage of the guide wire, the catheter, or both were associated with increased intimal damage and complications. *It is imperative that the wire or catheter be advanced only if no resistance is met!*

In actuality, arterial puncture and cannulation are safe procedures when care is taken and basic principles are kept in mind. The operator should be skilled and should seek an atraumatic insertion. Once the monitoring system has been set up, it should be manipulated as little as possible. Any handling should be performed with a flawless aseptic technique. The tubing and other fluid-filled devices should be changed every 48 hours, and catheters should be inserted into a vessel that provides a vessel-to-catheter ratio that is as great as possible without compromising other needs. If these principles are followed and the patient and system are carefully inspected at frequent intervals, complications of arterial puncture and cannulation can be minimized.

## INTERPRETATION

An indwelling arterial cannula can provide valuable information about the hemodynamic status of a patient (through continuous pressure monitoring) and about the patient's

respiratory and metabolic status (through intermittent sampling for blood gas analysis and other blood tests). The partial pressure of carbon dioxide and pH of the blood can be used to define 4 major groups of metabolic derangement: respiratory acidosis or alkalosis, and metabolic acidosis or alkalosis. Rarely will a disorder be strictly classified into 1 of these groups; however, a simple chart such as that shown in Figure 19-16 (Figure Not Available) will assist one in determining the relative effects of metabolic and respiratory influence on the blood pH. See also the discussion in the Appendix. A rough estimate of the contribution of respiratory factors may be made by assuming that for every 10 torr (mm Hg) that the $P_{CO_2}$ varies from 40, the pH will inversely vary 0.08 pH unit from 7.4. Adequacy of oxygenation of the blood can be determined from the measured $P_{O_2}$ of the arterial blood and from the known concentration of oxygen that the patient is inspiring. To avoid iatrogenic complications of intensive care, one must be absolutely certain that the data are from an arterial sample that has been properly analyzed before basing

**Figure 19-16** (Figure Not Available) In vivo nomogram showing bands for uncomplicated respiratory or metabolic acid-base disturbances. Each "confidence" band represents the mean ± 2 SD for the compensatory response of normal subjects or patients to a given primary disorder. Ac, acute; Chr, chronic; Resp, respiratory; Met, metabolic; Alk, alkalosis; Acid, acidosis. (From Thorn GW, Adams RD, Braunwald E, et al [eds]: Harrison's Principles of Internal Medicine. 8th ed. New York, McGraw-Hill, Inc, 1977, p 377. Reproduced by permission.) (See Appendix for more detail.)

one's treatment decisions on the numbers obtained. Not uncommonly, one may accidentally puncture a vein when attempting to obtain an arterial blood sample. Furthermore, false readings may result if the sample is not free of air bubbles, not promptly chilled, and not analyzed within 20 to 30 minutes. Although still controversial, blood gas values that are *uncorrected* for body temperature appear more appropriate for guiding therapy in hypothermic patients. [69] [70]

An indwelling arterial catheter also provides continuous blood pressure monitoring. The trend of a patient's pressure helps one assess the effect of various therapeutic interventions. The absolute systolic and diastolic pressures measured will vary at different catheter sites, with higher peak systolic pressures measured at the periphery; the pressures will also be higher when measured in the distal lower limb. [12] [48] A wide variance between direct arterial pressure and pressure measured with a standard pneumatic cuff will always exist in some patients. Data averaged over a population group, however, compare fairly well. [12] For this reason, the cuff pressure and that displayed on the monitor should be compared regularly. A change in their relationship may be the first indication of difficulties with the direct measuring system. Auscultatory methods usually give a slightly lower value than direct measuring systems.

Waveform analysis may also provide an early indication of thrombosis in the arterial catheter. Many variables affect the waveform, including cardiac valvular disease, arteriosclerosis, and other peculiarities of an individual's cardiovascular system that may contribute to pulse wave reflections. [71] Waveforms may vary tremendously among patients, but after an adequate monitoring system has been established, a change in an individual's pressure wave is usually indicative of thrombosis or other malfunction in the monitoring system. A change in waveform may also indicate a change in the patient's cardiovascular status, such as a papillary muscle rupture. Once again, before making a

therapeutic decision based on an electronically generated number, the patient should be rechecked with a pneumatic cuff; this device is less fallible than the electromechanical system.

## CONCLUSION

As intensive care knowledge and technology grow and develop, cannulation of the arterial system may become a more routine procedure. Nonetheless, devices have been developed that in some cases may decrease the frequency with which this procedure is performed. Oximeters can determine the quality of oxygenation of the blood percutaneously and are becoming more accurate and sophisticated (see Chapter 6) . Electronic sphygmomanometers are being refined for continuous indirect blood pressure monitoring. However, these devices will not soon replace the indwelling arterial cannula, because of the need for frequent blood sampling for chemical and hematologic analysis.

Arterial puncture and cannulation are invaluable aids to the emergency and critical care physician. Long-term catheterization is a safe procedure when the catheter is placed, maintained, and removed with care. The radial artery is the most favored location for puncture, but as more experience is gained and reported with femoral artery catheterization, the latter may become a more frequently used site. Selection of either site is associated with a low complication rate and should be determined by the skill of the physician and the nursing team and the relative convenience and comfort of the patient.

# Chapter 20 - Peripheral Intravenous Access

**Edith L. Hambrick, Georges C. Benjamin**

Peripheral IV catheterization is one of the mainstays of modern medicine. It is a common procedure done by all levels of health care professionals, including physicians, nurses, physicians' assistants, nurse practitioners, and emergency medical technicians. The procedure allows access to the peripheral circulation for blood sampling, fluid and nutrition administration, and administration of medications. Generally peripheral IV catheterization can be accomplished in adults and children in less than 5 minutes if the patient has adequate peripheral circulation and normal veins. [1] [2] [3]

Although bloodletting has been described since the time of Hippocrates around 460 B.C., [4] one of the first documented descriptions of IV therapy, in the form of a blood transfusion, was made by an Italian physician, Giovanni Francisco Colle, in 1628. [5] In 1654, Francesco Folli was said to have used a combination of a silver tube, bone cannula, and animal blood vessel to accomplish a blood transfusion. [4] In 1662, Johann Daniel Major, a German physician, is credited with being the first to inject a medicinal substance into the vein of a human successfully. [6]

Initially steel needles were used for catheterization; however, in the early 1950s, plastic catheters were introduced for continuous IV infusions and over time have largely replaced those made of steel.

## INDICATIONS AND CONTRAINDICATIONS

Peripheral IV cannulation is performed to provide access to the patient's circulatory system. This access can be used for

phlebotomy, delivery of medication and fluids, and short-term nutrition administration. Peripheral lines are also placed in patients who have potentially life-threatening conditions as a means of ensuring adequate venous access should a problem develop. [9]

Establishment of IV access is essential during cardiac arrest and major trauma. The role of high-volume resuscitation before hemorrhage is controlled and definitive care is available remains controversial (see Chapter 23) . [10] In patients with normal perfusion, delivery times to the central circulation with peripheral venous access are similar to those attained with central venous access. [11] During closed-chest cardiopulmonary resuscitation (CPR), medication is delivered to the left ventricle more than a minute faster with central venous lines than with peripheral venous access. The former is the preferred route, when available. [12]

Short-term nutrition and hyperalimentation, primarily intralipids, can be administered peripherally. However, all other hyperalimentation must be given through a central IV

line because of the high tonicity of the solution.

The use of a *saline lock* catheter system (formerly termed *heparin lock* because of its use with a heparin solution) is preferable when access to the circulation is needed for medication delivery and limited IV fluid administration is anticipated. This is especially useful in cases of cardiac, renal, or hepatic failure. It has been recommended by one group for use in the out-of-hospital setting in lieu of the traditional continuous IV infusion when prophylactic access or access only for administration of bolus medications is desired. [13]

Because of the potential for fluid extravasation or inadequate volume flow, peripheral IV lines should not be started in an extremity in which massive edema, burns, traumatic injury, sclerosis, phlebitis, or thrombosis are present. In addition, the ipsilateral arm of a patient who has undergone a mastectomy or who has neck trauma or an indwelling fistula also should be avoided, if possible. In patients with chest, abdominal, or proximal extremity trauma, the vein selected for an IV line should not empty into the affected area, because the integrity of the proximal veins cannot be ensured. For example, it is preferable for patients with gunshot wounds to the abdomen to have IV lines started in their upper extremities, neck, or chest, rather than in the lower extremities.

The potential risk of inoculating the central circulation with bacteria is increased when an IV line is started in an area in which cellulitis is present, and therefore local cutaneous infection is a contraindication. Patients who have renal shunts or fistulas should have IV lines placed in the opposite extremity because of an increased risk for infection or thrombosis of the shunt or fistula. The extremity with a shunt or fistula can be used by an experienced practitioner in an emergency situation if extreme aseptic precautions are used and arterial delivery of the solution is not contraindicated.

## ANATOMY

In the upper extremity, on the dorsal surface of the hand, the veins collect blood from the dorsal and oblique communicating veins of the fingers, which form the dorsal plexus (see Fig. 22-2) . These in turn drain into the superficial radial and ulnar veins on the lateral and medial sides of the forearm, respectively. The cephalic vein is located on the radial border of the forearm and receives branches from the small veins in the forearm. The cephalic vein ascends the arm, gives off the median cephalic vein, and empties into the axillary vein.

The basilic vein collects blood from the veins of the ulnar portion of the posterior forearm. It then joins the median cubital vein and ascends on the medial portion of the arm. The axillary vein begins at the lower border of the teres major muscle as a continuation of the basilic vein and ends at the outer border of the first rib, where it becomes the subclavian vein. The subclavian vein unites with the internal jugular to form the brachiocephalic (innominate) vein. The right and left brachiocephalic veins join to form the superior vena cava, which empties into the right atrium of the heart.

In the lower extremity, on the dorsal surface of the foot, the dorsal digital veins receive blood from the plantar veins and then join with the dorsal metatarsal veins to form the

dorsal venous arch. On the sole of the foot, superficial veins form the plantar cutaneous arch, which drains into the medial and lateral marginal veins. The medial marginal vein drains into the great (long) saphenous vein at the ankle, which, as it ascends, collects tributaries from superficial and deep veins and ends at the femoral vein in the proximal thigh (see Fig. 22-1) . The plantar digital veins drain into the plantar metatarsal veins, forming the deep plantar venous arch.

The arch then drains into the medial and lateral plantar veins, which receive tributaries from the great and small saphenous veins to form the posterior tibial vein. The posterior and anterior tibial veins unite and form the popliteal vein. The femoral vein is a continuation of the popliteal vein. After passing behind the inguinal ligament, the femoral vein becomes the external iliac vein. The internal iliac, which arises from a confluence of veins draining the pelvis, joins with the external iliac to form the common iliac. The inferior vena cava is formed by the union of the common iliac veins and in turn empties into the right atrium of the heart.

The reader is referred to Chapters 22 through 24 for additional discussion on anatomy.

## EQUIPMENT

The necessary items for achieving IV access are shown in Table 20-1 . Povidone-iodine, 70% alcohol, or both should be used to clean the skin. A tourniquet is necessary to occlude the venous blood flow. A flexible rubber tube, a

| **TABLE 20-1** -- Materials Required for Peripheral IV Line Insertion |
| --- |
| Povidone-iodine swabs |
| 70% alcohol pads |
| Tourniquet or blood pressure cuff |
| Gauze sponges |
| Arm board |

| |
|---|
| Tape |
| Antibiotic ointment or film barrier |
| Intravenous catheter |
| 1-inch tape |
| ¼-inch tape |
| Latex gloves |

commercial rubber tourniquet with Velcro, or a sphygmomanometer inflated to 10 to 15 mm Hg below the systolic blood pressure serves this purpose. Sterile gauze pads are used to wipe away excess povidone-iodine or alcohol and to stop any bleeding from the phlebotomy site after withdrawal of a catheter. An arm board may be necessary if the line is placed over a joint or requires stabilization. Additional items such as tape, topical antibiotics, and appropriate dressings are also required.

Several catheter types are commonly used to gain peripheral venous access: a hollow steel needle, a plastic catheter over a hollow steel needle, a plastic catheter inserted through a hollow steel needle, or a plastic catheter over a flexible metal guide wire. [9] The catheter over the needle is the most common type currently used for peripheral access. Multilumen catheters (e.g, Twin-Cath, Arrow International, Reading, Pa) are useful when multiple medicines must be administered simultaneously.

## PREPARATION

To allay patient fears, reduce apprehension, and correct misinformation, the purpose, methods, and outcome of the procedure should be explained clearly at the onset by the practitioner when the patient is conscious and time permits. Before initiating IV lines, all of the items necessary for performing the procedure should be assembled and placed in convenient proximity to the practitioner. This is especially important if the practitioner is performing the procedure alone. As with all invasive procedures, universal blood

precautions should be taken, which includes wearing latex gloves, at a minimum. [14] If the propensity for blood splatter is high (e.g., in an uncooperative or delirious patient), a gown, face mask with eyeshield, and other protective equipment should be donned as needed (see Chapter 75) .

## Site Selection

Straight, large, bifurcated, easily accessible peripheral veins with healthy subcutaneous (SQ) tissue are ideal for cannulation (Fig. 20-1 *A*). The veins of the upper extremity are generally chosen to initiate IV lines because many potential sites are available and because the patient will be more comfortable. The most distal vein should be selected first if several sites are available. This strategy allows the practitioner to use a more proximal site in the event that the initial attempt is unsuccessful and avoids proximal fluid leakage from a previous puncture wound during fluid infusion. The veins of the hand and the volar aspect of the forearm are the preferred sites. The veins of the antecubital space or other upper extremity joints, although popular, are best avoided as a primary site because of vein tortuousity and the need to immobilize the patient's arm to prevent kinking of the catheter. The foot and lower leg are acceptable secondary sites. The external jugular vein also may serve as a site for venous access, particularly in infants and adults without suitable arm veins. This site may negate the need for central venous catheterization in IV drug users (Fig. 20-1 *B*). A percutaneous approach to the brachial vein has been described and is useful in patients who lack other peripheral sites. [15] Evidence in dogs suggests that 90% of peripheral IV fluid reaches the central circulation, even when

**Figure 20-1** *A,* Prominent veins on the dorsum of the hand. *B,* The external jugular vein may be cannulated in the same manner as any other peripheral vein, which often negates the need for central venous catheterization. This site is especially useful in obese adults, infants, and IV drug users. The major disadvantages are that flow is very dependent on the position of the neck (a significant problem in children and obtunded or restless adults), and valves may abut against the tip of the catheter. Air embolism from a disconnected catheter is another potential hazard with this access route.

it is injected under an inflated pneumatic antishock garment (PASG). [16] However, the injected fluid must be infused under a pressure greater than that in the PASG.

## Intravenous Line Assembly

IV fluid, an IV tubing set, and a primary administration set are selected appropriately for the clinical situation. A standard nonshock resuscitation set is shown in Figure 20-2 . If medication is being administered IV, a shorter secondary administration set is also needed (Fig. 20-3) . The cap and protective tabs should be removed from the container and drip chamber end of the IV tubing. After closing the regulating clamp, the spiked end of the tubing is then inserted fully into the receptacle on the solution container (Fig. 20-4) . The container is then turned over so that the tubing is dangling from below. The drip chamber should be pinched so that it fills halfway (Fig. 20-5) . The regulating clamp is

**Figure 20-2** Standard nonshock resuscitation set.

**Figure 20-3** Secondary administration set to "piggyback" medication into the main IV infusion.

**Figure 20-4** Insertion of the spiked end of tubing into an IV fluid bottle.

**Figure 20-5** Pinching of the drip chamber to fill the bulb halfway before starting the continuous infusion.

then opened, and fluid is allowed to fill the tubing completely, expelling all air bubbles. The bottle is then hung from an IV pole higher than the level of the insertion site.

## Placement of the Tourniquet

The tourniquet is placed 3 to 4 cm proximal to the site of the phlebotomy, taking care not to pinch the patient's skin (Fig. 20-6 *A*). It is applied so that arterial blood continues to flow into the extremity, but venous flow is occluded. Often the practitioner must start the IV line without assistance. Therefore, the flexible rubber tube should be tied so that it can be released with one hand as the practitioner steadies the catheter with the other (Fig. 20-6 *D*). The rubber tube is stretched and, with tension maintained (Fig. 20-6 *A*), crossed in front of the extremity, making an "X," and leaving one portion longer than the other (Fig. 20-6 *B*). [17] The middle of the longer end is tucked under the stretched tube, creating a loop and leaving the tip dangling free (Fig. 20-6 *C* and 20-6 *D*).

After application of the tourniquet, the veins may not become prominent, or the practitioner may not adequately feel the distended veins subcutaneously. Several explanations are possible. The tourniquet may be too tight, occluding both venous and arterial blood flow; the patient may have inadequate blood volume; or the veins may be sclerosed. To determine whether the tourniquet is too tight, the practitioner should feel the pulses distal to the tourniquet. If the pulse is absent, the tourniquet should be released and reapplied so that only the venous flow is occluded. If a sphygmomanometer is used, the cuff is inflated to 10 to 15 mm Hg less than the systolic pressure. A blood pressure cuff tourniquet has been shown to produce greater venous distention than an elastic tube tourniquet. [18]

The patient's veins should be checked again for sclerosis or thrombosis. If venous pathology is the cause of inadequate venous prominence, another site should be selected. If inadequate volume or shock is suspected as the cause of peripheral venous collapse, central venous lines, peripheral venous cutdown, or intraosseous infusion can be used until peripheral veins become more prominent.

## Adjuncts for Venous Dilation

The practitioner may still have difficulty locating a vein, even though the patient has a normal blood pressure, there is no previous exposure to irritating medications, and the tourniquet is applied properly. Other techniques for vein location can be used. A warm, moist towel can be applied to the site for several minutes, or an infrared light can be allowed to radiate over the site (not in contact with the skin) to cause vasodilation of the veins. [17] If this method is chosen, patients

**Figure 20-6** Application of a tourniquet. *A,* Placement of a tourniquet 3 to 4 cm proximal to the planned insertion site. *B,* Crossing of the ends of the tourniquet under tension. *C,* Tucking the longer end under the "X" to make a loop. *D,* One-handed technique for releasing the tourniquet.

should have normal skin and normal sensation to avoid burns. Tapping sharply over the vein can result in mechanical reflex dilation of the vascular walls, [17] but the tapping must be light enough to avoid reflex vasoconstriction from pain. Active or passive pumping of the extremity can also distend the veins by enhancing flow. Although rarely used, reactive hyperemia can be created by completely occluding the circulation with a sphygmomanometer for a few minutes and then releasing the sphygmomanometer to 10 to 15 mm Hg below the diastolic pressure. [17] This technique should not be used in patients at risk for peripheral vascular disease or coagulopathies.

Nitroglycerin ointment (0.4%) applied to the venipuncture site has been shown to facilitate venous dilation in children younger than 1 year of age. [19] In adults, a 2% nitroglycerin ointment increases the success rate of cannulation of small-caliber veins without significant side effects. [20] This is accomplished by placing ¼ inch of the ointment over the planned puncture site, spreading it over a 2.5 cm2 area for 2 minutes, and then removing the ointment before skin preparation.

A venous distention device has been used to augment venous filling in the nonemergency patient. [18] By using this device, peripheral venous cannulation can be achieved in 90% of patients who have difficult-to-access veins. [21] Complications with its use are minimal and include mild discomfort and petechiae. This device is still experimental and is not available for common use, and therefore, its role in the care of critical patients remains undefined.

**Site Preparation**

The skin should be cleaned with a povidone-iodine solution; a circular motion should be used, starting at the planned site of insertion and spiraling outward for 4 to 5 cm. After allowing this area to dry, 70% isopropyl alcohol is applied by using the same motion. Patients with iodine sensitivity can be cleaned with 70% alcohol alone if necessary, but this method may be less effective than that using povidone-iodine. [22]

Most catheters can be placed without anesthesia. When many punctures are

anticipated, large-bore catheters are placed, or patient anxiety is a factor, 0.5% or 1% lidocaine (Xylocaine), 0.1 mL placed intradermally using a 27-ga needle and 1-mL syringe, can be used at the puncture site to provide local anesthesia for approximately 20 minutes. [23] A more temporary anesthetic effect (3 minutes) can also be obtained by intradermal injection of 0.1 mL of sterile normal saline. [23] A solution of 0.9% benzyl alcohol and normal saline has been shown in one study to be superior to normal saline alone for this purpose. [24] In general, topical anesthetics are of little value because of poor penetration through the skin. However, eutectic mixture of local anesthetics (EMLA), a topical mixture of lidocaine and prilocaine base, may be an exception to this generalization; in children, the mixture seems to be well tolerated and effective. [25] Disadvantages to its use include the long application time (1 hour), the need for an occlusive dressing, and occasional local irritation, probably from the adhesive tape.

## CATHETER INSERTION

The skin should be grasped and *pulled taut to stabilize the vein* with the nondominant hand (Fig. 20-7 *A*). The IV catheter is held in the dominant hand, and the skin is rapidly punctured by a sterile catheter, bevel up, at a 30° angle and parallel to the vein. After entering the SQ space, the needle should be aligned in a parallel fashion with the surface of the extremity and along the long axis of the vein (Fig. 20-7 *B*). This will lessen the chances of perforating the back wall of the vein. A technique favored by many practitioners is to enter at the junction of two veins as a means of ensuring cannulation. Once into the skin, the catheter is advanced until a "pop" is felt and blood is seen in the flash chamber of the catheter or in the syringe (Fig. 20-7 *C*). The catheter is then advanced over the needle to the hub and the needle is removed (Fig. 20-7 *D*), with pressure placed on the proximal vein over the catheter tip. A syringe can then be connected to the catheter hub for phlebotomy before infusion (Fig. 20-7 *E*). The IV tubing is then attached to the hub of the needle or catheter (Fig. 20-7 *F*); the tourniquet is released; and after opening the regulating clamp, fluids are infused at the preselected rate.

Occasionally the catheter system will not advance because of small vessel diameter or a spasm of the vessel wall. The practitioner can remove the needle and connect the IV tubing. As the infusion is slowly started, the catheter can often be advanced slowly. This technique may be especially valuable in infants or in adults with limited available veins. This procedure is used only when partial cannulation is ensured and the solution is nonirritating.

If a heparin or saline lock is desired (Fig. 20-8) , the hub of the catheter is sealed with a catheter lock, and 1 mL (10 or 100 units/mL) of heparin sodium or saline is injected to fill the lock. Interestingly, 0.9% sodium chloride has been found to be as effective as heparin and does not carry the minor risks associated with heparin. [26] [27] These catheter locks are safe, cost-effective adjuncts to ensure that practitioners have access to the patient's circulation when fluid administration or heparin is not indicated. [28] If a venous infusion is required, the hub of the saline lock may be punctured with an 18-ga needle.

In many institutions, "needleless" intermittent infusion devices, which use tubing and

replaceable caps, have replaced saline locks, which must be punctured with a needle. These devices are believed to decrease the chances of accidental needle sticks by medical personnel. Before inserting the IV catheter, the intermittent infusion device is flushed with saline, leaving 2 to 3 mL of saline in the attached syringe. The procedure for insertion of the IV catheter is the same. After the catheter is inserted and blood drawn, if indicated, the device is connected with a twisting motion until secure and is then flushed with the remaining saline. Then the syringe is disconnected, and a plastic cap is affixed using a twisting motion. A sliding clamp on the tubing is then pushed to occlude blood backflow or air embolism if the cap is removed for any reason. A major disadvantage to these infusion devices is that when using commercially prefilled syringes that have needles attached (e.g., for administration of cardiac resuscitation drugs), there is no rubber port to puncture so that medications can be injected. However, there are some devices that have injection ports and plastic caps, and these should be used in the emergency setting whenever possible.

After the indwelling catheter has been placed, an antibiotic ointment can be applied at the site of skin penetration (e.g., povidone-iodine or a multiple-antibiotic ointment such as polymyxin B-neomycin-bacitracin [Neosporin] or polymixin B-bacitracin [Polysporin]). An adhesive bandage or other sterile dressing (gauze pads or transparent polyurethane) is commonly placed over the puncture site, and the hub of the catheter and the IV tubing are then taped securely. Alternatively, a piece of ¼-inch tape, with the sticky side facing upward, may be placed under the hub and crossed in front of it, making an "X" (Fig. 20-9) . Clear polyurethane dressings allow direct examination of the site for signs of complications, such as thrombophlebitis and extravasation of IV fluid, and are used in many centers instead of tape (Fig. 20-10) . However, there is some evidence that plastic dressing has a higher incidence of phlebitis than gauze dressing. [29] After making a loop, the tubing should be taped down. This loop helps prevent the tubing or catheter from being dislodged accidentally if it is pulled. In many centers the dressing is dated, and the type and size of the catheter are recorded along with the practitioner's initials. As an additional measure, a short piece of stockinette or elastic netting can be applied over the insertion site and distal tubing to help prevent dislodgment.

**Percutaneous Brachial Vein Cannulation**

An alternative technique is percutaneous brachial vein cannulation. This technique is recommended for patients who

**Figure 20-8** Application of a saline lock. This was formerly called a *heparin lock,* but heparin is not required to keep the vein open; plain 0.9% saline will suffice as a flush. To convert this to an infusion site, puncture the rubber hub with an 18-ga needle. Blood may be drawn from the lock before beginning an infusion or flushing. If the lock contains saline, discard 3 to 4 mL of blood and wait 1 minute before collecting the sample.

**Figure 20-9** Securing the IV tubing. *A,* Placing the tape under the hub of the catheter, sticky side up. *B,*

Crossing the ends of the tape over the hub, forming an "X."

do not have easily accessible peripheral IV veins and is successful in 70% of cases. [15] [30] In one study, complications included transitory paresthesias in 18%, brachial artery puncture in 8%, and hematomas in 1.6%. [30] All of these complications were without long-term effects.

The practitioner first locates the brachial artery in the antecubital fossa by palpation. A tourniquet is then applied to the upper arm, and the skin is prepared as noted earlier. The brachial artery is again located, and a puncture site lateral or medial to the pulse is selected. The optimal side of the artery for use varies with the individual. An IV catheter attached to a syringe is advanced at a 30° to 45° angle to the patient's arm while the practitioner maintains suction on the syringe. After penetration of the vessel, the assembly is advanced 2 to 3 mm to ensure cannulation. The catheter is then advanced into the vein and secured. [15]

### Seldinger Technique

Occasionally a smaller gauge IV catheter (e.g., 20-ga) must be exchanged for one that is larger. This exchange can be made using the Seldinger technique (see Chapter 21) . This procedure may be necessary when other venous access sites are unavailable or more rapid access is required using a larger bore catheter. Such a catheter exchange set is made by Arrow, Inc. (product No. RC-09850). The site is prepared by swabbing povidone-iodine solution over the puncture site, indwelling catheter, and IV connector assembly. The administration tubing is disconnected from the indwelling catheter, and the flexible end of the guide wire is inserted through the lumen of the catheter. The catheter is then removed over the guide wire. The cutaneous puncture site is enlarged with a No. 11 scalpel blade, and a larger catheter is placed over the guide wire. The dilator is removed if a sheath-dilator assembly is used, and the catheter is checked for free-flowing blood. After successful cannulation, the catheter is attached to the IV tubing and secured.

## DIAGNOSTIC PHLEBOTOMY

Often the health care provider is unable to perform a diagnostic phlebotomy from a site away from the patient's IV line. This occurs when the patient has limited veins, multiple IV lines, amputated extremities, or relative contraindications such as mastectomy and renal shunts. In these situations, the practitioner must draw blood from the same extremity in which the IV line is placed. Phlebotomy can be done while the IV line is infusing if it is done *well below the infusion site.* [31] [32] This will not result in dilution of most serum chemistries; however, the serum glucose will be elevated if a glucose-containing solution is infusing. [31] A tourniquet can be placed in between the IV and phlebotomy sites for venous distention. [33] Blood can also be drawn directly from

the IV catheter after stopping the infusion and waiting for 2 minutes. [33] [34]

Another technique for diagnostic phlebotomy directly from the IV line has been described by Isaacson and colleagues. [32] The IV line is clamped above the injection port for 1 minute, and a tourniquet is applied above the IV site. A 10-mL Vacutainer tube and needle is inserted into the injection port, and 5 mL of blood is collected and discarded (Fig. 20-11) (Figure Not Available) . Uncontaminated blood should now be available

**Figure 20-11** (Figure Not Available) Drawing venous blood from an IV line. Contrary to the illustration, latex gloves should be worn; see text for details. Venous blood may also be drawn from a saline lock (see Fig. 20-8 ). *(From Isaacson G, Mansfield PF, Kirkland ML: An atraumatic technique for diagnostic phlebotomy. N Engl J Med 313:1478, 1985. Reprinted by permission of the New England Journal of Medicine.)*

for collection using the Vacutainer system. Tapping the vein at the catheter tip has been described as a method to restore blood flow by breaking away the seal between the vein and the catheter if the flow decreases. After collection of blood, the tourniquet and clamp are removed, and the tubing is flushed with a few milliliters of saline. Glucose and potassium levels may be elevated significantly by this technique if glucose- or potassium-containing solutions are infusing. Also if the tube is significantly underfilled, this can lead to a pseudometabolic acidosis.

## COMPLICATIONS

Complications of IV therapy may be local or systemic (Table 20-2) . Local complications include extravasation of fluids with local swelling, hematoma, phlebitis, cellulitis, and thrombosis. Hematomas are usually caused when the posterior wall of the vein is punctured during cannulation or after the cannula is removed. Bruising can be minimized in the antecubital fossa after venipuncture by leaving the elbow extended and applying direct pressure over the venipuncture site instead of flexing the elbow. [36] If the vein is not properly cannulated and there is resultant unrecognized SQ placement of the catheter and IV fluids are begun, the fluids will infuse into the tissues, causing swelling and pain. To avoid this, the

| **TABLE 20-2** -- Complications from Peripheral IV Lines |
| --- |
| Air embolism |
| Catheter embolism |

| |
|---|
| Cellulitis |
| Hematoma |
| Infiltration, possibly tissue necrosis |
| Phlebitis |
| Sepsis |
| Thrombophlebitis |
| Thrombosis |
| Volume overload |

practitioner should be careful to observe for blood in the flash chamber on insertion; check the IV drip chamber for flow; and check the IV site periodically for swelling, pain, and tenderness.

Extravasation of irritants or medications can result in tissue destruction with resultant sloughing, thrombophlebitis, cellulitis, or venous thrombosis. If extravasation occurs, the cannula must be removed and replaced at another site as appropriate. [37] Significant skin sloughing that occasionally requires skin grafting may occur with the extravasation of certain medications, such as hypertonic solutions, vasopressors, and chemotherapy agents. Occasionally an "antidote" is available, such as in the instance of the extravasation of vasopressors, in which the resulting injury may be ameliorated by local SQ injection of phentolamine (Regitine) (Tables 20-3 (Table Not Available) , 20-4 (Table Not Available) , and 20-5) .

Phlebitis may occur in up to 75% of hospitalized patients. [27] The risk of phlebitis is

increased by infusion of irritant solutions, mechanical irritation from the catheter, infection, prolonged placement, catheter type (steel carries more risk than plastic), and large catheter size. [37] [38] [39] It is generally agreed that catheters should not be left in place for longer than 3 days because of the risk of a mechanical phlebitis. [1] [38] To identify infection or phlebitis, the insertion site should be checked daily for erythema, tenderness, and swelling. If inflammation is found, the IV catheter should be removed and a new catheter placed in another site. To avoid phlebitis or thrombosis from irritant medications, the medications should be dissolved in a large volume of fluid or administered through a central IV line.

Heparin and steroids may reduce the incidence of thrombophlebitis and may be useful in appropriate settings. [26] [40] [41] Buffering of acidic infusions with sodium bicarbonate may also reduce the incidence of phlebitis. [41] [42] Peripheral IV lines placed in the out-of-hospital care setting should be replaced when possible because of the high incidence of phlebitis when compared with the incidence associated with IV lines placed in the emergency department. [1] [43] [44] Therapy for noninfectious phlebitis includes moist heat, elevation of the extremity, and anti-inflammatory agents.

---

**TABLE 20-3** -- Suggested Antidotes for Drugs Causing Extravasation Injury *
*Adapted from MacCara ME: Extravasation: A hazard of intravenous therapy. Drug Intell Clin Pharm 17:713, 1983.*

(Not Available)

---

* Antidote administered either via the offending IV cannula or via multiple subcutaneous (SQ) injections with the catheter removed.

Occasionally patients present to the emergency department a few days after hospital discharge with the complaint of a hard knot or linear mass under the skin at the site of an IV infusion. A firm cord may be palpated, but the site should be neither tender nor red. Patients may believe that part of the IV catheter has broken off and remains under the skin. This is rarely the case. Most often the cord represents a venous thrombosis from irritating IV fluids and is generally a benign process. The patient should be reassured that although a blood clot may be present, it is not associated with pulmonary embolism. If a foreign body or infection can be excluded clinically, no intervention is required, and the condition will gradually resolve (over weeks) without significant sequelae, although the induration may never fully resolve in the vein. Radiographs are not indicated unless there is a history or examination that suggests an infectious process. The latter condition ( *suppurative phlebitis*) is generally associated with fever, local inflammation, and marked tenderness. The condition requires an aggressive interventional approach as outlined below.

Thrombosis can occur if IV fluids are allowed to run out or the saline lock is not flushed periodically. Infiltrated IV lines should not be irrigated because of the risk of central

embolism. [45] If infiltration occurs, a 3-mL syringe is connected to the cannula using aseptic technique and gentle aspiration is performed. If the return is bloody, discard the return and flush the catheter gently with 3 mL of sterile saline. The infusion is then resumed. If the return is not bloody, flush the catheter gently, as noted earlier. If resistance

| TABLE 20-5 -- Other Drugs Known to Cause Tissue Damage When Extravasated |
| --- |
| Hyperalimentation solutions |
| Renografin-60 |
| Sodium thiopental |
| Phenytoin |
| Nafcillin |
| Tetracycline |
| Propylene glycol |
| Ethyl alcohol |
| Nitroglycerine |
| Chlordiazepoxide |

| |
|---|
| Diazepam |
| Colchicine |

is felt, the procedure is discontinued, and the catheter should be placed at another site. [46]

Nerve, tendon, or arterial damage may occur if these structures are directly punctured by the needle. [47] [48] [49] In addition, large hematomas may cause nerve damage and arterial insufficiency indirectly by compressing those structures and interrupting the blood supply.

*Suppurative phlebitis* is a rare complication and can result in sepsis and death. [50] This complication is suspected when phlebitis is associated with fever or purulent drainage. [50] Catheter-related infections of this type can be caused by the absence of aseptic skin preparation before cannula insertion, contaminated IV equipment, bacterial colonization, phlebitis, or contaminated infusate. [39] When these infections occur, any purulent drainage should be Gram stained and cultured, and the catheter tip should be sent to the laboratory to be cultured. Treatment includes removal of the catheter, administration of IV antibiotics and moist heat, elevation of the extremity, and venectomy.

Systemic complications include sepsis or embolism due to catheter, air, or clot. Bacteremia and sepsis can occur in 7% of patients with plastic catheters. [51] These infections are generally caused by common organisms such as *Staphylococcus aureus* and *Staphylococcus epidermidis.* [52] [53] As with noninfectious phlebitis, the incidence of catheter colonization and bacteremia increases significantly after 3 days. Hand washing and the use of aseptic technique on insertion (or system manipulation) and changing the IV sites every 3 days generally prevents this complication. Topical antibiotics such as povidone-iodine should be applied to the insertion site and may decrease the incidence of infection. [54] [55] [56] Despite earlier claims, frequent dressing changes are not necessary to prevent infection. In all cases, the site should be checked at least every 48 hours, and if infection or phlebitis is present, the catheter should be removed and placed at a new site. [56] [57]

Air embolism is a rare complication that occurs when air is introduced into the patient's bloodstream through the catheter, either through a syringe when improperly used to draw blood or more commonly through IV tubing when the container with IV fluids has been allowed to empty. Air embolism through an IV line is most common when fluids are delivered under pressure. Prevention is the key and is best done by ensuring the absence of air in the IV lines, keeping the IV container vertical during patient transfers, not allowing the IV containers to run dry, filling the drip chamber halfway before moving the patient, and paying careful attention to technique when changing tubing or

containers.

If air becomes trapped in the IV line, the least invasive method should be used to remove it. Boykoff and colleagues have described six ways to remove air from an IV line. [58] If the air is near the top of the IV line, it can be removed by holding the air-filled tubing taut and tapping on it with the finger. The air bubbles should rise into the drip chamber. Another technique is to curl a section of the tubing below the bubbles around a pen or syringe. The pen is then rolled upward toward the drip chamber, removing the bubbles.

If the bubbles are in the upper section of tubing, the bag can be lowered and the roller clamp opened to allow backflow of blood into the tubing. This may push the air back into the drip chamber. The Y connector can be used as a port from which to aspirate and is especially useful to

remove air found directly above the connector. To accomplish this, clamp the tubing below both the Y connector and the air. Clean the Y-connector port with 70% alcohol, insert a 10-mL syringe with a small-gauge needle into the port, and release the roller clamp. Aspirate the fluid and air into the syringe. The syringe is then removed, followed by the clamp. Using a similar technique, one can inject a sterile solution into the Y connector, pushing the bubbles into the drip chamber. This technique is especially useful if the drip chamber has collapsed. Finally, the tubing can be disconnected and flushed by opening up the rolling clamp. The IV tubing can be reattached directly to the catheter, or a heparin lock can be attached to the catheter and the IV tubing reattached using a sterile needle.

The use of in-line filters to prevent phlebitis is controversial. [59] [60] Filters are quite effective in removing particulate matter and reducing postinfusion phlebitis but are subject to filter clogging, air locking, and drug binding. [61] Drug binding is especially a problem when the amount of drug injected is small. In most cases, modern processing of parenteral materials, frequent changing of IV tubing and catheters, and the use of dilute solutions are effective in preventing phlebitis.

## CONCLUSIONS

The use of peripheral IV access, when performed correctly and appropriately, is easy and safe and may be life-saving, and it has few inherent disadvantages. It can be performed within minutes by any health care provider with minimal training.

### Acknowledgments

# Chapter 21 - Guide-Wire (Seldinger) Technique for Catheter Insertion

**Alfred D. Sacchetti**

The expedient placement of large-bore or central venous catheters can be a difficult task, especially in the unstable, critically ill patient. Frequently the physician is faced with the problem of being unable to locate a suitable vein or, more frustrating, being unable to cannulate a located vein. The guide-wire or Seldinger technique of catheter introduction is a valuable solution to vascular access problems in the emergency department.

The guide wire-through-the-needle technique was originally described in 1953 by Seldinger as a method for catheter placement in percutaneous arteriography. [1] The basic approach is simple and has been adapted for placement of devices in any hollow-lumen structure or body cavity. For vascular access, a small needle is used to enter the intended vessel. Once the introducing needle is positioned within the vessel lumen, a wire is threaded through the needle, and the needle is removed. The wire, which is now within the vessel, serves as a guide over which the selected catheter is placed.

## INDICATIONS AND CONTRAINDICATIONS

The primary advantage to the Seldinger technique of vascular access is that a relatively small needle can be used to place nearly any size or shape of catheter into a vessel. Table 21-1 lists the various sizes of vascular catheters along with the sizes of their introducer needles. A large 8.5 Fr high-flow infusion catheter or introducer sheath can be placed through a 22-ga puncture. This flexibility is of great value when attempting to place catheters in deep vessels, such as central veins. Subclavian, internal jugular, external jugular, brachial, femoral, and peripheral veins and arteries can all be cannulated using this technique. [2] [3] The use of the smaller introducer needle increases the probability of cannulating a hidden or collapsed vessel while theoretically reducing resultant injury should an adjacent structure, such as an artery or lung, be punctured.

Guide-wire approaches also allow for the rapid percutaneous placement of many catheters that might otherwise require a surgical procedure. A number of large-bore infusion catheters can be placed within the central circulation without the need for cutdowns of the femoral, saphenous, or brachial veins. [4] [5] In addition, this technique can be used to quickly convert small peripheral intravenous (IV) catheters to larger flow systems without the need for recannulation. [6]

Modifications of the guide-wire approach have expanded the indications for this technique beyond those of vascular access. Guide-wire systems exist now for almost every catheter system used in medicine. Table 21-2 presents a list of available guide-wire systems relevant to emergency medicine along with some of their manufacturers. The guide-wire technique is applicable to pediatric patients as well as

adults, and many commercial products are now available for use in children. The technique for placement is the same in children, only the size, and occasionally the composition, of the catheters differs.

The technique should not be used when large-bore catheter placement is not needed or may exacerbate hemorrhage. In general, patients receiving thrombolytic agents should not receive large-bore catheters.

| TABLE 21-1 -- Needle Sizes for Venous and Arterial Catheters [*] | |
|---|---|
| **Standard Full-Length Coil Guide Wire Catheter Size (Fr)** | **Needle Gauge** |
| 3 | 21 |
| 4.0-4.5 | 20 |
| 5.0-6.0 | 20-19 |
| 6.0-8.5 | 19-18 |

* Any sized catheter from 3.0 to 8.5 Fr may be introduced using a 22-ga needle if a solid wire (Cor-Flex, Cook Critical Care) is used.
All needle gauges are for thin-walled needles only.

| TABLE 21-2 -- Emergency Department Catheters and Devices Placed with Guide Wire Technique [*] |
|---|
| Single-lumen central venous catheters (1,2,3,6) |

| |
|---|
| Multilumen central venous catheters (1,2,3,6) |
| Arterial line catheters (1,2,3,5,6) |
| Sheath introducers (e.g., for pacemaker insertion) (1,2,4,5) |
| Large-bore infusion catheters (1,2,4,5) |
| Tracheostomy sets (1) |
| Peritoneal lavage catheters (1,2) |
| Tube thoracostomy (chest tubes) (1) |
| Pneumothorax aspiration catheters (1) |
| Cricothyrotomy sets (1) |
| Cystostomy sets (1) |

\* Included is only a partial list of manufacturers; other companies may produce similar items. Manufacturers: (1) Cook Critical Care; (2) Arrow International; (3) Burron Medical, Inc.; (4) Bard, Inc.; (5) Cordis Corp.; and (6) Deseret

## EQUIPMENT

The equipment needed for the guide-wire technique is listed in Table 21-3 . All of these items, with the exception of the needle, the wire, and the catheter, are part of the standard emergency department stock. The syringe in these systems should be a

non-Luer-Lok or slip tip type. The added twisting that is required to remove a Luer-Lok syringe from the introducer needle may actually dislodge a needle tenuously placed in a vein. Systems now exist in which the needle is modified to permit passage of the wire without removal of the aspirating syringe.

**Needle**

Virtually any needle or catheter can be used to introduce a guide wire into a vessel. The needle must be large enough to accommodate the desired wire. The needles contained in prepared sets are usually thin walled, and a smaller gauge can accommodate a larger wire. If a needle that is not thin walled is chosen, a size that is 1 ga smaller (larger bore) than that listed in Table 21-1 should be used. In standard peripheral IV systems that use a catheter-over-the-needle design, an 18-ga or larger-bore catheter will generally accept the standard guide wire that fits an 18-ga thin-walled needle.

The taper of the lumen at the proximal end of a specifically designed Seldinger needle is also different. Seldinger needles have a funnel-shaped taper that guides the wire directly into the needle (Fig. 21-1) . Ordinary needles may have a straight-bore lumen that leads squarely into the needle.

| TABLE 21-3 -- Necessary Equipment for Seldinger Technique | |
|---|---|
| Introducing needle | No. 11 scalpel |
| Guide wire | Lidocaine |
| Catheter or sheath introducer | 3-mL syringe (for anesthetizing) |
| Prep solution (iodine) | 3- to 5-mL syringe ( *not* Luer-Lok) |
| Sterile gloves | Suture (0-00) |

| | |
|---|---|
| Small anesthetizing needle (1.5 in., 25 ga) | Sterile drapes |
| | Antibiotic ointment |
| Gauze pads | |
| Prep razor | |

These needles can present a problem if the wire abuts the flat end-plate surface.

**Guide Wire**

Two basic types of guide wires are used: straight or J-shaped. The straight wires are for use in vessels with a linear configuration, whereas the J wires are for use in tortuous vessels. Both wires have essentially the same internal design (Fig. 21-2 (Figure Not Available) *A*). The flexibility of the wire is a result of a stainless steel coil or helix that forms the bulk of the guide wire. Within the central lumen of the helix is a straight central core wire, called a mandrel. The mandrel is fixed at one end of the helix and terminates between 0.5 and 3.0 cm from the other end. It adds rigidity to the portion of the wire surrounding it, whereas the remainder of the wire without the mandrel is the flexible or floppy end. Many guide wires also contain a straight safety wire that runs parallel to the mandrel to keep the wire from kinking or shearing.

Wires with two flexible ends contain the mandrel only in the central portion of the wire. In these systems one end is usually straight and the other end is J shaped.

The most common size for these wires is from 0.025 to 0.035 in. (0.064 to 0.089 cm) in diameter. Wire in this size range is generally introduced through an 18-ga thin-walled needle. A modification of this standard wire uses a bare mandrel with the flexible coil soldered to its end. This construction provides a wire with a diameter of only 0.018 in. (0.047 cm) but with the same rigidity as the larger wires. The manufacturer states that such a wire can be introduced through a 22-gauge thin-walled needle, yet still guide an 8.5 Fr catheter (Cook Critical Care Inc., Micropuncture Introducer Sets and Trays with Cor-Flex Wire Guides, Bloomington, Ind).

Wires may be coated with Teflon to reduce resistance or with heparin to inhibit clotting on wires left in place for extended periods.

The flexible end of the guide wire allows the wire to flex on contact with the wall of a vessel. If the contact is tangential, as in an infraclavicular approach to the subclavian vein, a straight wire is generally preferred. If the angle is more acute, as in an external jugular approach to the subclavian vein, or if the vessel is particularly tortuous or valves

must be traversed, a J-shaped wire is used. The more rounded leading edge of the J wire provides a broader surface to manipulate within the vessel and decreases the risk of perforation. This is especially advantageous when attempting to thread a wire through a vessel with valves.

It is important to emphasize that guide wires are delicate and may bend, kink, or unwind. A force of 4 to 6 lb may cause rupture of a wire. Embolization of portions of the guide wire is possible, and sharp defects in the wire may perforate vessel walls (see Fig. 21-2) (Figure Not Available) . One should carefully inspect wires for defects such as kinks, sharp ends, or spurs before use. [7] *Wires should thread easily and smoothly and should never be forced*

## Catheters

Once the guide wire is in place and the needle is removed, any number of catheter devices may be introduced. Single-, double-, and triple-lumen catheters are generally placed by sliding the catheter directly over the guide wire into the intended vessel. Larger catheters or non-lumen devices are generally introduced with a sheath-introducer system.

The Desilets-Hoffman type sheath introducer was designed by Desilets and Hoffman in 1965 to aid in arteriography procedures that required many catheter changes. [8] The sheath-introducer unit consists basically of two catheters, one inside the other (Fig. 21-3) . The first catheter, termed the *dilator*, is rigid; its inner lumen fits over the guide wire. This catheter is longer and thinner than its sheath and has a tapered end that serves as a dilator when the unit is passed through subcutaneous tissue and into the vessel. The dilator serves no other function than to facilitate placement of the sheath. The second catheter, termed the *sheath* (or *introducer* catheter when used as a cannula for introducing Swan-Ganz catheters, transvenous pacemakers, or other devices), has a

blunt end and is simply a catheter with a large diameter.

Many modifications of the sheath exist, with side arms and diaphragms to aid in placement of non-lumen devices. Care must be taken in the use of side-arm sets for rapid fluid administration, because some catheters may be 8.5 Fr in diameter but may have only a 5 Fr side arm. If faced with this problem, one can either remove the diaphragm or introduce an 8 Fr feeding tube through the diaphragm at the catheter hub for rapid fluid administration.

## PROCEDURE

The actual procedure for placement of Seldinger-type guide-wire catheters is quite simple. The vessel is first cannulated with a needle or an indwelling IV catheter. A guide wire is threaded through the needle or catheter and the needle or catheter is removed, leaving only the wire within the vessel. A catheter or introducer-sheath unit (often with a dilator) is passed over the guide wire through the skin and into the vessel. Once the introducer-sheath unit device is in place, the guide wire and dilator are removed. These steps are detailed in the following discussion.

**Guide-Wire Placement**

Sterile technique is maintained, bloodborne pathogen protection is taken, and the area of vascular access is prepared and cleaned.

An introducing needle or a standard over-the-needle catheter that is large enough to accommodate the guide wire is selected. The needle is attached to a small syringe (Fig. 21-4) . The needle and syringe are introduced together, and the selected vessel is cannulated. Once a free return of blood is obtained, the syringe is removed, and the needle is stabilized as it enters the skin to prevent displacement from the vessel. When cannulating a central vein, the needle hub is capped with a thumb before passing the guide wire; this minimizes the potential for air embolism. Next, the flexible end of the guide wire is threaded through the needle. If an IV catheter is already in place, the wire is simply passed through it and the catheter is removed.

The straight wire is easily introduced by threading the flexible end of the wire into the proximal hub of the indwelling needle. To introduce the J wire, a plastic sleeve contained in the kit is advanced to the floppy end of the wire to straighten out the J shape. This straightened end is then introduced into the proximal hub of the indwelling needle. Once the J wire has been advanced, the sleeve is removed

**Figure 21-3** Desilets-Hoffman sheath introducer. *A*, Guide wire. *B*, Sheath-introducer. *C*, Dilator. *D*, Assembled device.

and discarded (Fig. 21-5) . *Caution:* In an emergency this sleeve may be misplaced, making insertion of the J wire very difficult.

The wire should thread smoothly into the vein. If resistance is met, the wire should not be forced, but it should be removed from the needle and the syringe reattached to confirm intravascular placement. It is extremely important for the wire to slip easily from the needle during removal. If any resistance to removal of the wire is felt, *the wire and the needle should be removed as a single unit* to prevent shearing off of the wire. Despite its necessity, this last maneuver has occasionally been disregarded with resultant wire embolism. It is not that removal of both the wire and needle as a unit is technically difficult; rather, there is often reluctance on the physician's part to abandon a needle already in a vein.

Manipulation of the wire within an introducer needle should be done only with the standard coil wire guides. Manipulation should be avoided when using the solid wire sets in which the coil is soldered onto the mandrel. In these sets, the taper of the mandrel to the wire creates a very small lip that can become caught on the edge of the needle tip and shear off the coil portion of the wire. These wires must thread freely on the first attempt or the entire wire and needle assembly must be removed.

Some physicians recommend applying these restrictions to all guide wires and believe that no wire should ever be withdrawn through the introducing needle. [9] One method of avoiding this problem is to cannulate the vessel with a catheter-over-the-needle system and then use the soft catheter, rather than a sharp needle, as the entry source for the guide wire. The limitation of this approach is that the catheter itself cannot be advanced to relocate a vessel if the initial attempt at wire passage fails.

Occasionally, a wire must be teased into the vessel; rotating the wire or needle often helps in difficult placements. If the wire does not thread easily, another helpful maneuver is to pull back slightly on the needle itself just before advancing the wire. This helps if the opening of the needle is abutting the vessel's inner wall, blocking the wire's entry, or the vein is compressed by introduction of the needle. Changing wire tips from a straight to a J wire or vice versa also may solve an advancement problem. If the inner lumen of a vessel is smaller than the diameter of the J, it will prevent the wire from reforming its natural shape, causing the spring in the coil to generate resistance. Any advantages of a J wire will be negated if the wire fails to regain its intended shape. In this instance, a straight tip should be introducible without problems. Alternately, if the angle of entry of the needle and the vessel is more acute than was suspected, the straight wire may not be able to bend appropriately as it encounters the vessel's far wall. A J-tipped wire may be used and threaded in such a manner that the wire resumes its J shape away from the far wall. All of these maneuvers are performed with gentle free motions of the wire within the needle. If at any time the wire cannot be advanced freely, improper placement must be suspected and the attempt reevaluated.

If it is threading easily, the guide wire should be advanced until at least one quarter of the wire is within the vessel. The further into the vessel the wire extends the more stable its location when the catheter is introduced. Occasionally a wire threads easily past the

tip of the needle and then suddenly does not advance any farther. If the introducer needle demonstrated free blood return at the time of wire entry and the initial advancement of the wire met no resistance, the wire is most likely located properly in the vessel and can serve as a guide for the catheter. If further confirmation is needed, the needle may be removed, the wire fixed in place with a sterile hemostat, and a radiograph taken to confirm the position of the wire. [10] This confirmation may be advisable if the location of a wire is suspect and the introduction of a large-sized sheath is planned. A freely advancing wire may suddenly stop once it is well within a vessel if the vessel makes an unsuspected bend or is being compressed or deviated by another structure, such as a rib or muscle.

**Introducer Sheath Unit Placement**

Once the wire is placed into the vessel, the needle is removed and the desired catheter passed (see Fig. 21-4) . A small skin incision is made at the site of the wire. The incision should be approximately the size of the catheter to be introduced and should extend completely through the dermis. Precisely placing this skin incision directly over the wire entry site may be difficult if a small aspirating needle was used. Alternately, some physicians prefer to make the skin nick first and to introduce the needle through this incision. The disadvantage of this approach is that if the vessel is not where it is expected, the physician is forced to attempt a difficult entry through the existing incision or to make a second incision.

Once the incision is made, the introducer sheath with the dilator is threaded over the wire to a point 1 cm from the surface of the skin. The guide wire should be stabilized at the point of skin entry by the clinician during advancement of the introducer unit over the guide wire. *The wire must protrude from the back end of the introducer before advancement of the catheter past the skin*. This is a critically important procedural point. If the wire is not protruding from the proximal end of the introducer, it may be lost in the vessel and migrate to the central circulation. If the wire is not visible at the proximal end of the introducer, the introducer unit is not advanced until the guide wire is carefully withdrawn at the site of skin entry to the extent that it exits from the proximal end of the introducer unit. The wire must always be visible and graspable from the back end of the introducer throughout the remainder of the procedure. Guide wires are always longer than their catheters, and with experience it becomes easy to judge the optimal distance to thread the wire to ensure good introducer placement as well as to leave enough wire protruding from the proximal end of the introducer unit.

Once the wire is visible, it is grasped and held. The introducer unit is threaded into the skin with a twisting motion until it is well within the vessel. When using a sheath and dilator, it is best to grasp the unit at the junction of the sheath and dilator. This prevents the thinner sheath from kinking or bending at the tip or from bunching up at the coupler end. If a rigid-walled sheath is being used, the dilator need only be advanced a few centimeters into the vessel and the sheath slid off and advanced to its hub. If a thin-walled sheath is used, the introducer-sheath unit is kept intact and advanced through the skin to the hub. This adds rigidity to the sheath and prevents it from kinking before being fully seated in the vessel. In placing right-sided subclavian catheters, it is sometimes helpful to make a banana-like bend in the dilator before introduction to

facilitate proper location in the superior vena cava. [11] Once the introducer unit is in place, the wire and dilator are removed together. Precautions are again taken to cover the sheath hub to avoid air embolism after removal of the wire and dilator before attachment of the infusion tubing or cap. Once in place, the device is usually affixed with 1 or 2 sutures, and infusion or monitoring is begun.

If a single-lumen catheter is used instead of a sheath-dilator, the catheter itself is passed over the wire to its desired depth and the wire is removed. When a soft catheter is used, a tract from the skin to the vessel must be created before the catheter can be introduced. This tract is created by passing and removing a separate dilator over the guide wire after the needle is removed but before the catheter is placed. After the dilator is removed the soft catheter is threaded into the position over the wire. Occasionally, it is easier to advance the soft catheter over the wire up to the skin edge and then to advance the catheter and wire together as a unit into the vessel. As always, it is imperative that the guide wire protrude from the back of the catheter and be firmly grasped when the wire and catheter are being advanced.

To place multiple lumen catheters, the distal lumen and hub are identified. The distal lumen is the hole at the tip of the catheter. At the proximal end of the device, the hub that corresponds to the distal lumen will be labeled "distal" by the manufacturer. If there is any confusion, a small amount of sterile saline can be injected through each hub until it is observed coming out of the hole at the tip of the catheter. Once the distal hub is identified, its cover cap is removed to allow the guide wire to pass through. The catheter is placed by threading the guide wire into the hole at the tip of the catheter and advancing it until it is visible and graspable at the hub end of the device. At this point the device is placed in the same manner as a single-lumen catheter. If a soft

multiple-lumen device is placed, a separate dilator is used to create a tract over the guide wire *prior* to placing the catheter. An alternate method of placing multiple lumen catheters is to thread the catheter through a standard Desilets-Hoffman sheath introducer system.

Any lumen in a multiple-lumen device that is not immediately used for an infusion must be flushed with a heparin solution. Saline solutions alone should not be used as flushes for central catheters. When removing the wire from a catheter it must slip out easily. If any resistance is met, both the wire and catheter must be removed as a single unit and the procedure reattempted. A common cause of a "stuck wire" is a small piece of adipose tissue wedged between the wire and the lumen of the catheter. This problem can be avoided by creating a deep enough skin nick and adequate dilation of the tract before insertion of the catheter.

**Replacement of Existing Catheters**

In addition to placing new catheters, the guide-wire technique can be used to change existing devices. The utility of this technique in converting a peripheral small-gauge catheter to a larger infusion catheter is obvious. Within a few seconds, an existing peripheral or central 18-ga IV catheter can be changed to an 8.5 Fr infuser. The guide-wire technique can also be used to change single-lumen central venous catheters

to triple-lumen or sheath-introducer sets. In these instances a guide wire longer than either of the devices to be exchanged is selected. It is inserted into the existing central venous catheter until approximately a few centimeters are protruding and are graspable from the proximal end. With one hand holding the wire securely, the catheter and wire are removed as a single unit until the tip of the catheter just clears the patient's skin. The wire is grasped at the point at which it exits the skin, and only then is the other end of the wire released. The catheter is then slid off the wire, and the new device is inserted in the normal fashion. Caution must be exercised with this technique because catheter embolization can occur if a catheter is cut to allow use of a shorter guide wire for the exchange. [9] In patients without evidence of sepsis, the technique of guide-wire exchange does not increase the incidence of catheter-related infections. However, in septic patients this maneuver does appear to increase infections related to central catheters. [12]

**Modifications of Guide-Wire Catheter Systems**

Modifications of the basic guide-wire technique have been developed to permit easier wire placement into the introducing needle. Self-contained systems are available in which the wire is introduced through a side sleeve in the needle or through a central hole in the aspirating syringe (e.g., Safety Syringe, Arrow International, Inc, Reading, Pa). Another variation of this system maintains the in-line wire introduction while aspiration is done though a small tube and side port of the needle. Such systems are designed to permit a more rapid or reliable placement of the wire once the vessel is cannulated. Although quicker, these devices sacrifice operator sensitivity in teasing difficult wires into place.

The guide-wire technique is readily adapted to placement of catheters in any body cavity. The technique used in these situations is identical to that for vascular access, except that blood is not aspirated through the introducing needle. If a technique requires aspiration of air (e.g., puncture of the cricothyroid membrane), it is helpful to partially fill the aspirating syringe with sterile saline. When the trachea is entered, bubbles will appear in the syringe as the air is aspirated. Other situations in which a wire-guided technique is used include intra-abdominal abscess drainage, peritoneal lavage, percutaneous nephrostomy and cystostomy, cricothyrotomy, pericardiocentesis, tube thoracostomy, and retrograde endotracheal intubation. [13] [14] These procedures are discussed more fully elsewhere in the text.

# COMPLICATIONS

Vascular access using the guide-wire technique has gained wide acceptance in emergency and critical care units. Any complication inherent in the performance of central or peripheral vascular access with other systems may also occur with this technique. Intuitively, the smaller needles used with the guide-wire technique should produce fewer complications than the larger needles used in the catheter-through-the-needle systems. However, the only comparison study between these two techniques failed to demonstrate any significant decrease in complications with the guide-wire technique. [15] Yet, in another study in which only the guide-wire technique was used, few complications were noted in central venous catheter

placements. [16] Other reports have shown quicker access or higher success rates with wire-guided techniques compared to standard or cutdown approaches. [5] [17] [18]

Some complications unique to guide-wire use may occur. Wires have been reported to form knots or to perforate vessels if forcibly introduced. Experience has shown that the wire will indeed kink or separate when ego exceeds wisdom and removal of the wire from a catheter, rather than removal of the two as a unit, is forcibly attempted. If kinking occurs, whatever portion of the system that contains the wire should be removed along with the damaged wire.

Lost wires have embolized when the technique was not performed correctly (e.g., the wire was not grasped at all times or was forcibly removed from an introducer needle). If embolization occurs, the procedure should be stopped and radiographs obtained to localize the wire. Portions that embolize into the central circulation must be removed using some form of retrieval device in an angiography suite.

Lost wire fragments that are subcutaneous and are easily palpated may be fixed in place by application of direct pressure just proximal to the location of the piece. Once the wire is immobilized it may be removed like any other palpable foreign body. Wires that cannot be felt but can be seen on x-ray in the area of the attempted catheter placement are best removed surgically with fluoroscopic assistance. Patient movement should be minimized to prevent more proximal migration of the wire during preparations for surgery.

On occasion, wire fragments can be isolated in an extremity by placing a tourniquet proximal to the wire. This will prevent any central migration until surgical removal is performed under fluoroscopy.

The use of the guide-wire technique also has allowed the introduction of a number of new catheter styles, each with its own complications. Multi-lumen central venous catheters tend to be larger and stiffer than their single-lumen

counterparts, making them more likely to cause vessel perforations. In addition to size, the configuration of the catheter affects its tendency to perforate. A model comparing the relative perforating potential of various catheters revealed that pigtailed or flexible-tipped catheters were less likely to perforate a simulated vessel wall than were straight catheters. [19]

Sheath introducers also have demonstrated some unique complications. Thin-walled sheaths may collapse or crimp from external tissue pressure or from being forced into a severe bend. This is not a problem if the sheath is being used to introduce a pacemaker, but if it is being placed for fluid resuscitation, its efficacy decreases markedly. Newer devices with internal supports or flexible sections avoid this problem and allow much greater freedom in the bending of the sheaths.

Another complication of such devices concerns the adaptors used to seal the proximal ends. Because of the large size of these catheters, the chance for air entry into the system is greater if the caps or diaphragms are not fitted properly. Air leakage and

actual air emboli have been reported to result from improperly sealed devices.

**CONCLUSION**

The development of the guide-wire technique for catheter placement has made possible a simple, rapid, percutaneous system for entry of almost any desired vessel or cavity. Because it can be learned quickly and it presents minimal complications, this approach is well suited to emergency and critical care vascular access.

# Chapter 22 - Venous Cutdown

**Steven C. Dronen** , **Patricia Lanter**

"The standard cutdown is well known to all trauma surgeons and needs no description." [1] Were this a true statement, the remainder of this chapter would be superfluous; however, physician training in the technique of venous cutdown has been largely informal. For 4 decades, the mechanics of venous cutdown have been handed down from house officer to house officer as one of the rites of internship. There are, in fact, few detailed descriptions of the procedure in the medical literature, and the scientific data documenting its usefulness or complication rate are sparse.

An early description of the technique of venous cutdown was provided in 1940 by Keeley, who offered the procedure as an alternative to venipuncture in patients in shock or in individuals with small, thin veins. [2] In 1945, Kirkham gave the first detailed description of the saphenous vein cutdown at the ankle. [3] Although the article is somewhat dated, most of the steps remain unchanged.

The most significant changes over the past 4 decades have involved not the technique itself, but the cannulas that are used. Keeley and Kirkham used metal needles. With the advent of plastic cannulas in the mid-1940s, the cutdown became more popular as a means of providing long-term IV infusion. Physicians have used IV tubing, feeding tubes, and even nasogastric tubes as cannulas in the management of hypovolemic patients. Currently, large-bore catheters (10-ga, 8 Fr) are often inserted by cutdown in the management of hypovolemic shock. [4]

The frequency with which the venous cutdown is performed is impossible to estimate. The growing popularity of central venous cannulation by the internal jugular, subclavian, and femoral routes has most probably decreased the frequency of venesection. Nevertheless, the cutdown remains

an excellent method of obtaining venous access in several emergent clinical situations. Although a cutdown is mechanically simple to perform, ease of performance does not guarantee that the procedure will be performed efficiently and without complications. Performance of a rapid, effective cutdown can be achieved only by thorough knowledge of the procedure and attention to its many details.

## INDICATIONS

There are no absolute indications for venous cutdown, simply because several options for venous access usually exist. The indications for use of the procedure are relative, depending to a great extent on physician experience and preference. There are several clinical situations in which the venous cutdown may be used.

**Venous Access in Infants**

Small children present a unique challenge to the clinician who does not perform pediatric venipuncture regularly (see Chapter 18) . The challenge is greater still if the procedure must be performed rapidly in a child or infant in shock or in a child in whom few, if any, veins are visible. Other options frequently used include intraosseous infusion or cannulation of the femoral, subclavian, or internal jugular veins. In selected circumstances (e.g., status epilepticus), the rectal route of administration, with mucosal absorption, is an effective alternative to cutdown.

A venous cutdown may be performed when all accessible peripheral sites, including scalp veins, have been exhausted. The distal saphenous vein is large enough to cannulate in most children and has a predictable anatomic location. Therefore, venous cutdown at the ankle is commonly used for both emergent management and long-term venous access. [5] [6]

**Hypovolemic Shock**

Rapid percutaneous insertion of large-bore (14-ga) catheters is appropriate in most cases of hypovolemic shock. Unfortunately, peripheral vessels frequently collapse in hypovolemia or have been rendered useless by IV drug abuse or previous venous catheterization. The venous cutdown is an acceptable alternative in these instances, although percutaneous insertion of a large-bore introducer device into a central vein can usually be performed more rapidly. When the cutdown can be performed quickly, it offers the advantage of direct visualization of the vessel during cannulation.

The use of the cutdown as a vehicle for the insertion of IV extension tubing and rapid transfusion was made popular during the Vietnam war. [7] The technique also has been found useful in civilian practice for the resuscitation of patients with profound hypovolemia. [8] The flow rate for saline through a standard IV extension set (3 mm inside diameter) cut to a length of 28 cm (12 inches) and inserted directly into the vein is 15 to 30% greater than that through a 5-cm, 14-ga catheter. The difference is greater if pressure is applied to the system. The improvement in flow rate through large-bore lines is greater for blood than for crystalloid solutions, because the viscous characteristics of blood greatly impede its passage through small-bore tubing. [8] [9] A unit of blood can be transfused in 3 minutes using IV extension tubing inserted into the vein. Consequently, large-bore lines placed by venous cutdown are an excellent mechanism for the treatment of severe hypovolemia. High-flow infusion techniques are discussed further in Chapter 23 .

# CONTRAINDICATIONS

Venous cutdown is contraindicated when less invasive alternatives exist or when excessive delay would be required for the procedure to be performed. [10] Although highly skilled operators may perform a cutdown in <60 seconds, [11] studies by Rhee and colleagues [12] and Iserson and Criss [13] have shown that, on average, the procedure requires 5 to 6 minutes to complete. Percutaneous insertion of large-bore catheters is the preferred method of rapid fluid infusion unless high flow rates are required or

peripheral vessels have collapsed. Another method of rapid fluid infusion that is technically easier and faster than venesection is the percutaneous insertion of large-bore introducer devices into the subclavian, internal jugular, or femoral veins (see Chapters 21 , 23 , and 24) . These devices typically use 8 Fr catheters with flow rates comparable to those obtained with IV tubing. The use of the subclavian and internal jugular vessels also is preferable to the cutdown for long-term applications.

Other contraindications are relative. In the presence of coagulation disorders, impaired healing, or compromised host-defense mechanisms, the need to perform a cutdown should be weighed carefully against the potential complications. Cutdowns should be avoided when there is an infection over the site and in extremities with injuries proximal to the cutdown site.

## ANATOMY

Detailed knowledge of anatomy is imperative to the success of this procedure. Veins in both the upper and lower extremities may be used. The choice of a particular vein should be governed by its accessibility and size and by the physician's experience and training. The anatomy of individual vessels and their relative merits as cutdown sites are described in the following paragraphs.

**The Greater Saphenous Vein**

The greater saphenous vein is the longest vein and runs subcutaneously throughout much of its course (Fig. 22-1) . It is most easily accessible at the ankle but may also be cannulated below the knee and below the femoral triangle. The greater saphenous vein begins at the ankle, where it is the continuation of the medial marginal vein of the foot. The vein crosses 1 cm anterior to the medial malleolus and continues up the anteromedial aspect of the leg. [14] At the level of the malleolus, the vein lies adjacent to the periosteum and is accompanied by the relatively insignificant saphenous nerve, which if transected causes sensory loss in a small area along the medial aspect of the foot. At the ankle, the vessel can be exposed with minimal blunt dissection. The vein's superficial, predictable, and isolated location has made the distal saphenous vein the classic pediatric cutdown site. [15]

The saphenous vein lies superficially on the medial

**Figure 22-1** Superficial veins of the lower limb.

aspect of the knee. A cutdown performed 1 to 4 cm below the knee and immediately posterior to the tibia has been described in the pediatric literature. [5] This site is distal enough to avoid interference with the performance of other resuscitative procedures, yet proximal enough to allow the passage of a long line into the central circulation. [16] However, it is seldom used for venous cutdown. Disadvantages of this site include kinking of the line as the knee is flexed and the risk of injury to the saphenous branch of

the genicular artery and the saphenous nerve. [17]

In the thigh, the saphenous vein begins on the medial aspect of the knee and crosses anterolaterally as it ascends toward the femoral triangle. Proximally, it enters the fossa ovalis and joins the femoral vein. Three to 4 cm distal to the inguinal ligament, the saphenous vein is of large caliber (4 to 5 mm outside diameter) and is easily isolated from the surrounding fat. Also lying anteromedially in the thigh is the lateral femoral cutaneous vein, which has a smaller diameter (2 to 3 mm) and lies lateral to the greater saphenous vein. [18] [19] The accessibility and large diameter of the greater saphenous vein in the thigh make its use an option in the treatment of profound hypovolemia. [8]

**The Basilic Vein**

The basilic vein is a preferred site for venous cutdown in the upper extremity. Veins of the dorsal venous network of the hand unite to form the cephalic and basilic veins, which travel along the radial and ulnar sides of the forearm, respectively (Fig. 22-2) . At the level of the mid-forearm, the basilic vein crosses anterolaterally and is consistently found 1 to 2 cm lateral to the medial epicondyle on the anterior surface of the upper arm. The medial cubital vein crosses over from the radial side of the arm to join the basilic vein just above the medial epicondyle. The basilic vein then continues proximally, occupying a superficial position between the biceps and pronator teres muscles. In this segment it lies in close association with the medial cutaneous nerve, which supplies sensation to the ulnar side of the forearm. The vein penetrates the brachial fascia in the distal third of the upper arm and then occupies a deeper position. [20]

The basilic vein is generally cannulated at the antecubital fossa 2 cm above and 2 to 3 cm lateral to the medial epicondyle. It is exposed through a transverse incision on the medial aspect of the proximal antecubital fossa. The size of this vein enables it to be located easily, even in the hypotensive or hypovolemic patient; large catheters can generally be passed without difficulty. The median cubital vein is accessible through the same incision. Superficially at this level, there are no important associated structures, but the brachial artery and the median nerve are found deep to the basilic vein.

A more proximal insertion site has been recommended by Simon and colleagues [21] to avoid the network of interconnecting veins at the level of the antecubital fossa. However, in the distal third of the upper arm, there is a closer association between the basilic vein and the medial cutaneous nerve. Transection of this nerve produces sensory loss on the ulnar side of the forearm.

**The Cephalic Vein**

This vessel begins on the radial aspect of the wrist and crosses anteromedially, ascending toward the antecubital

**Figure 22-2** Veins of the upper limb.

fossa. In the forearm it lies in close association with the lateral cutaneous nerve, which supplies sensory innervation to the radial aspect of the forearm (see Fig. 22-2) . In the antecubital fossa it lies subcutaneously, just lateral to the midline, and then ascends in the upper arm, overlying the lateral aspect of the biceps muscle. At the shoulder the cephalic vein lies in the deltopectoral groove. Just below the clavicle, it passes deep to end in the axillary vein. [20]

Venesection is easily performed on the cephalic vein because of its large diameter and superficial location. In the forearm it is important to avoid the lateral cutaneous nerve. A good location is in the antecubital fossa at the distal flexor crease. Cutdown on the cephalic vein at the wrist has also been reported, [22] but the thin skin overlying the vein at this level usually permits simple percutaneous cannulation when the vein is available for cannulation. The cephalic vein may also be entered in the deltopectoral groove. The slightly deeper position and physical interference with the performance of other procedures make this approach more difficult.

### The Brachial Veins

The brachial veins are small, paired vessels lying on either side of the brachial artery. In contrast to the vessels described earlier, these are not superficial and will not accommodate large cannulas. Their most superficial location is 1 to 2 cm above the antecubital fossa just medial to the biceps muscle. Palpation of the brachial pulse serves as a useful landmark. Because of its proximity, the brachial artery may be inadvertently cannulated in the pulseless patient. In addition, there is the risk of injury to the closely associated median nerve. Time-consuming blunt dissection is usually required because of the vessels' greater depth. For these reasons, brachial vein cutdown is *not recommended* as an emergency venous access route [10] and should be used only in the absence of a suitable alternative. This site may be acceptable when time and vessel size are not critical factors, but it is difficult to justify the deep dissection and associated risks that are involved.

### The External Jugular Vein

The external jugular vein begins below the angle of the mandible and is formed by confluence of the posterior auricular and retromandibular veins. It descends posterolaterally across the surface of the sternocleidomastoid muscle and then pierces the fascia to join the subclavian vein deep to the clavicular head of this muscle. The greater auricular nerve, which supplies sensation to the external ear, travels parallel to the external jugular vein. [14]

A venous cutdown may be performed on the external jugular vein at its superficial location on the sternocleidomastoid muscle. This is not recommended as a first-line means of venous access for the following reasons:

1. Performance of a cutdown may cause physical interference with airway management and central venous cannulation.

2. There is risk of injury to the greater auricular nerve.
3. It is difficult to immobilize the area adequately.
4. Cervical spine immobilization frequently prohibits access to the area.
5. It is a hazardous procedure in the uncooperative patient. [10]

As a general rule, cutdown on the external jugular vein should be performed only when other means of venous access are exhausted. The external jugular vein is an acceptable site for emergency *percutaneous* venous cannulation, especially in children.

## EQUIPMENT

The materials required to perform a formal venous cutdown are listed in Table 22-1 . All necessary instruments should be available on a sterile tray before the procedure is begun. The standard cutdown tray is shown in Figure 22-3 . A time-consuming search for the proper instrument can be avoided if only necessary instruments are included on standardized trays. Pediatric patients also require both a warming table or radiant warmer and a padded extremity board.

Catheter choice depends on the desired function of the venous line. When central venous pressure (CVP) monitoring is needed, the catheter chosen must be long enough to reach the superior vena cava. The average distance from the antecubital fossa to the superior vena cava is 54 cm in the adult male. This distance can be approximated by aligning the catheter over the chest with the tip at the level of the manubrial-sternal junction. Lumen size is relatively unimportant when the line is inserted for monitoring the CVP or to infuse drugs, but it is a critical factor in the treatment of hypovolemia. Short, large-bore catheters are preferred when fluid must be delivered rapidly. Silastic catheters, IV plastic tubing, or 5 or 8 Fr pediatric feeding tubes may be used as infusion catheters in older children and adults.

Tables 22-2 (Table Not Available) through Tables 22-4 (Table Not Available) list the flow rate of various fluids through some commonly used catheter systems. Knowledge of relative flow rates is essential if maximal benefit is to be obtained from the time spent performing the cutdown. Excellent flow rates can be achieved by threading IV tubing (sterile tubing may be cut to the appropriate length) directly into the vein or by using a 5 cm, 10-ga IV catheter.

## TECHNIQUE

The technique of venous cutdown is essentially the same regardless of the vessel cannulated (Figs. 22-4 through (Figs. 22-13

**TABLE 22-1** -- Materials Required for Venous Cutdown *

| |
|---|
| Curved Kelly hemostat |
| Scalpel with No. 11 blade |
| Small mosquito hemostat |
| Tissue spreader |
| Iris scissors |
| Plastic venous dilator or lifter |
| 4-0 silk suture ties |
| 4-0 nylon suture on cutting needle |
| Antibiotic ointment |
| Gauze sponges |
| 1-in. tape |
| Arm board |

| |
|---|
| Intravenous catheter |
| Rolled gauze bandage |

**Figure 22-3** Venous cutdown tray. Note the small plastic vein dilator-lifter *(arrows)*, which is especially useful in children.

Detailed knowledge of the local anatomy is important if the procedure is to be performed rapidly without injury to associated structures. Adequate immobilization on a padded board should be accomplished prior to beginning the procedure. This is especially important in children (see Fig. 22-13) . Even in emergency situations, reasonable precautions should be taken to avoid infection. The area of the skin incision should be widely prepared with an antiseptic solution and then draped. A tourniquet placed proximal to the cutdown site helps in the visualization of the vein.

In the conscious patient, the site is infiltrated with 1%

**Figure 22-4** A skin incision is made perpendicular to the course of the vein.

lidocaine. A skin incision is made transverse to the course of the vessel (see Fig. 22-4) . A longitudinal incision may decrease the risk of transecting neurovascular structures but does not provide sufficient exposure. A transverse incision involving all layers of the skin is the best approach. Subcutaneous (SQ) fat should bulge from the incision. The clinician bluntly dissects SQ tissue by spreading the tissue gently with a curved hemostat *in a direction parallel to the course of the vein.* Bleeding is usually minimal unless the vein is nicked. A tissue spreader or a self-retaining retractor may be used to provide a wider field. The vein is then isolated from the adjacent tissue and mobilized for 1 to 3 cm (see Fig. 22-5) (Figure Not Available) .After the vein is mobilized, a hemostat can be used to pass proximal and distal silk ties under the vein for stabilization. An alternative approach is to simply use the hemostat tips (with no ligatures) in a "spread" position to elevate and stabilize the exposed vein during incision and cannulation. The hemostat is placed as in Fig. 22-7 (Figure Not Available) , only without taking the time to pass ligatures.

If ligatures are passed prior to cannulation, the distal ligature may or may not be tied after initial placement. If the distal ligature is tied, it should not be cut, because the proximal tie is useful in controlling the vein (see Figs. 22-6 (Figure Not Available) and 22-7) (Figure Not Available) . Using a hemostat, the vessel is elevated and stretched flat. This provides good visualization and control of the vessel and limits bleeding when the vessel is incised. Alternatively, placing gentle traction on the proximal tie will control

oozing around the puncture site. The vessel is incised at a 45° angle, through one third to one half of its diameter (see Fig. 22-7) (Figure Not Available) . A No. 11 blade (as illustrated) or a pair of iris scissors may be used to incise the vessel. Too small an incision may cause threading of the catheter into a false channel in the adventitia; conversely, the vessel may be torn completely and may retract from the field if the incision is too large. [23] A longitudinal incision is sometimes made to avoid transecting the vessel, but the lumen is more difficult to identify with this technique. *The vessel incision must enter the actual lumen of the vein*, although some bleeding will occur after the vein has merely been nicked. Incision of the vessel is unnecessary when an IV catheter with an introducing needle is used. The vessel is simply punctured, as in percutaneous venous cannulation (see Mini-cutdown).

Before being introduced into the vessel, the cannula is beveled at a 45° angle unless a cannula with a tapered tip is used. A short bevel is preferred, and a sharply pointed tip is to be avoided, because it may pierce the posterior wall or otherwise damage the vein. The rounded tip of a feeding tube may be more difficult to introduce, but it may be advanced less traumatically. If using an IV cannula, the cannula may be introduced directly through the skin incision or through a separate stab wound. The latter method, using an Intracath device, is illustrated in Figures 22-8 (Figure Not Available) and 22-9 (Figure Not Available) . Theoretically, the percutaneous approach reduces the risk of infection. [24] Threading the catheter into the vein is often the most difficult and time-consuming portion of the procedure.

Difficulty in threading may be encountered for several reasons. The lumen may have been incorrectly identified, or a false passage may have been created. This frequently occurs and may be difficult to recognize, because the catheter can easily advance between layers of the vessel wall. Other causes of difficult threading are penetration of the posterior vessel wall, the presence of venous valves, or use of a catheter that is too large for the vessel being cannulated. Identification of

**Figure 22-13** The cutdown site is securely dressed and splinted.

the vessel lumen may be facilitated through use of a plastic venous dilator or elevator. The small, pointed tip of the device is threaded into the vessel to expose the lumen in advance of the catheter (see Fig. 22-10) . A sterile 20-ga needle bent at a 90° angle may also serve as a vein elevator. A vein dilator is useful in pediatric cutdowns but is generally unnecessary in adults. The clinician can facilitate the threading of large catheters in adults by grasping the proximal surgical edge of the vessel with small forceps or a mosquito hemostat. Counter traction is applied as the catheter is advanced (see Fig. 22-11) . At no time should one force a catheter that will not advance.

Once the catheter is advanced into the lumen, air is back-bled from the cannula, and the cannula is connected to IV tubing. The proximal ligature is tied around the vessel wall and the intraluminal cannula. The tourniquet is now removed, the catheter is affixed to the skin, and the incision is closed (see Fig. 22-12) . An antibiotic ointment is applied at the point at which the catheter passes through the skin, and the wound is dressed. In an emergent situation, skin closure can be delayed and the wound simply wrapped with a

sterile dressing (e.g., Kerlix) (see Fig. 22-13) . The IV tubing should be looped under the outer layers of the dressing to minimize the risk of inadvertent removal of the cannula should the external IV line be tugged.

## Mini-Cutdown

An alternative method designed to preserve the vein and bypass the time-consuming step of placing a catheter into the vessel has been described. [25] A skin incision and blunt dissection are used to locate the vessel. Once identified, the vein is punctured under direct vision with a standard percutaneous venous catheter. Alternatively, a large-bore introducer device with or without a dilator can be threaded over a guide wire using this technique. The needle may be introduced through a separate stab incision or through the skin incision. If an over-the-needle device (e.g., Angiocath, Medicut) is used, the needle is withdrawn and discarded. With a through-the-needle device, the cannula is threaded into the vein, and the needle is withdrawn to the skin surface (Fig. 24-14) (Figure Not Available) . A guard is placed on the needle tip, the

**Figure 22-14** The mini-cutdown technique is an alternative to the venous cutdown method. The vein is cannulated under direct vision using standard percutaneous catheters. A separate entry site (shown) may be used, or the vein can be cannulated through the skin incision. Note that the vein is not tied off with this technique. A standard Angiocath IV set also may be used instead of the through-the-needle catheter shown here.

catheter device is fixed to the skin, and the incision is closed. This method eliminates the need for tying or cutting the vein, thereby permitting repeated catheterization. Venipuncture is easier and uses the same equipment as for percutaneous venous cannulation. The mini-cutdown is therefore used in the treatment of chronically ill patients who require long-term IV therapy or in children who have limited accessible veins. A simple skin incision may also permit direct visualization of veins in the obese patient and may facilitate standard percutaneous venipuncture.

Hansbrough and coworkers [4] described the mini-cutdown procedure with a 10-ga IV catheter (Deseret 10-ga Angiocath). The flow rates of blood and saline with this catheter are equal to the rates obtained when IV extension tubing is placed in a vein using the more time-consuming standard venous cutdown technique. This catheter allows one to infuse a unit of whole blood in 2 to 3 minutes if pressure and oversized IV tubing (e.g., urology irrigation tubing) are used.

Shockley and Butzier describe a further modification whereby the guide wire technique is modified by passage of the guide wire, dilator, and sheath system following standard cutdown and venotomy. [26] They found this technique to save more than 2 minutes' time over the standard technique when performed by novices.

Removal of catheters inserted by cutdown requires only cutting of the skin stitches

holding the catheter in place, followed by withdrawal of the catheter. Backbleeding from the proximal venous end is controlled by a simple pressure dressing and is generally not a significant problem.

## COMPLICATIONS

The complications of venous cutdown include local hematoma and infection, sepsis, phlebitis, embolization, wound dehiscence, and injury to associated structures. An indirect but significant complication is deterioration of an unstable patient during a time-consuming cutdown attempt. Documentation of complications and their frequency has been sparse. Bogen reported a 15% complication rate in 234 cases. [27] Infection and phlebitis each occurred at a rate of 4%. Infectious complications may result from the introduction of pathogens during line placement, transcutaneous invasion along the course of the cannula, or deposition of blood-borne organisms on the catheter tip. [28] A clear correlation exists between the incidence of infectious complications and the length of time that a catheter is left in place. Moran and associates found that the infection rate rose from 50 to 78% when a catheter was left in place for more than 48 hours. [29] Druskin and Siegel, studying a mixed population of patients who had undergone cutdowns and others who had catheters percutaneously inserted, found that the incidence of culture-positive catheter tips rose from 0 to 52% after 48 hours. [28] In the study by Moran and colleagues, *Staphylococcus albus* was the predominant organism that was isolated, but organisms more commonly thought of as pathogenic ( *S. aureus*, *Enterococcus* spp., and *Proteus* spp.) were isolated with greater frequency from cutdowns that had been in place for long periods. [29] Rhee and colleagues reported a 1.4% infection rate (one episode of cellulitis) following 73 cutdown attempts. [12] All catheters were removed within 24 hours.

There is some evidence that the rate of infectious complications decreases when a broad-spectrum antibiotic ointment is applied to the cutdown site. Moran and coworkers found a rate of infectious complications of 18% when topical polymyxin B-neomycin-bacitracin (Neosporin) was used, compared with a 78% rate in a placebo-treated group. [29] In this study, it was also shown that topical antibiotic use results in only a moderate decrease in the incidence of phlebitis (from 53 to 37%) but a significant decrease in the incidence of phlebitis associated with positive cultures (from 86 to 14%). This suggests that phlebitis is primarily a chemical or an irritative process rather than the result of infection. [29] Whatever the cause, the incidence of phlebitis is clearly related to the duration of catheterization. [7] [27] [30] Early catheter removal is a key factor in the prevention of both phlebitis and the infectious complications of venous cutdown. This is especially true of lines inserted during emergency resuscitative treatment. Such lines should be removed as soon as the patient's condition stabilizes and alternative routes exist. [8] [10]

Proper attention to the details of surgical technique will limit the occurrence of minor complications, such as local hematoma, abscess, and wound dehiscence. One can avoid injury to associated structures by selecting a site in which the vein is isolated and specifically avoiding brachial vein cutdown.

# Chapter 23 - High-Flow Infusion Techniques

**Kenneth V. Iserson**

High-flow infusion techniques date back to the Vietnam War era. During that period, surgeons placed intravenous (IV) tubing directly into veins to facilitate rapid high-volume infusions. Rapid isotonic volume resuscitation following hemorrhage has been promoted as a means to minimize shock-related organ injury and death. [1] Recently, laboratory [2] [3] and clinical investigations [4] have raised concern that overzealous volume resuscitation of patients with penetrating trauma prior to definitive control of hemorrhage may increase hemorrhage volume and mortality. However, similar laboratory studies have demonstrated that delayed volume resuscitation is associated with increased metabolic acidosis. [5] Regardless of the optimal timing for fluid resuscitation (i.e., either "as soon as possible" or "after control of hemorrhage"), high-volume techniques remain an important therapeutic intervention for patients with significant volume deficits.

A difficulty in describing high-flow techniques is that there has been a progressive increase in flow rates in what is described as "high-flow." Whereas 1 L/hour of isotonic fluid was at one time considered a rapid infusion, isotonic fluids can now be placed through peripheral IV lines into normovolemic adults with commercially available equipment at rates exceeding 800 mL/min. [6] "High-volume" infusion of crystalloids may be set arbitrarily at 500 mL/min. Massive transfusion of blood has been traditionally defined as transfusion of an amount at least equal to the recipient's blood volume within a 24-hour period. [7] However, with commercially available equipment, the in vitro flow rate of admixed erythrocytes can exceed 800 mL/min through one line. [8] One case has been reported of a patient surviving after having received more than 12 units of blood product per hour for 30 hours.

## IMPORTANCE OF TECHNIQUE

Clinicians often have little time in which to restore effective circulating intravascular volume in patients who have had acute loss of volume (e.g., through traumatic or nontraumatic hemorrhage or from septic or anaphylactic shock). In patients who have lost large volumes of fluid in the period of minutes, as is seen with acute injury and illness, high-flow infusion techniques are often used.

Hypovolemic shock also results in an increase in intravascular capacity, requiring much more IV fluid to be infused than the volume of blood lost. [10] At some point in resuscitation, sufficient intravascular volume (preferably with adequate oxygen-carrying capacity) to supply nutrients for cellular metabolism must be restored. Those techniques of rapid high-volume infusion that can be used successfully with only one large-bore IV line in place gain added significance in situations in which multiple lines can be established only with difficulty (e.g., in patients with severe shock, a history of IV drug abuse, fragile veins, massive swelling, or obesity).

## ROLE OF HIGH-FLOW INFUSION

High-flow infusion may be used by all clinicians, paramedical personnel, and nurses who treat critically ill patients. Trauma patients, patients with gastrointestinal bleeding, and those in septic or anaphylactic shock are prime candidates. The techniques involved are relatively easy to learn, and the equipment is both inexpensive and similar enough to that already being used for standard IV fluid therapy that no major barriers should exist to using high-flow techniques when indicated.

Some of the techniques, especially those using the automated external pressurization devices, reduce the time needed for personnel to infuse fluid by conventional IV techniques (Table 23-1) (Table Not Available) . [12] Large (12- or 10-ga) peripheral IV catheters are not much more difficult to place than the 14- or 16-ga catheters that are routinely recommended. In the pediatric population, a venous cutdown, whether to directly insert IV tubing or catheters, requires a significant amount of time and skill. [13] Although venous cutdown lines (see Chapters 18 and 22) can be used in conjunction with high-flow infusion systems, cutdowns are not addressed further in this chapter.

## INDICATIONS AND CONTRAINDICATIONS

The primary indication for high-volume infusion is hypovolemic shock. Although the infusion may be needed only for a short period until vital signs stabilize, preparations are often made for the necessary equipment to be available "on standby" in case the patient's condition deteriorates again.

Often it is unclear initially whether the patient requires high-volume resuscitation. Despite historical evidence suggesting massive volume loss, patients may be hypotensive for other reasons or may arrive in a volume-resuscitated condition after a prolonged transport with associated volume administration. If any doubt exists regarding the need for high-volume infusion, short, rapid boluses (10 to 20 mL/kg) of fluid can be given and the patient repeatedly reassessed.

The goals of shock resuscitation are to minimize further volume loss and to maintain organ perfusion until the shock-induced pathophysiologic state can be reversed. In hemorrhagic shock, control of ongoing blood loss is essential. When the blood loss is internal, excessive volume resuscitation accompanied by elevation of blood pressure may exacerbate the blood loss preoperatively. The classic clinical example of this phenomenon is the patient with a leaking aortic aneurysm. Hence volume resuscitation must be closely monitored.

Rapid high-volume infusion is relatively contraindicated in cases of neurogenic shock following trauma. Because the hypotension of neurogenic shock is not due to hypovolemia, hemodynamics do not improve with administration of large amounts of fluid. Rather, the fluid often collects in the patient's lungs and leads to pulmonary edema. Patients who can be recognized as having hypotension that is due to a pericardial tamponade may do well with administration of small, rapid boluses of fluid

until a pericardiocentesis or definitive repair is performed, but large volumes of fluid do not appear to be beneficial. Patients whose hypotension is not due to hypovolemia (e.g., those with pump failure or impaired venous return) can be harmed by the fluid overload that would accompany a rapid, high-volume infusion.

# EQUIPMENT

The key to performing effective rapid infusion therapy and to obtaining the proper equipment is to consider the entire infusion system, rather than just the individual parts. Each

part of the infusion system must be able to meet the desired flow and time requirements (see Table 23-1) (Table Not Available) .

Unfortunately, IV catheters with "exactly the same measurements" can deliver fluid at surprisingly different rates. [14] (Note in Table 23-1 (Table Not Available) that three different manufacturers make 14-ga 2-in. catheters, and the flow rates by gravity range from 93 to 134 mL/min.) Therefore, when choosing an IV system for an emergency department, it should be tested to ensure that the system and all components perform as advertised.

## Catheter

Fluid flow through a tube, as in the case of an IV catheter, generally behaves in a fashion described by Poiseuille's law:

With all elements of a high-flow infusion system, it is the internal diameter (ID) that is overwhelmingly important in determining flow rates. The ID affects flow rates to the fourth power, whereas all other elements affect flow only to the first power. Therefore, to obtain a maximal flow rate, the goal is to optimize the ID of all parts of the system. For example, more rapid infusion can occur by increasing the diameter (smaller gauge) of one IV catheter than by starting a second line with another small-ID catheter. [15]

Although some very large experimental catheters, exceeding 4.5 mm (14 Fr) ID, have been developed and used successfully for infusions in humans, [16] [17] large, commercially available percutaneous IV catheters have a maximum ID of 2.16 mm (12 ga, Vygon Mosquito 123) or 3 mm (9 Fr). [14] Swan-Ganz introducers are not tapered and have a very thin-walled construction. Because they are designed to accommodate the size catheter for which they are labeled, the labeled size is usually their ID.

Flow resistance in a tube is inversely proportional to tube length. However, even with long IV catheters, the ID is much more important for determining flow resistance than catheter length. Catheter length is significant primarily because the catheter must be long enough to mechanically remain in place within the vascular space. Theoretically, an IV catheter for rapid fluid replacement should have its maximum ID maintained throughout its length. This means not only that the main portion of the catheter should have a maximum ID consistent with structural stability, but also that the reduction in ID

from narrowing at the catheter tip should be minimal. Some catheters lose more than 36% of their ID in this taper.

Choosing an intraosseous infusion site constitutes a major exception to using the catheter ID to extrapolate the resultant flow rate. Even when large-ID catheters are placed, the resistance to flow out of the intraosseous space appears to limit rapid, high-volume infusion at this site.

**Intravenous Tubing and Extension Tubing**

Poiseuille's law applies to IV tubing as well as to indwelling catheters. Most standard IV tubing for blood transfusions has an ID of 3 mm. [6] [18] Larger tubing is now available, some with an ID of 3.66 (11 Fr) to 4.0 mm. A more recent development is the Medex Hi-Flow Trauma Quad System tubing, with an ID of 5.7 mm. Although this large-bore tubing can sustain flows of more than 1 L/min of admixed blood, it essentially ceases to function because of obstruction of its own filter after 3 units of packed cells have been infused. This problem can be overcome successfully by using an in-line Pall filter. [8]

Using even the 3.66-mm tubing can increase flow rates of saline from 70% (12-ga catheter, pressurized) to 126% (8 Fr catheter, no pressure) over rates with the 3-mm tubing. [6] A comparable difference would be expected with blood products.

Even if large-diameter IV tubing is used, adding a smaller-sized extension tubing can reduce the ID of the system enough to make this element the rate-limiting step, negating the benefit of the other components of the system. If large-ID IV tubing is used, it is essential that similarly sized extension tubing also be available. Improving the efficiency of the drip chamber also would improve flow, but little progress has been made so far in this system component. [15]

Even when crystalloid is infused, all IV tubing must be of the "Y"-type. That is, there must be at least two attachments for IV bags. This feature allows one bag to be changed while the other is infusing. At the flow rates achieved by rapid high-flow systems, not having Y-type tubing will normally cut the overall system infusion rate by at least half. Y-type tubing is also necessary to admix blood with warmed saline, as described later.

**Solution Container**

All fluids used in the resuscitation of hypovolemic patients are now available in compressible, soft plastic containers. The more rigid plastic and glass containers should be avoided, because they are much more difficult to externally pressurize in the former case and present the danger of breaking in the latter case. To conserve the number of bag changes that personnel have to perform, a time-consuming element of infusion, 1-L bags are preferable to smaller sizes for the adult patient. Blood, generally in the form of packed red blood cells, is packaged in standard plastic containers that can accommodate up to 600 mL of fluid (see Blood Warmers).

**External Pressure Device**

Pressurizing the fluid that is being infused increases the fluid flow rate dramatically. [6] [11] Traditionally, clinicians have tried to pressurize IV fluid systems bags by manually squeezing them, kneeling on them, using blood pressure cuffs around them, and using commercial pressure devices. Only manually squeezing the bags or using commercial pressure devices is effective. [19]

The most practical means of adding pressure to a high-flow IV system is through the use of external pressure devices (Fig. 23-1) . The standard maximal external pressure exerted by these units is 300 mm Hg. Until recently, most centers were still using pneumatic pressurization devices, with the inflow and outflow of air being controlled by a screw-down mechanism--the blood pump. [20] This device is not only cumbersome but also unreliable in either maintaining pressure or releasing pressure so that a new bag of fluid or blood can be placed. [12] More recently, somewhat less expensive pressure bags with 3-way stopcocks have become available.

**Figure 23-1** Three types of external pressure devices. *A*, Infuser-1. (As modified in Iserson KV, Reeter A, Woods W, Criss E: Pressurization of IV bags: A new configuration and evaluation for use. J Emerg Med 3:89, 1985.) *B*, Infusable (Biomedical Dynamics Corp., Minneapolis). *C*, Alton Dean infuser (North Salt Lake City, Utah).

These appear to be easier to use but still have the problem of not maintaining a constant pressure on the fluid while the bag empties. Some pressure bags (Infusable, Biomedical Dynamics Corp., Minneapolis) are considered to be disposable, even though many uses can be obtained from each unit.

Other, more elaborate external pressure devices also have been advocated, including blood pumps attached to a constant air-pressure source. [21] More recently, these pressurization devices not only have been powered by wall air, but also have been contained in a rigid metal or plastic container for safety, more rapid access, and extremely rapid pressurization and depressurization (Infuser-1 and Alton Dean Infuser, North Salt Lake City). These devices have the advantage of maintaining a constant pressure on the fluid container during emptying. It has been shown that using the air-pressure devices dramatically decreases the time needed for changing bags during infusions (Table 23-2) (Table Not Available) , thus enhancing the effective flow rate during high-volume infusion situations. [12] [22]

Some initial work has been done with devices that exert a higher external pressure on the fluid containers. Pressures of 600 mm Hg have been obtained. As expected from Poiseuille's law, the larger the ID of the tubing and catheters in the system, the greater the increase in flow rate achieved with increased pressure. Although this high pressure does not damage the equipment, it is unclear whether blood cells can be damaged at this pressure. [11]

**Blood Warmers**

In nearly all high-volume infusions, blood products are administered. This poses the problem of infusing blood products at or around 4 °C (the temperature at which blood is stored) or using a blood warmer in the system. Rapid infusion of cold blood is associated with an increase in ventricular fibrillation and cardiac arrest. [23] [24] Hypothermia is a major, yet often unappreciated, problem during the resuscitation of seriously ill patients, particularly those who require multiple blood transfusions. [25] Blood warmers heat blood either before the infusion (prewarming) or during the infusion (in-line). Most commonly available warmers are the in-line type, and most of them significantly slow the flow rates of high-flow systems. [24] [26] [27] Some blood warmers have been developed that have more rapid flow rates, but these are not readily available. [28]

One proposed method for keeping flow rates high while warming blood to approximately body temperature is to dilute the blood with an equivalent volume of 70 °C saline. [29] [30] [31] [32] This technique, because of the rapid equilibration of temperatures between the two mixing fluids, does no damage to the erythrocytes. Of course, because of the resulting warm temperature of the blood (approximately 37 °C), it must be infused rapidly. In addition, *the 70 °C saline must never be used for direct infusion* (Fig. 23-2) .

One commercial system combines warmed saline at 45 °C with cold blood in a single pressurized mixing bag and delivers the admixed solution through a high-flow infusion set (Sangui-Stat System, Ackard Laboratories, Cranford, NJ). This system is designed specifically for the rapid warming and transfusion of packed erythrocytes in the emergency department, although the resultant temperature is below body temperature and will contribute to hypothermia with large-volume infusions (Fig. 23-3) (Figure Not Available) .

**Fluids**

Although a great deal of experimental work has been done to compare lactated Ringer's solution and normal saline in hypovolemic resuscitation, the solutions seem to be equivalent in clinical settings. The application of small-volume hypertonic saline in the resuscitation of patients in hypovolemic shock is promising, but use of this fluid continues to be experimental. [33] [34] [35]

When transfusing cellular blood products rapidly, it is essential not only to warm the fluid but also to dilute it adequately to reduce the incidence of erythrocyte hemolysis. Cellular destruction in blood flow systems is directly proportional to the magnitude of shear stress and cellular-solid surface interactions. These are affected directly by hematocrit and plasma protein concentration. The greater the dilution of the blood product, the less hemolysis. [36] [37]

**System Perspective**

Although new equipment items continue to be developed to assist in high-volume infusion, all items are limited by the same essential principle--the minimum ID of the system must be as large as possible. [16] [17] Equipment that only offers increased pressure, decreased setup and takedown time, or decreased system tubing length should be accompanied by an appropriately large system ID.

## PROCEDURE

### General Concepts

Usually the most difficult part of initiating a rapid, high-volume fluid system is ensuring adequate preparation through in-service training of physicians and nurses, stocking essential components, and setting up the components to be used. The equipment should be located conveniently close to the patient and within reach of the practitioners.

Given appropriate preparation, the next difficulty is establishing IV access with a large-bore catheter. Catheter placement should proceed with the largest size catheter that can be quickly placed. Even if only a small-bore catheter can be placed initially, a larger catheter often can be inserted through this catheter using the Seldinger technique (see Chapter 21) . Furthermore, alternative sites can be sought for a larger-bore catheter as volume resuscitation begins through a smaller-bore catheter. Ideally a 10- or 12-ga, 9 Fr, or larger catheter is placed in either a peripheral or femoral vein. Central veins about the neck should be avoided if possible, because extravasation with rapid flow rates may lead to a large hematoma and possible airway obstruction.

Once one or more catheters are in place, large-bore Y-type tubing, no less than 3.66-mm ID, should be attached. If an extension tube is needed, it must have at least as large an ID as the rest of the tubing. Prewarmed 1-L bags of crystalloid solutions (ideally at 37 °C) should be initially hung in an external pressure device. Either an easily used manually operated pressure bag (Infusable, Biomedical Dynamics Corp, Minneapolis) or a wall-operated pneumatic device (Infuser-1 and Alton Dean Infuser, North Salt Lake City) is acceptable.

When erythrocyte units are available, they should be diluted 1:1 with 70 °C preheated 0.9 N saline. ( *Note:* Do not use lactated Ringer's solution.) When performing this procedure, use a special system (e.g., Fenwal plasma transfer set, No. 4C2243, Fenwal Laboratories, Deerfield, Ill) to guarantee that the hot saline will not be directly infused into the patient. [32] [38] Many types of laboratory incubators can be used for preheating the saline. A constant temperature monitor should be available to ensure adequate heating. According to tests by the manufacturer, bacterial growth within the unopened heated saline units should not occur for at least 2 weeks if the units are left in their overwrap packaging. [29] When the 1:1 dilution is used, the admixed blood is then at approximately 37 °C and ready for rapid infusion. Mistakes may be minimized by preheating only one particular-sized bag (250 mL is appropriate and usually the right amount for diluting the erythrocyte unit) and premarking the bags for dilution with bright orange or yellow tape. Heated saline bags should be discarded if not used after 2 weeks

to avoid chemical leaching from prolonged heating. [38]

If a rapid-infusion system is used in one part of an institution, it is essential not only that other interactive units (e.g., the operating room and intensive care unit) be made aware of it, but also that any equipment that the other units use in managing critical care patients be compatible with that used for rapid infusion in the emergency department.

**Vascular Access Sites Affecting Flow In Vivo**

The limitation to placing large venous catheters in an adult or a larger child is generally the skill of the operator. Any

large peripheral or central vein is large enough to accommodate very large catheters, with the natural distention that occurs. There is virtually no obstruction to flow from any anatomically intact venous site. [17] However, placing catheters in veins distal to an area of vascular injury, such as in a lower extremity in a patient with abdominal bleeding, may limit the usefulness of the fluid resuscitation.

**Intravascular Volume Affecting Flow In Vivo**

It does not appear that intravascular pressure significantly limits the flow rate of rapid-infusion IV fluid. Although the estimated backpressure is 15 to 18 mm Hg in supine, normovolemic males, this seems to decrease the in vitro flow rates only by a negligible 5 to 6%.

## COMPLICATIONS

The complications of high-volume infusion usually are related to one of three problems:

1. The catheter may be in the wrong location so that large amounts of fluid enter into a closed space, such as the chest or pericardium, with untoward effects. Rather than being resuscitated by the rapid fluid infusion, the patient deteriorates as a result of, and in proportion to, the amount of fluid infused.
2. Too much fluid is inadvertently and unknowingly infused. Unfortunately, there is no totally satisfactory way to monitor intravascular volume in the acute setting, and excessive fluid resuscitation is surprisingly easy. Close attention to vital signs, pulmonary status, and urine output is important. When available, central venous pressure monitoring or, preferably, Swan-Ganz

   catheter monitoring may be used, but these are generally available in the acute resuscitation setting.

3. Problems occur related to massive blood transfusions. The major and most immediate problems relate to hypothermia (with resultant dysrhythmias) and coagulopathies. These may be mostly ameliorated with the use of warmed blood and the timely use of fresh frozen plasma (see Chapter 29) .The complication rate

for high-volume fluid therapy in patients with severe hypovolemia is unknown. The patient's condition, "hypovolemic shock," usually implies a poor outcome from the start. Limited information on massive, rapid fluid boluses in otherwise healthy animals suggests that there are few problems that do not resolve once the infusion is stopped. [39] However, in the injured patient, cellular changes accompanying injury (e.g., pulmonary, neurologic) may not allow reversal of fluid-induced changes as easily. Further, concerns have been raised that over-resuscitation may exacerbate hemorrhage through a variety of mechanisms. [2] [3] [4] If a patient is believed to have been overloaded with fluid, normal techniques to reduce the amount of fluid (e.g., stopping the infusion, using diuretics, and appropriately using cardiovascular or respiratory support) should be instituted.

## INTERPRETATION

Success with rapid IV infusion techniques is often fleeting. Often the goal is only to buy enough time to institute definitive operative intervention. Interpretation of fluid resuscitation, at least at the present time, is largely clinical. Evaluation of the entire clinical picture, including vital signs, urine output, peripheral perfusion, and mentation, is necessary to determine the success of infusion therapy. Occasionally, of course, when a thoracotomy has been done, direct observation of the central circulation allows an accurate determination of the state of vascular filling.

With rapid infusion consisting primarily of crystalloid solutions, patients sometimes have enough blood loss to leak "water" from bleeding sites. This is essentially a visual manifestation of an extremely low hematocrit and indicates a nearly hopeless prognosis. Otherwise, hematocrit measurements are virtually useless in assessing the intravascular status of unstable patients. Some patients, especially younger ones, continue to have enough vascular tone to appear to stabilize after an initial large bolus of fluid. These patients must be carefully observed and evaluated, because their condition can deteriorate again without warning.

## CONCLUSION

Unfortunately, success with rapid, high-flow IV infusion does not guarantee a successful outcome for the acutely ill patient. Further, in the emergency department or out-of-hospital setting, it is often impossible to assess the severity of injuries. Therefore, rapid, high-flow infusion therapy should be considered in conjunction with other therapies when seriously injured patients remain hypotensive and display clinical signs of shock despite delivery of oxygen, ventilation, and a modest volume challenge.

# Chapter 24 - Central Venous Catheterization and Central Venous Pressure Monitoring

Steven C. Dronen, John G. Younger

## Central Venous Catheterization

The popularity of central venous access has paralleled the medical advances of recent years and is increasingly part of patient care in the emergency department and intensive care setting. Advanced monitoring techniques, transvenous pacemakers, and parenteral nutrition all require rapid, reliable methods of central venous access. Peripheral venous sites can be used for some of these procedures, but use of peripheral sites requires long catheters that must be threaded accurately into the superior or inferior vena cava. Peripheral veins may be collapsed, thrombosed, buried in subcutaneous (SQ) tissue, or otherwise difficult to locate. Several large veins, including the subclavian, jugular, and femoral, have predictable relationships to easily identified landmarks and can be cannulated within minutes. Consequently, large vessel venipuncture has become a common practice in a variety of clinical settings, and it is a technique with which all physicians involved in the care of acutely ill patients should be comfortable.

### BACKGROUND

Subclavian venipuncture was described by Aubaniac in 1952, and its use was promptly supported by others. [1] [2] [3] Wilson and colleagues described the role of central venous pressure (CVP) monitoring in the maintenance of optimal blood volume and are credited with popularizing subclavian venipuncture in the United States. [4]

Numerous reports of clinical experience with the infraclavicular (IC) subclavian technique followed Wilson's article. These stressed the clinical usefulness of the procedure, the ease with which it is performed, and its low complication rate. Subclavian venipuncture was described as useful in the management of hypovolemia, burns, cardiac arrest, chronic IV therapy, and septic shock. [5] [6] [7] [8] [9]

Early enthusiasm for subclavian venipuncture was eventually tempered by a growing awareness of serious and occasionally fatal complications. [10] The reported complications of the IC approach suggested the need for a safer method. In 1965, Yoffa described the supraclavicular (SC) approach with its more direct approach to the subclavian vein and low complication rate. [11] Dronen and associates compared the 2 approaches during the performance of cardiopulmonary resuscitation (CPR) and found a significant decrease in catheter tip malposition and CPR interruption when the SC approach was used. [12]

An early mention of the internal jugular (IJ) approach was in a pediatric handbook by Silver and coworkers in 1963. [13] An IJ approach in the adult (with what later became known as the *central approach*) was described by Hemosura in 1966. [14] Subsequently a variety of approaches were described, ultimately to be grouped into the anterior,

central, and posterior approaches by Defalque in 1974. [15]

These techniques, as well as the femoral and cephalic-basilic approaches, have a place in the practice of emergency medicine, and often the choice of a particular approach is determined solely by the confidence of the individual physician in his or her ability to use the approach. In general, success rates are higher and complications less frequent when these techniques are used by more experienced technicians. [16] Although every clinician has a preferred method for achieving central venous access, any physician caring for critically ill patients should master several of these techniques.

## INDICATIONS

There are several commonly encountered clinical situations in which central venous access is indicated. If necessary, any central venous approach can serve the following functions. However, experience suggests that certain approaches offer advantages over others in most clinical settings. Advantages and disadvantages of each technique are outlined in Table 24-1 (Table Not Available) and discussed in detail after the general indications.

### Central Venous Pressure Monitoring

Although supplanted to a great extent by more sophisticated and accurate methods, particularly right heart catheterization with a balloon-tipped pulmonary artery catheter, CVP measurement remains a useful tool in the treatment of selected hypovolemic patients.

| **TABLE 24-1** -- Advantages and Disadvantages of Central Venous Access Techniques *From Knopp R, Dailey RH: Central venous cannulation and pressure monitoring. JACEP 6:358, 1977.* |
| --- |
| (Not Available) |

### Volume Loading

Subclavian venipuncture has been widely used as a vehicle for rapid volume resuscitation. Unfortunately, it is often misused in this regard. The flow rate of saline through a peripheral 5-cm, 14-ga catheter is roughly twice that through a 20-cm, 16-ga central venous catheter, with equivalent pressure heads. [17] The difference in flow is

even greater for blood products because of higher viscosity, which slows the passage of red cells through small-gauge catheters. [18] Consequently, the placement of peripheral large-bore catheters is the preferred method of rapid volume loading, unless time would be lost searching for a venipuncture site or unusually large volumes need to be infused.

Use of a large-bore (8 Fr) introducer catheter overcomes the impairment of flow seen with standard central venous catheters. Introducers offer flow rates that exceed even those of common IV tubing. [19] In trained hands this is a useful method of rapid fluid resuscitation, although it is associated with significant risk if the catheter is misplaced. Dutky and colleagues note that kinking of introducer catheters may halve their flow rate. [20] They found that kinking is common when long introducer catheters are placed via the IC route at the junction of the medial and middle thirds of the clavicle.

**Emergency Venous Access**

The predictable anatomic location of the subclavian, jugular, and femoral veins and the speed with which they can be cannulated (usually in 15 to 30 seconds) have prompted their use in cardiac arrest and other emergency situations. Although subclavian venipuncture by the IC route is an ideal means of rapid venous access, it may not be the best technique to use during CPR. Chest wall motion and physical interference with the performance of effective CPR make IC venipuncture more difficult and perhaps more dangerous in this setting. [12] The need for a central line during CPR is controversial, although many clinicians advocate use of a central line routinely. It has been suggested that therapeutic drug levels are reached more rapidly if given centrally. [21] [22] When easily obtained, central venous cannulation is preferred over peripheral venous access, because it provides a rapid and reliable route for the administration of drugs to the central circulation of the patient in cardiac arrest.

**Routine Venous Cannulation**

Drug abusers, burn victims, and obese or long-term care patients may have inadequate peripheral IV sites. Subclavian vein cannulation may be indicated under these circumstances.

**Routine Blood Drawing**

The potential complications of central venous cannulation do not justify its use in routine blood sampling. Lines already in place may be used for this purpose if they are properly cleared of IV fluid. A 20-cm, 16-ga catheter contains 0.3 mL

of fluid, so at least this much must be withdrawn to avoid dilution of blood samples. Because of the increased risk of infectious complications, air embolus, and venous backbleeding, the IV tubing should not be repeatedly disconnected from the catheter hub. Interposition of a three-way stopcock in the IV tubing simplifies access and is an acceptable method of blood sampling in the intensive care setting.

**Hyperalimentation**

In 1969, Dudrick and colleagues described the beneficial results of long-term parenteral nutrition in patients with various gastrointestinal disorders. Hyperalimentation by way of the subclavian vein was found to be safe and reliable. [23] Use of the IC technique frees the patient's extremities and neck; this procedure is therefore well suited to long-term applications. However, strict aseptic technique is necessary to minimize infectious complications. [24]

**Infusion of Concentrated Solutions**

Hyperosmolar or irritating solutions that have the potential to cause thrombophlebitis if given through small peripheral vessels are frequently infused by way of the subclavian vein. Examples are potassium chloride (>40 mmol/L), hyperosmolar saline, chemotherapeutic agents, and acidifying solutions, such as ammonium chloride.

**Other Indications**

Other indications for central venous access include placement of a pulmonary artery catheter or transvenous pacemaker, performance of cardiac catheterization and pulmonary angiography, and hemodialysis. Catheters such as the Uldall device can be inserted within minutes, permitting use of the subclavian vein for emergency or short-term hemodialysis. [25]

**Relative Indications for Different Approaches**

**Subclavian approaches.**

The IC subclavian approach is the most frequently used means of central venous access and is useful in most clinical settings. However, it is associated with a slightly higher incidence of complications. The SC approach is an important alternative to IC venipuncture, particularly in the setting of cardiac arrest. During CPR, the SC approach is often preferred because it minimizes physical interference with the functions of chest compression and airway management. The IC approach requires deep penetration of a moving chest wall and frequently demands an interruption of chest compression. An SC subclavian venipuncture can be performed without cessation of CPR and involves superficial penetration of the relatively motionless neck. [12] The technique also avoids interference with airway management, which commonly occurs when the internal jugular vein is cannulated. [26] When a true central venous location is required, the SC approach is superior to the IC and long peripheral line insertion techniques because of the low incidence of catheter tip malposition with the SC approach. [27] In addition, the SC technique has been performed in the sitting position in patients with severe orthopnea. Placement of a central line with the patient in a sitting position is virtually impossible with other central venous access routes. [28] Finally, the low complication rate reported for SC venipuncture makes it a more attractive alternative, especially in the seriously ill patient.

[26] [27] [28]

**Internal jugular approach.**

As is true of the SC subclavian approach, the IJ technique is useful for routine central venous access and for emergency venous access during CPR, since the site is removed from the area of chest compressions. [29] Comparison of IJ and subclavian cannulation has found a significantly greater incidence of proper venipuncture and catheter passage with the IC subclavian approach as compared with the posterior IJ method (98% vs 84%). [30] A 20% rate of catheter malposition was noted with each method. In 1 retrospective study, only 0.4% of 248 IJ cannulations resulted in clinically significant morbidity, compared with 4.2% of 298 subclavian insertions, even though the overall complication rate was similar. [31] Although there may be a slight difference in complications between the 2 routes, in the absence of specific contraindications, the physician should use the technique with which he or she is most familiar. The IJ route is slightly more technically difficult than the subclavian route but is faster and easier than a venous cutdown.

**Femoral approach.**

The cannulation of the femoral vein for central venous access has become increasingly popular in the last several years, especially for the passage of transvenous pacemakers and pressure measurement catheters in critically ill patients. [32] Mangiante and associates have had such success with femoral catheters for trauma patients that they have recommended that all hypotensive patients have a femoral line established with an 8.5 Fr Teflon catheter connected to genitourinary irrigation tubing immediately after 2 peripheral antecubital catheters are established. [33] They report no significant iliofemoral thrombosis, major hematoma, or infectious complications due to catheters. Other indications for urgent femoral cannulation include emergency cardiopulmonary bypass for resuscitation purposes, charcoal hemoperfusion for severe drug overdoses, and dialysis access. [34] Advantages of the femoral site over other central venous access sites are that the femoral area is less congested with monitoring and airway equipment than the head and neck area and that the conscious patient, who is still bedridden, may turn the head and use the arms without movement of the central line. Obviously, the femoral site is contraindicated in the ambulatory patient who requires central access.

**Other approaches.**

In instances where other methods of central venous catheterization are not possible, one may obtain access to the central venous circulation via the external jugular (EJ) vein or basilic-cephalic vein. [35] [36] Although generally accessible for peripheral IV access, the valves and tortuousity of these veins often preclude or delay placement of standard central venous catheters. Successful cannulation of the central venous circulation is generally possible only with the use of guide wires. The EJ vein must be visible for percutaneous cannulation to be successful. When time is available for a careful, deliberate attempt, these methods avoid the complications of pneumothorax, carotid or subclavian artery puncture, and hidden hemorrhage associated with other

methods of central venous cannulation.

The EJ approach can be used in both children and adults, but success is more common in adults. [36] [37] Central venous catheterization by the EJ route is technically more difficult than IJ cannulation, but it is successful 70-100% of the time in adults. [38] While use of a straight guide wire has been described, the use of a J wire is more reliable and is the preferred method. [37] The J wire is more easily advanced because its round tip bounces off vessel walls and navigates sharp angulations in the vessel course more easily.

When rapidity of access to the central venous circulation is not important, the basilic-cephalic route should be considered. This route has the lowest incidence of complications since the basilic and cephalic veins are located far away from vital organs and major arteries. When the patient is upright, the basilic vein is preferred over the cephalic vein because of a higher incidence of successful central catheter passage, although the overall success rate of superior vena cava cannulation is similar for both techniques in the supine patient. [39] Nonetheless, both veins have valves, which may impede catheter advancement. [40]

## CONTRAINDICATIONS

Contraindications to the various techniques of central venous access are shown in Table 24-2 . Most listed contraindications can be considered relative with clinical use of these techniques based on clinical conditions and available options for vascular access. Each technique in general is contraindicated in patients with distorted local anatomy or landmarks. Skin lesions such as local cellulitis, burns, abrasions, or severe dermatitis are relative contraindications to any access routes. Furthermore, use of any access technique with which an unsupervised clinician is inexperienced is contraindicated. Other relative contraindications include those conditions predisposing to sclerosis or thrombosis of the central veins, such as vasculitis, prior long-term cannulation, or illicit IV drug use via any of the deep venous systems.

**Subclavian Approach**

Patients in whom subclavian access is contraindicated include those who have undergone previous surgery or trauma

| **TABLE 24-2** -- Contraindications to Specific Central Venous Access Routes* |
| --- |
| General |

| |
|---|
| Distorted local anatomy |
| Extremes of weight |
| Vasculitis |
| Prior long-term venous cannulation |
| Prior injection of sclerosis agents |
| Suspected proximal vascular injury |
| Previous radiation therapy |
| Bleeding disorders |
| Anticoagulation or thrombolytic therapy |
| Combative patients |
| Inexperienced, unsupervised physician |
| Subclavian vein |

| |
|---|
| Chest wall deformities |
| Pneumothorax |
| Chronic obstructive pulmonary disease |
| Jugular vein |
| Intravenous drug abuse via the jugular system |
| Femoral vein |
| Need for patient mobility |
| Basilic-cephalic veins |
| Cardiac arrest § |
| Anticipated future use of these vessels |

\* Note that these contraindications are generally relative and clinical use of this technique must be based on clinical conditions and available options for vascular access.

May use contralateral side.

May use ipsilateral side.

§ May use standard (short) large-bore IV catheters.

involving the clavicle, the first rib, or the subclavian vessels; patients who have undergone previous radiation therapy to the clavicular area; patients with significant chest wall deformities; and those with marked cachexia or obesity. However, physicians in burn centers routinely place central catheters through burned areas. Patients with

unilateral deformities not associated with pneumothorax (e.g., fracture clavicle) should be catheterized on the opposite side.

Subclavian venipuncture is not contraindicated in patients who have penetrating thoracic wounds unless the injuries involve the superior vena cava. Generally, the vein on the same side of the chest wound should be cannulated to avoid the possibility of bilateral pneumothoraces, unless one suspects that the subclavian vessels have been injured. In such instances, the opposite side is cannulated. [41] In penetrating wounds that may involve the superior vena cava, neither subclavian vessel should be cannulated, and venous access below the diaphragm should be sought. Use of the subclavian approach in patients with coagulation disorders or in those receiving heparin therapy is contraindicated. A more visible and accessible site should be chosen (preferably percutaneous cannulation of a peripheral vein), because it is impossible to apply direct pressure to an oozing subclavian vein. The procedure should not be performed in combative patients because of the greater possibility of pneumothorax, vessel laceration, air embolism, and septic complications. Although subclavian venipuncture has been used successfully in children <2 years of age, [42] it is not generally recommended for use in small children. [43]

**Internal Jugular Approach**

Cervical trauma with swelling or anatomic distortion at the intended site of IJ venipuncture is the most important contraindication to the IJ approach. Neck motion is limited when the IJ central line is in place, and this limitation represents a relative contraindication in conscious patients. Although bleeding disorders are relative contraindications to central venous cannulation, the IJ approach is preferred over the subclavian route, as the IJ site is compressible. In the setting of severe bleeding diatheses, the femoral approach should be considered. Carotid arterial disease (obstruction or atherosclerotic plaques) is a relative contraindication to IJ cannulation, since inadvertent puncture or manipulation of the artery may dislodge a plaque. In addition, prolonged compression of the artery to control bleeding may impair cerebral circulation if collateral blood flow is compromised. If a preceding subclavian catheterization has been unsuccessful, the ipsilateral IJ route is generally preferred for a subsequent attempt. In this manner, bilateral iatrogenic complications are avoided.

**External Jugular Approach**

The greatest disadvantage of the EJ approach for central venous access is the time required to successfully pass a J wire and catheter into appropriate position. [44] However, the ease and relative safety of this approach make the EJ vein an excellent site of simple IV cannulation for fluid or drug administration or blood sampling.

**Femoral Vein Approach**

Contraindications to femoral cannulation include trauma to the groin, iliac vessels, or inferior vena cava. Percutaneous femoral line placement is not recommended in a

patient who

is in cardiac arrest or has an absent femoral pulse, unless other alternatives have been exhausted. [45] In a canine model of cardiac arrest, palpable pulsations in the groin were found to be venous rather than arterial in origin 50% of the time. [46]

## ANATOMY

### Subclavian System

The subclavian vein begins as a continuation of the axillary vein at the outer edge of the first rib (Fig. 24-1) (Figure Not Available) . It joins the IJ vein to become the innominate vein 3 to 4 cm proximally. The subclavian has a diameter of 10 to 20 mm and is valveless. After crossing the first rib, the vein lies posterior to the medial third of the clavicle. It is only in this area that there is an intimate association between the clavicle and the subclavian vein. The costoclavicular ligament lies anterior and inferior to the subclavian vein, and the fascia contiguous to this ligament invests the vessel. Posterior to the vein, separating it from the subclavian artery, lies the anterior scalene muscle, which has a thickness of 10 to 15 mm. The phrenic nerve passes over the anterior surface of the scalene muscle and runs immediately behind the junction of the subclavian and IJ jugular veins. The thoracic duct (on the left) and the lymphatic duct (on the right) pass over the anterior scalene muscle and enter the subclavian vein near its junction with the IJ vein. Superior and posterior to the subclavian artery lies the brachial plexus. The dome of the left lung may extend above the first rib, but the right lung rarely extends this high.

### Jugular System

The anatomy of the IJ vein is relatively constant, regardless of body habitus. The vein drains the cranium, beginning as the superior jugular bulb, which is separated from the floor of the middle ear by a delicate bony plate. The IJ vein emerges deep to the posterior belly of the digastric muscle. At its origin the IJ vein courses adjacent to the spinal accessory, vagus, and hypoglossal nerves, as well as the internal carotid artery. Several tributary veins enter the IJ vein at the level of the hyoid bone. The IJ vein, the internal (and, later, the common) carotid artery, and the vagus nerve course

**Figure 24-1** (Figure Not Available) *A* and *B,* Subclavian vein and local anatomy. *C,* Subclavian area, sagittal section. *( A from Linos D, Mucha P, von Heerden J: The subclavian vein: A golden route. Mayo Clin Proc 55:316, 1980. B from Davidson JJ, Ben-Hur N, Nathan H: Subclavian venipuncture. Lancet 2:1140, 1963. C from Brahos G: Central venous catheterization: SC approach. J Trauma 17:873, 1977. Reproduced by permission.)*

together in the carotid sheath. The IJ vein occupies the anterior lateral position in the carotid sheath. [47] The only structure that maintains a fixed anatomic relationship with the IJ vein is the carotid artery. The vein invariably lies lateral and slightly anterior to the carotid artery, and the course of the artery serves as a guide to venous cannulation. At the level of the thyroid cartilage, the IJ vein can be found just deep to the

sternocleidomastoid muscle (Fig. 24-2) (Figure Not Available) .

The IJ vein emerges from under the apex of the triangle of the 2 heads of the sternocleidomastoid muscle and joins the subclavian vein behind the clavicle. As the vein approaches its supraclavicular junction with the subclavian vein, it assumes a more medial position in the triangle formed by the 2 heads of the sternocleidomastoid muscles, following the anterior border of the lateral head. In this lower cervical region, the common carotid artery assumes a deep paratracheal location. The brachial plexus is separated from the IJ vein by the scalenus anterior muscle. The phrenic nerve is anterior to the scalenus anterior muscle. Although quite deep, the stellate ganglion lies anterior to the lower brachial plexus.

Unlike the subclavian vein, the IJ vein is quite distensible. The vessel diameter is increased with performance of a Valsalva maneuver and the assumption of the head-down (Trendelenburg) tilt position. Prolonged palpation of the carotid pulse will decrease the diameter of the IJ vein. [47] Rotating the head 90° toward the opposite side or extending the neck will not change the size of the IJ vessel significantly. Severe rotation of the head, however, will bring the sternocleidomastoid muscle anterior or medial to the IJ vein. Severe rotation may make it impossible to cannulate the IJ vein without first traversing the carotid artery when the anterior approach is used. The diameter of the IJ vessel is largest below the cricoid ring, where it may reach 2 to 2.5 cm. [47]

**Figure 24-2** (Figure Not Available) Structures in a dissection of the neck. The superficial veins and the sternocleidomastoid muscle have been removed, as have the submandibular gland and a segment of the facial vein. The cutaneous nerves have been cut down to short stumps arising from the second, third, and fourth cervical nerves. The internal jugular vein is drawn somewhat more medial in this illustration than is commonly found. *(From Hollinshead WH: Textbook of Anatomy. 3rd ed. New York, Harper & Row, 1974, p 765. Reproduced by permission.)*

**Femoral System**

The femoral vein is most easily cannulated percutaneously in patients with a palpable femoral pulse. The femoral vein lies just medial to the artery in the femoral canal below the inguinal ligament. Beneath the femoral vessels lie the psoas muscle and the hip (Fig. 24-3) (Figure Not Available) .

**Basilic and Cephalic System**

Considerable variation is present in the venous vasculature of the upper extremities. Nonetheless, the cephalic and basilic veins can usually be located in the volar antecubital region (Fig. 24-4) (Figure Not Available) . The interconnecting median antecubital vein is often the most prominent, thus making it a popular site for venipuncture during blood sampling. The basilic vein merges proximally with the brachial vein to form the axillary vein, which subsequently meets the cephalic vein to form the subclavian vein near the distal clavicle. The IJ and EJ veins join the subclavian vein to form the innominate vein bilaterally. Many venous valves exist in the peripheral vessels. Vascular anastomoses may permit aberrant advancement of a long line from the upper extremity. In particular, lines threaded up the cephalic vein may dead-end in a

venous plexus or enter the EJ vein. Furthermore, lines passed through the basilic vein may easily enter the IJ vein.

## EQUIPMENT

The materials required for central venous cannulation are listed in Table 24-3 . The catheter may be of the over-the-needle or through-the-needle variety, or it may be a component in a guide wire system that combines both technologies (see Chapters 20 and 21) .

**Figure 24-3** (Figure Not Available) The right femoral vessels and some of their branches. The femoral nerve (not shown) lies lateral to the artery and may be deep to the artery. *(From Warwick R, Williams PL [eds]: Gray's Anatomy. 35th ed. Edinburgh, Churchill Livingstone, 1973, p 676. Reproduced by permission.)*

**Figure 24-4** (Figure Not Available) Major veins of the upper half of the body. *(From Hedges JR: Vascular access. Curr Top Emerg Med 2:1, 1981. Reproduced by permission.)*

*Over-the-needle devices* (such as the Angiocath) use a tapered plastic catheter that passes through the vessel wall into the lumen using the needle tip as a guide (Fig. 24-5) . There are several advantages of this system. The catheter does not pass through a sharp needle, and the risk of shearing with resultant catheter embolization is thus decreased. The needle is removed following cannulation, making a guard unnecessary. The hole made by the needle in the vessel wall is smaller than the catheter, producing a tighter seal. Because these devices are placed via a percutaneous stick without the use of the guide wire technique, these catheters are used primarily when rapid central venous access is required (e.g., during a cardiac arrest). The catheters are not suitable for high-volume fluid resuscitation, and they are too small for passage of a pacemaker lead.

The main disadvantage of the over-the-needle system is the relatively short length of the catheter. Also, catheter threading is made more difficult by the longer length of the

| TABLE 24-3 -- Materials for Central Venous Cannulation |
| --- |
| 1 % lidocaine |
| 26-ga needle |
| 2-mL Luer-Lok syringe (for anesthetic) |

| |
|---|
| 10-mL non-Luer-Lok syringe (for catheter placement) |
| Swabs |
| Preparation solution |
| Gloves |
| Drapes |
| Catheter device |
| Intravenous tubing |
| Intravenous solution |
| Needle holder |
| 4-0 silk (or nylon) sutures |
| Suture scissors |
| Antibiotic ointment |

| |
|---|
| Gauze pads |
| Tincture of benzoin |
| Cloth tape |

**Figure 24-5** Most central venous catheterization uses the Seldinger technique, but for rapid access to the central venous system, the over-the-needle catheter/syringe system is ideal. The upper catheter is a 16-ga 5¼-in. Angiocath (shown assembled and attached to syringe and also shown disassembled) that can access the subclavian, internal jugular, and femoral vein in an adult. The lower 18-ga 1¼-in. Jelco catheter can be attached to a syringe to cannulate the central veins in an infant. The operator should advance the needle very slowly and aspirate continually. The flashback into the hub may be delayed, causing the needle to traverse the vein before the operator realizes that the lumen has been entered.

needle relative to the catheter. With over-the-needle catheters, the needle extends a few millimeters past the tip of the catheter. A blood return will be obtained when the tip of the needle is in the vein, but the catheter may actually be outside the lumen. If the needle is withdrawn before the catheter is advanced, the catheter tip will remain outside the vein. One must be certain to keep the needle steady and advance the catheter forward over the needle to ensure intravascular placement.

*Through-the-needle devices* (such as the Intracath) use a catheter of smaller gauge than the puncturing needle. Generally, the needle is 14 ga, and the catheter is 16 ga. This catheter is threaded through the lumen of the needle after the vessel is entered by the needle tip. After threading, the needle is withdrawn and left on the skin surface with a plastic guard protecting the patient and the catheter. A modification of this system uses a detachable hub on the catheter so that the needle can be removed. The main advantage of through-the-needle systems is that catheters of any length may be used. Also, the rate of successful entry of the catheter into the vessel lumen may be higher.

The main disadvantage of the through-the-needle system is the potential for catheter shearing during or after insertion. Systems using a detachable catheter hub (Deseret 755) allow the needle to be removed completely following insertion but require assembly of small plastic parts. This is undesirable in an emergency situation. Another disadvantage of through-the-needle devices is that the caliber of the catheter must be smaller than that of the needle. Standard catheters are 16 ga, a size that is not optimal for rapid infusion of resuscitation fluids or blood.

Newer types of catheters tend to combine through-the-needle and over-the-needle technology. The Argyle Intramedicut catheter has a short over-the-needle catheter that

serves

as an introducer for a longer (30 cm) infusion catheter. Following successful cannulation of the vein with the introducing catheter, the needle is withdrawn, and the infusion catheter is advanced through the introducing catheter.

Probably the most commonly used means of central venous cannulation today is the Seldinger (guide wire) technique, by which a thin-walled needle is used to introduce a guide wire into the vessel lumen (see Chapter 21) . A larger infusion catheter is then advanced over the wire into the vein. Physicians inexperienced with guide wire systems may encounter difficulty passing the guide wire. At times detachment of the syringe from the thin-walled needle may lead to loss of the needle's intravascular position. The need to detach the syringe can be eliminated by use of the Arrow Safety Syringe. This device incorporates a hollow syringe through which the guide wire can be passed directly into the thin-walled needle without detachment. In addition, this reduces the risk of air embolism, which can occur when the needle is open to the air.

There is no evidence that the complications of subclavian cannulation are related to the size of the puncturing needle. Schug and colleagues compared subclavian venipuncture performed with a standard through-the-needle device (Deseret Intracath 3162) to a system using a thin-walled needle and guide wire (Arrow AK 04300). They found no difference in the success or complication rates, but there was a significantly higher incidence of catheter misplacement or malfunction when the guide wire technique was used. [48]

Although more steps are required with the guide wire technique, it has several uses once it is mastered. Minor modification of the process and the equipment used permits the insertion not only of standard infusion catheters, but also of multilumen catheters, large-bore rapid infusion systems, and introducer devices. If this degree of flexibility is desired, use of Seldinger-type systems may be advantageous, despite their significantly greater cost.

Catheter length and size are also important considerations. The superior vena cava begins at the level of the manubriosternal junction and terminates in the right atrium, approximately 5 cm lower. For lines placed in the subclavian, jugular, basilic, and cephalic systems, the proper position of the catheter is in the superior vena cava, *not the right atrium or ventricle*. Therefore, the catheter should be threaded approximately 2 cm below the manubriosternal junction. One can estimate this distance by placing the catheter parallel to the chest wall before insertion. The standard catheters marketed for subclavian venipuncture are 20 to 30 cm long. The 30-cm catheters should not be inserted fully into the subclavian vein, because the catheter tip is likely to terminate in the right atrium or ventricle. Generally, 20 cm is an appropriate length for the average adult male, but one should consider advancing the catheter less than its full length in small adults. Proper placement is best confirmed by a postprocedure chest radiograph, not by preplacement measurements.

Central veins are often cannulated in seriously ill patients who will require subsequent pulmonary artery wedge pressure monitoring or transvenous pacemakers. Any CVP

catheter that is initially inserted should have a lumen that is large enough to accept a guide wire, should one need to later insert an introducer device for placement of the aforementioned devices. Not all commercially available CVP catheters will accept a guide wire through the lumen.

A number of different catheters are currently manufactured. It is best to use one brand routinely and to ensure that all medical personnel are thoroughly schooled in its use. Stocking several different catheter types adds unnecessary confusion to the duties of supply clerks and nurses, not to mention the physician who might be handed an unfamiliar brand during a patient resuscitation.

## VENIPUNCTURE TECHNIQUE

Strict adherence to the principles of sterile technique is important if septic complications are to be avoided. Violation of these principles for the sake of speed is seldom justified. The few extra seconds required to put on gloves and to swab and drape the chest will rarely make a critical difference in patient survival. It is recognized that optimum practice of aseptic technique often cannot take place during a resuscitation. For this reason, all central lines placed in this setting should be replaced at the earliest possible opportunity.

The area of the needle puncture should be widely prepared with povidone-iodine solution. In iodine-allergic patients, pHisoHex or Hibiclens are acceptable alternatives. If subclavian or IJ approaches are planned, the prepared area should include puncture sites for the IC and SC subclavian and IJ approaches. This permits the physician to change the site following an unsuccessful attempt without repeating the preparation. In this circumstance, a standard preparation should include the ipsilateral anterior neck, the supraclavicular fossa, and the anterior chest 3 to 5 cm past the midline and the same distance above the nipple line. Preparation for femoral access includes shaving the groin as needed, followed by application of povidone-iodine solution to cover an area the breadth of, and extending 10 cm above and below, the inguinal ligament.

Levy and associates described the virtual elimination of catheter contamination by inserting the catheter through a previously placed iodophor-impregnated sterile film after the usual sterile preparation and draping. [49] However, Clayton and colleagues found no statistical difference in catheter contamination rates during 137 insertions using an aseptic technique versus a "standard nonsterile" technique of insertion. [50] The patients and their skin were prepared similarly in both groups, except for the presence or absence of sterile towels for draping. Physicians in the aseptic group prepared as for surgery, with a 5-minute scrub and full gown, hat, mask, and gloves, whereas the "nonsterile" physicians did not necessarily even wash their hands or use sterile gloves. The contaminating organism in 87.1% of the overall cases was *Staphylococcus epidermidis.*

The need to perform an operating suite-style preparation in patients receiving hyperalimentation is unproved. In a study of 63 patients with long-term subclavian catheterization for hyperalimentation, Merk and Rush reported only 1 infection using a simple iodophor spray preparation. They emphasized that experienced personnel have

prime importance in the prevention of infection. [24]

Each of the approaches to central venous cannulation is described separately below. It is assumed that proper sterile procedure and any needed local anesthesia will be provided. As in any invasive task, the procedure should be described briefly to awake patients, and each step should be restated as it is about to be performed. After the following descriptions of the common approaches to the central veins, discussion

of catheter passage using the 3 catheter systems, puncture site care, placement verification, and other adjuncts to the procedure will be summarized.

**Subclavian Vein, IC Approach**

Descriptions of subclavian venipuncture often unduly focus on angles and landmarks and overstate the effects of patient positioning. The most important factors governing success or failure are knowledge of anatomy and meticulous attention to the details of the procedure. [51]

**Positioning.**

The patient is placed in the supine position with the head in a neutral position and the arm abducted. Placing the patient in the Trendelenburg position (10° to 15°) decreases the risk of air embolism. The claim that this position distends the vein is controversial at best. [52] Land has demonstrated by venographic studies that there is no change in the diameter of the subclavian vein associated with the Trendelenburg position. [53] Because his patients were normovolemic, this finding cannot necessarily be extrapolated to hypovolemic patients. Nonetheless, the vessel is hemmed by the semirigid costoclavicular ligament on its anteroinferior aspect and therefore will not distend in a direction that facilitates IC venipuncture. [11] Magnetic resonance imaging clearly demonstrates that the caliber of the subclavian vein is determined by its attachment to adjacent structures and is not affected by the CVP. [54] These findings are in conflict with those of Fortune and colleagues, who noted mild dilation of the subclavian vein on B-mode ultrasound of normal subjects during Trendelenburg positioning. [55]

Abduction of the arm has been recommended to flatten the deltoid bulge. [56] This is sometimes a helpful maneuver in muscular individuals but is not generally necessary. Land has demonstrated that abduction moves medially the point at which the subclavian vein passes beneath the clavicle. [53] Turning the head to the opposite side, as advocated by Borja, [57] has no effect on the vessel size or on the relative positions of the vessel and the clavicle. [53] This maneuver does change the relative positions of the subclavian and IJ vein and has been postulated to cause an increased incidence of catheter malposition in the jugular vein. [54] Placing a pillow under the back is commonly recommended to make the clavicle more prominent, but as the shoulder falls backward, the space between the clavicle and first rib narrows, making the subclavian vein less accessible. [56] Jesseph and colleagues demonstrated significant compression of the subclavian vessels between these bony structures as the shoulders retract. [54]

**Venipuncture site.**

The right subclavian vein is usually cannulated because of the lower pleural dome on the right and because of the need to avoid the left-sided thoracic duct. The anatomically more direct route between the left subclavian vein and the superior vena cava is a theoretic advantage of left-sided over right-sided subclavian venipuncture. It has not been proved that there is a higher incidence of catheter malposition when the right IC approach is used.

In the conscious patient, the point of needle entry is anesthetized with 1% lidocaine. SQ infiltration of the periosteum of the clavicle will make the procedure less painful but is not always necessary. When using a through-the-needle device, one attaches the 14-ga needle to a 10-mL non-Luer-Lok syringe. When using the Deseret Intracath 3162 system, it is advisable to remove the catheter from the plastic package to facilitate later passage through the needle (Fig. 24-6) (Figure Not Available) .

Opinions vary as to the best point of needle entry. The

**Figure 24-6** (Figure Not Available) *A,* Insertion of a through-the-needle device, as packaged. *B,* It is advisable to remove the packaged catheter and to puncture the vein with a syringe attached to the needle, then carefully remove the syringe and pass the catheter through the needle. Contrary to the illustration, latex gloves should be worn. *(Redrawn from Gallitano AL, Kondi ES, Deckers PJ: A safe approach to the subclavian vein. Surg Gynecol Obstet 135:97, 1972. Reproduced by permission.)*

junction of the middle and medial thirds of the clavicle is the standard site. There the vein lies just posterior to the clavicle and just above the first rib, which acts as a barrier to penetration of the pleura. This protective effect is lost when a more lateral location is chosen. Westreich advocates entry just laterally and inferiorly to the junction of the clavicle and the first rib, with the needle aiming at this junction. [58] Simon advocates entry at the site of a small tubercle in the medial aspect of the deltopectoral groove. [59] In our opinion, the point of entry is less important than the direction taken by the needle after entry. Points lateral to the midclavicle should be avoided, because this location requires a deeper puncture and potentially increases the risk of pneumothorax.

**Needle orientation.**

The *needle bevel should be oriented inferomedially* for the IC approach to direct the catheter toward the innominate vein rather than toward the opposite vessel wall or up the IJ vein (Fig. 24-7) . Alignment of the needle bevel with markings on the barrel of the syringe permits awareness of bevel orientation after skin puncture. Some investigators advise puncturing the skin with a No. 11 scalpel blade to avoid skin plugs in the needle. Others suggest filling the syringe with 2 to 3 mL of local anesthetic to both anesthetize the SQ tissue and flush the needle.

Before insertion of the needle, the left index finger is placed in the suprasternal notch and the thumb is positioned at the costoclavicular junction (Fig. 24-8) (Figure Not Available) . These serve as reference points for the direction of needle travel. The

needle is aimed immediately above and posteriorly to the index finger. Vessel entry, signaled by a flashback of dark venous blood, usually occurs at a depth of 3 to 4 cm. If the needle

tip is truly intraluminal, there will be free flow of blood. The return of pulsatile flow signifies arterial puncture. A single arterial puncture without laceration rarely causes serious harm. Use of this technique eliminates the need to measure angles, to "walk" the clavicle, or to concentrate excessively on maintaining the needle parallel to the chest wall. All of these techniques are based on fear of complications rather than knowledge of anatomy. [57] Mogil and colleagues have advocated changing the direction of the needle after it passes posteriorly to the lower edge of the clavicle. [9] In our opinion,

this adds a step that is not only unnecessary but also dangerous. One should avoid any sweeping motions of the needle tip to prevent unseen injuries. In patients who are being ventilated with positive pressure, it is advisable to halt ventilation for a moment as the needle penetrates the chest wall. Interruptions should be kept to a minimum and should not exceed the 30-second standard. [60]

**Unsuccessful attempts.**

Cannulation of the subclavian vein may not be successful on the first attempt. It is reasonable to try again, but after 3 or 4 unsuccessful attempts, it is best to try another approach or allow a colleague to attempt the procedure. One must use a new setup each time blood is obtained, because clots and tissue will clog the needle and mislead the physician even if subsequent procedures are performed correctly. If several attempts are made, the admitting physician or anesthesiologist must be informed so that proper precautions are taken to identify subsequent complications. It is advisable to obtain radiographs of the chest even after unsuccessful attempts. If the initial puncture site was properly placed, then for aesthetic reasons, one should use the same needle hole for subsequent attempts (i.e., one should avoid an embarrassing pincushion appearance of the upper chest). If the subclavian route is unsuccessful on one side, it is best to attempt an IJ catheterization on the same side rather than attempt a subclavian cannulation on the opposite side. In this manner, bilateral complications are avoided.

**Subclavian Vein, SC Approach**

**Positioning.**

The goal of the SC technique is to puncture the subclavian vein in its superior aspect just as it joins the IJ vein. The needle is inserted above and behind the clavicle, lateral to the sternocleidomastoid muscle. It advances in an avascular plane, away from the subclavian artery and the dome of the pleura. The right side is preferred because of the lower pleural dome, because it is the direct route to the

superior vena cava, and because the thoracic duct is on the left side. The Trendelenburg position may be helpful for distending the vein, because the subclavian vein is not bound by fasciae on its superior aspect. [27] The patient's head may be turned to the opposite side to help identify the landmarks.

**Needle orientation.**

After the area of the supraclavicular fossa has been prepared and draped, a point is identified 1 cm lateral to the clavicular head of the sternocleidomastoid and 1 cm posterior to the clavicle (Fig. 24-9) . The area is anesthetized with 1% lidocaine. If a 3-cm-long needle is used for anesthesia, it may also be used to locate the vessel in a relatively atraumatic manner. The subclavian vein can almost always be located with this needle because of the vein's superficial location and the absence of bony structures in the path of the needle. A 14-ga needle (or 18-ga thin-walled needle) is then advanced, following the path of the scout needle; gentle negative pressure is applied using an attached syringe.

When seeking the subclavian vein, the needle is aimed so as to bisect the clavicosternomastoid angle, with the tip

**Figure 24-9** *A* and *B,* For the supraclavicular approach, the needle is inserted above and behind the clavicle, bisecting the angle made by the clavicle and the lateral border of the sternocleidomastoid muscle (clavisternomastoid angle). The point of entry is 1 cm lateral to the clavicular head of the muscle and 1 cm posterior to the clavicle. The needle traverses an avascular plane, puncturing the junction of the subclavian and internal jugular vein behind the sternoclavicular joint. The right side is preferred because of a direct route to the superior vena cava and the absence of the thoracic duct. The needle is directed 45° from the sagittal plane and 10° to 15° upward from the horizontal plane, aiming toward the contralateral nipple. Note that the vein is just posterior to the clavicle at this juncture. *C,* Sagittal view of the supraclavicular area. As the subclavian vein passes over (and somewhat anterior to) the first rib, it is separated from the subclavian artery by the anterior scalene muscle. The dome of the pleura is posterolateral to the confluence of the great veins.

pointing just caudal to the contralateral nipple. The *bevel is oriented medially* to prevent catheter trapping against the inferior vessel wall. The tip of the needle is pointed 10° above the horizontal. Successful vessel puncture generally occurs at a depth of 2 to 3 cm.

### IJ Approach

**Positioning.**

In preparation for all 3 IJ approaches, the patient is tilted 15° to 30° in the Trendelenburg position and the head is turned slightly away from the side of venipuncture. The IJ vein is distensible, and tilting the patient increases the diameter of the vessel. If the patient is awake, he or she should be instructed to perform a Valsalva maneuver during vessel cannulation. In the unconscious patient, abdominal compression by an assistant can be used to help distend the vein.

**Venipuncture site.**

Familiarity with the anatomy of the neck is important to increase the probability of successful cannulation and to avoid complications. Most authors favor cannulation of the right side of the neck, which provides a more direct route to the superior vena cava and avoids the thoracic duct. Although it is probably clinically insignificant, the cupola of the pleura is also slightly lower on the right side. The left IJ approach is more circuitous and, when used with a stiff Teflon catheter, may result in a major venous puncture leading to hydrothorax, hydromediastinum, or even pericardial tamponade. [61] [62] [63] [64]

**Central route.**

This approach is favored by Kaplan and Miller, who believe that the incidence of cannulation of the carotid artery is decreased and the cupola of the lung is avoided with this method. [65] The triangle formed by the clavicle and the sternal and clavicular heads of the sternocleidomastoid is first palpated and identified. The lateral border of the carotid pulse can be marked by a local anesthetic skin wheal or a marking pen, and all subsequent needle puncture can be performed laterally to that point.

Some practitioners prefer to attempt cannulation with the catheter apparatus initially. Others use a small-gauge "locator" needle to locate the vein. The smaller needle allows one to ascertain the location of the vein and minimizes injury to deep structures by an incorrectly placed larger needle. Use of a locator needle can be time consuming in a cardiac arrest situation.

When using the scout needle technique, a 22-ga, 3 cm needle attached to a 5- to 10-mL syringe is introduced near the apex of the triangle and is directed caudally at an angle 30° to 40° to the skin. The needle should initially be directed parallel and slightly laterally to the course of the carotid artery (Fig. 24-10) (Figure Not Available) . If 3 fingers are lightly placed over the course of the carotid artery, the parallel course of the IJ vein can be estimated. The vein consistently lies just lateral to the carotid artery. Prolonged deep palpation of the carotid artery may decrease the size of the vein, and the 3-finger technique should be used only long enough to identify the course of the artery.

Negative pressure should be maintained on the syringe at all times as the needle is

advanced or retracted. The vein is more superficial than might be expected, and deep probing with the needle should be avoided; the vein is usually encountered at a depth of 1 to 1.5 cm. If the IJ vein is not entered at a depth of 3 to 5 cm, the needle should be withdrawn to just below the skin surface and directed toward the ipsilateral nipple underneath the medial border of the

lateral (clavicular) head of the sternocleidomastoid. *During any type of central venous cannulation, a needle must always be withdrawn to the surface before being redirected to avoid lacerating or otherwise damaging important nerves, vessels, or other structures*.

The vein should be entered at 1 to 3 cm, and dark blood should be easily aspirated (bright red blood indicates carotid artery penetration and the need for needle repositioning). The locator needle is withdrawn and replaced with a 14-ga, 5-cm needle attached to a syringe. A drop of blood from the locator needle can be placed at the edge of the sterile field in line with the point of vessel entry, thus serving as a guide to recannulation. The larger needle is advanced through the skin along the path determined by the smaller needle until blood is aspirated. Care must be taken to cover the needle hub with a gloved thumb whenever the needle lumen is exposed to air. This practice will prevent an air embolus when the patient inspires.

The central approach has been used in children with good success. [36] [66] However, success is greater with larger infants (>10 kg) and those with higher CVPs (>10 cm $H_2O$). With infants and children, needle puncture occurs at the apex of the triangle bordered by the 2 heads of the sternocleidomastoid muscle and the clavicle. Similar to the approach for adults, one passes a 22-ga needle attached to a 2- to 5-mL non-Luer-Lok syringe into the skin at a 45° angle and directs it caudally and laterally toward the ipsilateral nipple. The vessel is usually entered at a depth of 1 to 2 cm. The locator needle is then withdrawn, and a 17- to 19-ga needle is inserted into the skin until the IJ vein is penetrated.

**Posterior and anterior routes.**

For the *posterior approach*, the skin is entered at the lateral edge of the sternocleidomastoid muscle one third of the way from the clavicle to the mastoid process (Fig. 24-11) (Figure Not Available) . The locator needle is directed caudally and medially toward the sternal notch until blood is aspirated.

To perform the *anterior approach* described by Mostert

and coworkers, the course of the carotid is identified and marked by the index and middle fingers (Fig. 24-12) (Figure Not Available) . [67] The small needle should then enter the skin at the midpoint of the medial aspect of the sternocleidomastoid muscle. The needle is directed at an angle of 30° to 45° to the coronal plane caudally toward the ipsilateral nipple. Kaplan and Miller [65] alter this approach by starting at the level of the thyroid notch. The proximity of the carotid artery in the anterior approach may prohibit venous cannulation without

**Figure 24-12** (Figure Not Available) Anterior approach to the internal jugular vein. The index and middle fingers are outlining the course of the carotid artery. Contrary to the illustration, surgical gloves should be worn during the procedure. *(From Delfaque RJ: Percutaneous catheterization of the internal jugular vein. Anesth Analg 53:116, 1974. Reproduced by permission.)*

carotid puncture. [47] Legler and Nugent report the use of Doppler ultrasound to facilitate difficult IJ cannulation, a matter discussed in more detail below. [68] Use of ultrasound guidance is obviously restricted to nonemergency situations.

**EJ Vein Approach**

**Positioning.**

With the patient in the Trendelenburg position, the EJ vein is distended by instructing the patient to perform a Valsalva maneuver and then tamponading the vein just cephalad to the clavicle with a finger.

**Venipuncture.**

The vein is approached from the side while slight traction is placed on the vein to stabilize it. The needle is advanced at a small angle from the skin plane (about 10°) until the operator feels it "pop" into the lumen of the vein. The needle or catheter should be advanced slightly after feeling the pop to ensure intraluminal placement. As discussed below, use of the EJ vein as a site for central vein access requires the use of a guide wire.

**Femoral Vein Approach**

**Positioning and needle orientation.**

The patient must be supine. The femoral pulse is identified and the scout needle (at least 4.4 cm length) is inserted at 45° to the skin in a cephalic direction just medial to the femoral pulsation. Because CPR can produce venous pulsations, unsuccessful venous aspiration medial to the pulsations should be followed by an attempt directly over the pulsations.

During needle advancement, negative pressure is maintained within the syringe at all times while the needle is under the skin. The needle is directed posteriorly and advanced until the vein is entered, as identified by a flash of dark, nonpulsating blood. If the vessel is penetrated when the syringe is not being aspirated, the blood flash may be seen only as the needle is being withdrawn.

**Basilic and Cephalic Approach**

**Venipuncture.**

The basilic and cephalic venous systems are entered through the large veins in the antecubital fossa (Fig. 24-13) (Figure Not Available) . Tourniquet placement aids venous distention

**Figure 24-13** (Figure Not Available) Proximal anatomy of the basilic-cephalic venous system. *(From Webre DR, Arens JF: Use of the cephalic and basilic veins for introduction of central venous catheters. Anesthesiology 33:389, 1973. Reproduced by permission.)*

and initial venous puncture. When veins are not visible, they may be reached with a cutdown procedure, as described in Chapter 22 . The basilic vein, located on the medial aspect of the antecubital fossa, is generally larger than the radially located cephalic vein. Furthermore, the basilic vein generally provides a more direct route for passage into the axillary subclavian vein and superior vena cava.

## CATHETER PASSAGE TECHNIQUE

Once a venous flashback into the syringe is obtained, the syringe is detached from the needle, and a catheter is passed. Removing the syringe may be frustrating if the needle tip is dislodged from the lumen of the vein (Fig. 24-14) (Figure Not Available) . If the syringe is tightly attached to the needle, a hemostat may be used to grasp and secure the needle hub during removal of the syringe. Needle tip displacement may also occur if blood specimens are drawn at this time. Hence, it is best to delay blood sampling until the catheter has been advanced. The needle hub should be occluded with the thumb to avoid air embolism.

The technique used for catheter passage is dependent on the site of venipuncture and the needle-catheter system used. Many variations are available, but the most common

**Figure 24-14** (Figure Not Available) Subclavian venipuncture, venous backflow. After venipuncture, the syringe is removed carefully to avoid inadvertent motion of the needle tip, which may dislodge the needle from the vein lumen. A hemostat may help hold the needle hub to prevent motion during withdrawal of the syringe. *(From Phillips SJ: Technique of percutaneous subclavian vein catheterization. Surg Gynecol Obstet 127:1080, 1968. Reproduced by permission.)*

**Figure 24-15** (Figure Not Available) Internal jugular line secured by looping around the ear. *(From*

method uses the guide wire technique, described further in Chapter 21 . If a through-the-needle catheter is used, it should be emphasized that *at no time should a catheter be withdrawn through the needle or forced when it will not thread into the vessel easily*. A properly placed catheter should thread easily. Difficulty in catheter threading may be caused by passage out of the vessel lumen, trapping against the opposite vessel wall, kinking, or deviation up the IJ vein. *When the catheter will not thread, the needle and catheter should be withdrawn as a unit*; the vein should then be recannulated before reattempting catheter passage. Proper direction of the needle bevel will help alleviate catheter trapping.

Once the catheter has been passed, it is secured to the patient. All tubing and connections should first be checked for tightness to prevent air embolism, fluid loss, or bleeding. The technique for securing a catheter depends on the type of equipment and the site of puncture. In general, all catheters should be secured with sutures (or skin staples [69] ) and a sterile dressing placed. Most systems have some type of needle guard and/or anchoring device to simplify securing of the catheter. Since dressings are inspected and changed periodically, it is prudent to place a simple dressing, avoiding excessive amounts of gauze and tape. An effective method for securing an IJ catheter is shown in Figure 24-15 (Figure Not Available) . Care is taken to protect the skin against maceration if a plastic needle guard is used. Transparent dressings such as Op-Site are popular simple dressings.

**EJ Vein Approach**

Use of the EJ vein for achieving central venous access requires use of a guide wire. After cannulation of the vein and intraluminal placement of the guide wire, the guide wire is advanced into the thorax by rotating, teasing, or otherwise manipulating the tip into the central venous circulation (Fig. 24-16) (Figure Not Available) . Guide wire advancement is the most difficult and time-consuming portion of the procedure, and this time constraint limits the usefulness of the technique in an emergency. A small-radius J-tipped wire, a distended vessel lumen,

**Figure 24-16** (Figure Not Available) Insertion of a catheter over a wire via the external jugular vein. Successful passage may require many attempts and manipulations of the J-wire to navigate turns and valves. *(From Blitt CD, Wright WA, Petty WC: Central venous catheterization via the external jugular vein, a technique employing the J-wire. JAMA 229:817, 1974. Reproduced by permission.)*

and exaggeration of patient head tilt, coupled with skin traction, may facilitate successful guide wire passage. Partially withdrawing the wire and twisting it 180° before readvancing the tip may also be helpful.

Once the wire is in the correct position, a standard central venous catheter may be threaded over the wire, or a sheath introducer may be passed (with the aid of a vein dilator, as described in Chapter 21) to facilitate the introduction of a transvenous pacemaker or a pulmonary artery catheter. Even with central venous placement of the guide wire, the catheter may not pass centrally. After catheter placement, the guide wire

is removed, and the IV line is attached.

Central venous catheterization via the EJ vein is time consuming and often difficult. It also sometimes results in significant complications, especially with the use of the left EJ vein. [36] [62] For these reasons, it is not recommended in an emergency. Nonetheless, the EJ approach does provide central venous access in selected stable patients. Furthermore, simple cannulation with a short catheter is useful for fluid and drug administration during an emergency when peripheral veins cannot be cannulated.

**Femoral Vein Approach**

Once in the femoral vein, the needle is stabilized; often, a hemostat is helpful for holding the needle during removal of the syringe. A premeasured section of a 90-cm catheter may then be inserted using a through-the-needle system. One determines the appropriate length by holding the catheter over the patient's body and estimating the distance from the skin puncture site to the right atrium. Contamination of the catheter must be avoided while this maneuver is performed. Once the catheter is placed, it is secured with sutures and is dressed in the same manner as other central lines.

In situations requiring rapid volume infusion, in the absence of intra-abdominal trauma, the femoral vein may be cannulated with a sheath introduced via the guide wire technique. The introducer will allow rapid transfusion of large volumes of blood or crystalloid solution for fluid resuscitation. [33] Various catheters are available with single large-bore lumens or as many as 3 lumens for infusion of separate IV solutions and medications. The femoral vessels may also be cannulated under direct visualization using a cutdown technique (see Chapter 22) .

**Basilic and Cephalic Approach**

Once a vein has been entered, a premeasured length of a 90-cm catheter is threaded aseptically into the superior vena cava. Catheter length is estimated using the combined distance from the puncture site to the axilla and from the axilla to the middle of the manubrium.

Inability to pass the catheter is common. The cephalic vein may terminate inches above the antecubital fossa or bifurcate before entering the axillary vein, sending a branch to the EJ vein. The cephalic vein may also enter the axillary vein at right angles, defeating any attempt to pass the catheter centrally. Furthermore, both the basilic and cephalic systems contain valves that may impede catheterization. Abduction of the shoulder may help to advance the catheter if resistance near the axillary vein occurs. The incidence of failure to place the catheter in the superior vena cava ranges from a high of 40% to a low of 2%. [70] [71] This contention is not supported by Bridges and coworkers, who found an 80% to 84% success rate with slow catheter advancement without infusion versus a 44% success rate with crystalloid infusion in supine patients. [70]

The greatest success rate (98%) reported was obtained with slow catheter advancement with the patient in a 45° to 90° upright position. [70] Flexible Bard (C. R.

Bard, Inc., Murray Hill, New Jersey) (16-ga) catheters were introduced into the basilic vein until the tip was judged to be proximal to the junction of the cephalic and basilic veins and distal to the junction of the IJ vein with the innominate vein. The wire stylet was withdrawn 18 cm, and the catheters were advanced slowly 1 cm at a time, with 2 seconds allowed between each 1-cm insertion. The natural flexibility of the

Bard catheters contributed to negotiation into the superior vena cava when the patient was upright. Obviously, this time-consuming technique is contraindicated when the patient cannot tolerate an upright position.

## ASSESSING LINE PLACEMENT

Before the infusion of fluids, the IV fluid reservoir should be lowered below the level of the patient's right atrium and the line checked for backflow of blood. The free backflow of blood is suggestive, but not diagnostic, of intravascular placement. However, backflow may occur with a hematoma or a hemothorax if the catheter is free in the pleural space. A pulsatile blood column may be noted if the catheter has been inadvertently placed into the carotid artery. Less pronounced pulsations may also occur if the catheter is advanced too far and reaches the right atrium or ventricle. Pulsations also occasionally may be noted with changes in intrathoracic pressure due to respirations, although one hopes that such pulsations will be at a much slower rate than the arterial pulse! Finally, one may attach a syringe directly to the catheter hub to check for free aspiration of venous blood. Radiographs also are indicated to verify catheter location and assess for potential complications (see below).

### Radiographs

Following the procedure, the lungs should be auscultated to detect an inequality of lung sounds suggestive of a pneumo- or hemothorax. One should obtain a chest film as soon as possible, checking for hemothorax, pneumothorax, and catheter tip position. Because small amounts of fluid or air may layer out parallel to the x-ray plate with the patient in the supine position, the film should be taken in the upright or semiupright position whenever possible. Proper catheter tip position is shown in Figure 24-17 . Misplaced catheters should be repositioned.

### Redirection of Misplaced Catheters

Misdirection of the central venous catheter is not uncommon. A number of options are available to remedy this problem. Schaefer has described a novel technique in which a 2 Fr Fogarty catheter is inserted through the lumen of the central line and advanced 3 cm beyond the tip. The entire assembly is withdrawn until only the Fogarty catheter is in the subclavian vein. One milliliter of air is injected into the balloon, and the Fogarty catheter is advanced. It is hoped that the blood flow will direct the assembly into the superior vena cava. The balloon is deflated and the central line is advanced over the Fogarty catheter, which is then withdrawn. [72]

Other manipulations with guide wires have been suggested, but often reinsertion with

another puncture is required for the misplaced catheter to be positioned properly.

**ULTRASOUND-GUIDED CENTRAL VENOUS ACCESS**

In maximizing the success and minimizing the complications of central venous access, there is no substitute for experience. However, occasional patients will present venous access challenges to the most seasoned emergency physician. Anatomic

**Figure 24-17** A chest film showing the proper catheter tip placement in the superior vena cava *(arrow)*. The tip should *not* lie within the right atrium or the right ventricle.

(skeletal or vascular) abnormalities, whether congenital and acquired, may be encountered and can thwart successful cannulation. As noted above, when time permits, a Doppler ultrasound device can be used to identify the location of major veins. The course of these vessels can be marked on the skin surface and used as an anatomic guide during needle placement. Alternatively, more sophisticated ultrasound imaging systems have been adopted for guiding venous cannulation.

The most commonly described imaging tool is a hand-held 7.5 MHz real-time mechanical sector transducer with an attached needle guide. The device is coupled with a small video display (Fig. 24-18) (Figure Not Available) . To use the instrument, the nonsterile transducer is covered with acoustic coupling gel and placed inside a sterile sheath. Additional sterile gel is placed on the skin over the site being imaged, and the unit is used to determine the location, orientation, and diameter of the target vessel. When accessing the jugular or femoral systems, this is done by placing the transducer according to traditional puncture site landmarks. Imaging the subclavian vein from the IC approach is more difficult. Gualtieri and colleagues suggest identifying the axillary vessels at their most proximal position under the distal clavicle and then following the vessels medially as they course beneath the clavicle. [73] Once the vessel is identified, the overlying skin may be marked for later venipuncture or a needle and syringe secured to the transducer for immediate cannulation. Under ultrasound guidance, the needle is advanced through the skin and SQ tissue toward the target vessel. Once the needle is in close proximity to the vessel, one will see compression of the vein. Once the vessel wall has been penetrated, the vein will refill with blood and assume its original shape. The transducer can then be detached and cannulation proceed.

**Figure 24-18** (Figure Not Available) *A,* Surface ultrasound-directed central vein identification. A handheld transducer allows noninvasive localization of veins, in this case the subclavian. The device shown includes a needle guide, which allows simultaneous visualization and penetration of a targeted vessel. *B,* Surface ultrasound image of the subclavian artery *(left)* and vein *(right). (Courtesy of Irene R. Skolnick, Dymax Corp, Pittsburgh.)*

The majority of prospective analyses of the device have examined IJ vein cannulations [74] [75] [76] and have uniformly suggested advantages to the technique. Ultrasound-guided attempts demonstrated greater overall success, as well as an increased rate of successful first punctures. Clinically significant complications were too infrequent to definitely conclude greater patient safety with the ultrasound technique; studies specifically examining complications demonstrated less frequent hematoma formation

and inadvertent arterial puncture. [73] [74] Ultrasound guidance may assume greater prominence in central venous access in the future. However, given the generally favorable success rate and low complication rate of traditional access techniques, the relative infrequency of central venous cannulation in many emergency departments, and the considerable cost of the device, the role of ultrasound imaging guidance remains uncertain.

## COMPLICATIONS

The medical literature is replete with reports of the complications of large vein venipuncture. Common complications for the different approaches are summarized in Tables 24-4 and 24-5 (Table Not Available) . Key injuries categorized by organ system and by approach are discussed in the sections that follow.

### Pulmonary Complications

Pulmonary complications of subclavian and internal jugular venipuncture include pneumothorax, hemothorax, hydrothorax, hemomediastinum, hydromediastinum, tracheal perforation, and endotracheal cuff perforation. Pneumothorax is the most frequently reported complication, occurring in up to 6% of subclavian venipunctures. [77] Initially the importance of this complication was minimized, but reports of fatalities caused by tension pneumothorax, bilateral pneumothorax, and combined hemopneumothorax followed. [78] [79] One would expect a higher incidence of pneumothorax if the procedure were performed during CPR or positive-pressure ventilation. A small pneumothorax can quickly become a life-threatening tension pneumothorax under positive-pressure ventilation. Hemothorax may occur following subclavian vein or artery laceration, pulmonary artery puncture, or intrathoracic infusion of blood. Hydrothorax occurs as a result of infusion of IV fluid into the pleural space. Hydromediastinum is an uncommonly reported complication that is potentially fatal. [80]

### Vascular Complications

Air embolism is a potentially fatal complication from any central venous cannulation. Flanagan and colleagues determined that a 14-ga needle can transmit 100 mL of air per second with a 5-cm $H_2$ O pressure difference across the needle. [81] Air embolism may occur if the line is open to air during catheterization or if it subsequently becomes disconnected. The recommended treatment is to place the patient in the left lateral decubitus position to relieve air bubble occlusion of the right ventricular outflow tract. [82] If this is unsuccessful, aspiration with the catheter advanced into the right ventricle has been advocated. [83]

Catheter embolization resulting from shearing of the catheter by the needle tip is a serious and avoidable complication. This occurs when the catheter is withdrawn through the needle or if the guard is not properly secured. It is more likely to occur when using catheters that are not permanently affixed to the catheter hub. Complications occur frequently following embolization and include arrhythmias, venous thrombosis, endocarditis, myocardial perforation, and pulmonary embolus. [84] The mortality rate in patients who did not have these catheters removed has been reported to be as high as

60%. [85] Transvenous retrieval techniques are usually attempted and followed by surgery if they are unsuccessful. [86]

Perforation or laceration of vascular structures may cause hemothorax, hemomediastinum, and volume depletion. These are rarely serious complications, but fatalities

| **TABLE 24-4** -- Complications of Central Venous Access |
| --- |
| *General* |
| Vascular |
| Air embolus |
| Adjacent artery puncture |
| Pericardial tamponade |
| Catheter embolus |
| Arteriovenous fistula |
| Mural thrombus formation |
| Large vein obstruction |

| |
|---|
| Local hematoma |
| Infectious |
| Generalized sepsis |
| Local cellulitis |
| Osteomyelitis |
| Septic arthritis |
| Miscellaneous |
| Dysrhythmias |
| Catheter knotting |
| Catheter malposition |
| *Subclavian and internal jugular approaches* |
| Pulmonary |

| |
|---|
| Pneumothorax |
| Hemothorax |
| Hydrothorax |
| Chylothorax |
| Hemomediastinum |
| Hydromediastinum |
| Neck hematoma and tracheal obstruction |
| Tracheal perforation |
| Endotracheal cuff perforation |
| Neurologic |
| Phrenic nerve injury |
| Brachial plexus injury |

| |
|---|
| Cerebral infarct |
| *Femoral approach* |
| Intraabdominal |
| Bowel perforation |
| Bladder perforation |
| Psoas abscess |

have been reported. Surgical repair is occasionally required. [87] Arteriovenous malformation has also been reported. [88]

Perforation of the myocardium is a rare but generally fatal complication of central venous catheterization by any route. [89] The presumed mechanism is prolonged contact of the rigid catheter with the beating myocardium. [90] The catheter perforates the myocardial wall and causes tamponade either by bleeding from the involved chamber or infusion of IV fluid into the pericardium. The right atrium is involved more commonly than the right ventricle. [61] This complication can be prevented by determining the catheter tip position on a postinsertion chest film and repositioning improperly placed catheters. Cardiac tamponade can also occur with misplacement of the central venous line in the pericardiophrenic vein. [91] Premature atrial or ventricular beats may be observed if the catheter tip is in contact with the endocardium. Serious dysrhythmias do not occur, and the ectopic beats may be abolished if the catheter is repositioned.

Catheter knotting or kinking may occur if the catheter is forced or repositioned or if an excessively long catheter is used. [92] The most common result of kinking is poor flow of IV fluids. Johnson and Lazardchick reported a case of superior vena caval obstruction caused by a kinked catheter. [93]

Thrombosis and thrombophlebitis occur rarely because of the large caliber and high flow rates of the vessels involved. [57] It is important to determine that the catheter tip rests in the superior vena cava, especially during the infusion of irritating or hypertonic solutions.

[93] Thrombi may also form secondary to prolonged catheter contact against the vascular endothelium. Ducatman and colleagues reported a 29% incidence of mural thrombi in the innominate vein, superior vena cava, and right ventricle of patients who had central lines in place an average of 8 days before death. [94] However, no complications were directly attributable to these small, firmly adherent thrombi.

Thoracic duct laceration is a frequently discussed complication of left-sided subclavian venipuncture; however, it is extremely uncommon. McGoon and coworkers cite this as a complication of internal jugular cannulation but not subclavian cannulation. [77]

**Infectious Complications**

Infectious complications include local cellulitis, thrombophlebitis, generalized septicemia, osteomyelitis, and septic arthritis. [77] The incidence of septic complications varies from 0% to 25%. [95] In a retrospective audit, Herbst documented only 1 culture-proven infectious complication in 117 patients. [96] The frequency with which infectious complications are seen is directly related to the attention given to aseptic technique in insertion and aftercare of the catheter. One study suggested a higher incidence of contamination with triple-lumen catheters [97] ; however, Kelly and associates reported an acceptably low incidence of bacteremia and sepsis (3.1%) using this device. [98]

**Neurologic Complications**

Neurologic complications are extremely rare and are presumably caused by direct trauma from the needle during venipuncture. Brachial plexus palsy and phrenic nerve injury with paralysis of the hemidiaphragm have been reported. [99] [100]

**Miscellaneous Complications**

Improper catheter tip position occurs commonly. Conces and Holden reported that only 71% of subclavian catheters were located in the superior vena cava on the initial chest film. [101] Complications of improper positioning include hydrothorax, hemothorax, ascites, [102] chest wall abscesses, [103] embolization to the pleural space, [104] and chest pain. [105] More commonly, improper location yields inaccurate measurements of the CVP or is associated with poor flow caused by kinking. [106] An unusual complication caused by improper tip position is cerebral infarction. Hurwitz and Posner reported 2 patients who developed fatal cerebral infarcts following inadvertent cannulation of the subclavian artery. [107]

**Subclavian Approaches**

The claim by Moosman that the SC approach is more likely to be associated with complications is not substantiated by the medical literature. [56] In all reported series the complication rate has been low, ranging from 0% to 6%. [11] [28] [108] A compilation of the data from published series yields an overall complication rate of 1.3%. In a randomized prospective comparison of SC and IC venipuncture in 500 emergency department

patients, Sterner and colleagues reported complication rates of 2.0 and 5.1%, respectively. [109] The most significant complications have been pneumothorax and subclavian

---

**TABLE 24-5** -- Anatomic Structures That Can Be Injured by Central Venous Cannulation
*From Knopp R, Dailey RH: Central venous cannulation and pressure monitoring. JACEP 6:358, 1977.*

(Not Available)

---

artery puncture; the highest incidence of pneumothorax is 2.4%. [28] Adherence to Yoffa's technique [11] decreases the risk of these complications, because the needle is directed away from the pleural dome and subclavian artery. The relatively superficial location of the vein when approached from above the clavicle (1.5 to 3.5 cm) lessens the risk of puncture or laceration of deep structures.

Catheter tip malposition is also quite infrequent because of the more direct path to the superior vena cava. For those series in which malposition has been reported, the overall rate is 1.1%. [12] [27] [28] Fischer and coworkers noted a malposition rate of 27.6% using the IC technique. [110] The highest incidence of malposition using the SC technique is 7% and occurred during the performance of CPR. [12] In the same series, a 26% malposition rate was reported for the IC technique.

The incidence of failure to establish a functioning SC line ranges from 0% to 5%, [11] [27] [28] [111] with an overall rate of 4%. The failure rate reported for the IC technique ranges from 2.5% to 8%. [57] One death has been reported as a result of air embolism, a complication not specifically related to the SC technique. [111]

**IJ Approach**

Many of the complications of IJ cannulation are similar to those of subclavian access. Infection, catheter malposition, thrombosis, and damage to surrounding structures are complications common to all puncture sites for central venous cannulation. The reported rates of thrombosis for internal jugular vein catheterizations range from a report of no significant thrombosis in 1 study to a high of 66% of patients exhibiting some thrombosis in a study of 33 medical intensive care unit patients. [112] No reports of significant pulmonary embolus directly attributable to an IJ catheter were found. Such wide variation in the reported incidence of complications is common, in part because of the

different methods of detecting and reporting complications, variable experience

with the different techniques, and the different patient populations.

The number of complications increases, especially those due to thrombosis and infection, with longer duration of catheterization and increasing severity of the patient's illness. [16] Complications also seem to be higher with the use of the left IJ vein as opposed to the right. [47] [61] [62] Reported complications thought to be due at least in part to the use of the left-sided approach include mediastinal migration of the catheters and at least 1 instance of fatal pericardial tamponade.

One fairly common complication unique to the IJ approach is a hematoma in the neck. [32] With the IJ approach, pressure can be maintained easily on the area of swelling, and most hematomas will resolve spontaneously. If carotid arterial puncture is recognized and treated with compression, it rarely causes significant morbidity in the absence of marked atherosclerotic disease, although arteriovenous fistulas have been reported after IJ puncture. [113] Several neurologic complications unique to the IJ site of venipuncture have also been reported as a result of hematomas or of direct injury. [114] [115] These complications include damage to the phrenic nerves, an iatrogenic Horner syndrome, trauma to the brachial plexus, and even an instance of the passage of a catheter into the thecal space of the spinal canal. [116] [117] [118] [119] If the carotid artery is punctured, one may again attempt IJ or subclavian cannulation on the same side after appropriate, prolonged (15 to 20 minutes) compression.

Arterial puncture is a contraindication to attempting the IJ route on the opposite side, because bilateral hemorrhage may occur with resultant airway compromise. The physician should be prepared to rapidly intubate should this occur. Even in the face of a coagulopathy, however, the IJ approach has been found to be successful (99.3% of cases) and safe (<1% complication rate). [120]

**Femoral Approach**

Femoral lines are generally associated with less severe complications than are IJ or subclavian approaches because of the avoidance of thoracic trauma. The peritoneum can, however, be violated, with possible resulting perforation of the bowel. Bowel penetration is especially likely if the patient has a femoral hernia. [121] Injury to the bowel is likely to be minimal and is unlikely to require specific treatment. Nonetheless, the potential bacterial contamination of the femoral puncture site may pose a significant problem. Aspiration of air on placement of a femoral line necessitates removal of the catheter and reinsertion at another site. Bonadio and associates reported a case in which a patient developed clinical signs of peritonitis that were found to be due to infiltration of IV fluids into the anterior abdominal wall from a femoral catheter. [122]

A psoas abscess may result from penetration posteriorly of the underlying psoas fascia. [121] The bladder, when distended, can also be punctured during femoral cannulation, although bladder puncture is unlikely to require therapy beyond removal of the aberrantly placed catheter. Strict aseptic technique should be maintained to prevent septic arthritis in the unlikely event that the hip capsule is punctured. This complication

has been reported in infants. [123]

The most common complications of femoral venipuncture are inadvertent arterial and venous perforation. As mentioned in the Indications section, arterial puncture is more common in the patient who is in a low- or no-flow circulatory state. Emerman and colleagues reported that during cardiac arrest, the success rate for femoral catheterization was only 77%, compared with 94% for subclavian vein catheterization. [124] Prolonged (>15 minutes) pressure should stop any arterial hemorrhage in a patient with normal clotting mechanisms.

The femoral nerve can also be injured by an errant needle puncture. [120] [122] Complications can be minimized if the patient has a pulse and the femoral vein is approached medial to the femoral pulsations. A helpful mnemonic is NAVEL, which describes the anatomy of the region from lateral to medial: *n*erve, *a*rtery, *v*ein, *e*mpty space, and inguinal *l*igament. One study of 5306 femoral vein cannulations using the guide wire technique reported 5 severe complications (3 episodes of severe retroperitoneal bleeding after internal iliac artery puncture, 1 case of fatal femoral artery hemorrhage, and 1 reversible femoral nerve injury).

Getzen and Pollak, in a large study of military casualties, reported a 1.6% incidence of major hematomas, a 3.3% incidence of infection, and a 5.6% incidence of thrombophlebitis. [125] Thrombophlebitis does occur more frequently with femoral lines than with IJ and SC methods. [121] Thrombophlebitis is usually seen only with prolonged catheterization. There have been reports of pulmonary emboli in 2 of 24 patients that were thought to result from the thrombosis caused by prolonged (3 to 14 days) femoral cannulation. [126] Kanter and coworkers reported an 11% incidence of leg swelling or documented thrombosis in a series of pediatric patients. [127]

### Basilic-Cephalic Approaches

Cannulation of the central venous system through the arm veins has the lowest major complication rate of all. Superficial local infections are common (10% to 20% incidence) and rarely may lead to more serious problems, including sepsis. Catheter malposition is common, and studies have shown this to happen in 10% to 40% of placements. [128] [129] One nuisance of this type of catheter is the need to immobilize an entire extremity and the shoulder to prevent catheter movement and kinking.

## CONCLUSION

Cannulation of the central venous circulation is a necessary skill for emergency physicians. Safe application of the various techniques available requires detailed knowledge of anatomy and operative technique, as well as a healthy respect for potential complications. Inexperienced physicians should not undertake these techniques without supervision. While the IC subclavian approach is most commonly encountered in clinical practice, familiarity with the SC, IJ, femoral, and basilic-cephalic approaches will provide the emergency physician with a full complement of techniques

for gaining access to the central venous circulation under a variety of clinical demands.

## Central Venous Pressure Measurement

Although described by Forssman in 1931, it was not until the early 1960s that measurement of CVP became commonplace

as a means of assessing cardiac performance and guiding fluid therapy. [130] Clinically, CVP measurements are most frequently used as a guide for determination of a patient's volume status and fluid requirements and for investigation of tamponade. [131] [132] Critical commentaries have been written by some researchers who regard CVP monitoring as ineffective, outmoded, and unreliable. [133] The astute clinician, however, can maximize the usefulness of this diagnostic tool by careful consideration of its indications and limitations. CVP is one of many variables that must be correlated in the development of an overall management plan for the care of critically ill patients.

### PHYSIOLOGY

Simply stated, the CVP is the pressure exerted by the blood against the walls of the intrathoracic venae cava. Because the pressure in the great veins of the thorax is generally within 1 mm Hg of right atrial pressure, the CVP reflects the pressure under which blood is returned to the right atrium. [134] The pressure in the central veins has 2 significant hemodynamic effects. First, the pressure promotes filling of the heart during diastole, a factor that helps determine cardiac output. Second, the CVP is also the backpressure of the systemic circulation, opposing the return of blood from the peripheral blood vessels into the heart. [135] The CVP therefore affects both the ability of the of the heart to pump blood and the tendency for blood to flow from the peripheral veins. [26] [134] The CVP reading is determined by a complex interaction of intravascular volume, right atrial and ventricular function, venomotor tone, and intrathoracic pressure. [134]

One can measure CVP accurately by placing the tip of a pressure monitoring catheter into any of the great systemic veins of the thorax or into the right atrium. [135] Because the risks of catheter placement in the atrium include atrial perforation and cardiac dysrhythmias, any large vein within the thorax is preferred. [136] The catheter is commonly connected to an electronic pressure transducer interfaced with a monitoring system capable of calculating a mean pressure value and displaying pressure waveforms.

### INDICATIONS AND CONTRAINDICATIONS FOR CVP MEASUREMENT

The four major indications for CVP monitoring are as follows:

1. Acute circulatory failure
2. Anticipated massive blood transfusion for fluid replacement therapy

3. Cautious fluid replacement in patients with compromised cardiovascular status
4. Suspected cardiac tamponade

The procedure is contraindicated when other resuscitation therapeutic and diagnostic interventions take priority over central venous access and CVP transducer setup and calibration.

A common misconception is the incorrect assumption that CVP consistently reflects pressures found in the left side of the heart. The measurement that best reflects left ventricular pressure changes and reserve is the left atrial pressure, or the nearly equivalent pulmonary capillary wedge pressure (PCWP). The development of the flow-directed pulmonary artery catheter has allowed repeated measurements of PCWP, thus permitting accurate assessment of the left atrial pressure.

The CVP is most helpful in patients without significant preexisting cardiopulmonary disease. No consistent relationship between isolated CVP and left atrial pressure measurements has been demonstrated in patients with *significant cardiopulmonary disease.* Forrester and coworkers, in a study of 50 patients with myocardial infarction, demonstrated that CVP measurements had no consistent relationship to PCWP. [137] James and Myers studied 3 parameters--CVP, pulmonary arterial pressure, and PCWP--in 116 patients who either were in shock or had undergone major surgery. [138] In 76 of 116 instances, the PCWP differed significantly from the CVP. An early rise in the PCWP was noted before the rise in CVP. Samii and coworkers [133] studied 13 relatively elderly patients (mean age, 62 years) without obvious cardiopulmonary disease and found a disparity between the right and left ventricular filling pressures. They concluded that CVP may be a misleading index for predicting the PCWP in elderly patients.

Toussaint and associates reported a significant correlation between CVP and PCWP in 14 patients with no prior history of cardiopulmonary disease. [139] Yet in the same study, a poor correlation between CVP and PCWP was shown in 13 patients with a history of cardiopulmonary disease. Interestingly, Rajacich and colleagues found that CVP accurately predicted left atrial pressures in 17 cardiac bypass graft patients, even in the setting of high positive end-expiratory pressure. [140] Their patients had well-preserved cardiac function and normal valvular function. These findings suggest that CVP provides a reliable assessment of cardiac function only in the absence of cardiopulmonary disease.

**PROCEDURE**

Although CVP may be determined with a manometry column assembled at the bedside (Fig. 24-19) , the most common technique in practice is measurement with an electronic transducer interfaced to a monitoring system (Fig. 24-20) . Typical transducers include a nipple valve attached to a pressurized bag of saline to allow easy flushing of the system. To use these manometers, the transducer is attached to the patient's central line with a length of flexible yet fairly rigid-walled tubing filled with saline. A three-way stopcock is placed between the patient and the transducer to simplify line flushing and calibration.

All air bubbles are flushed from the system by opening the stopcock to air and flushing saline through the line. Air bubbles should not be flushed into the patient. Even tiny bubbles left in the tubing will dampen the CVP wave and potentially cause underestimation of venous pressure.

After the system has been flushed, the stopcock (with the transducer still open to air) is placed at the level of the patient's tricuspid valve. The monitor detecting the transducer's signal is then "zeroed," or calibrated. The transducer is calibrated at the level of the tricuspid valve, which can be approximated on the skin surface as a point at the midaxillary line and fourth intercostal space. [134] Finally, the stopcock

**Figure 24-19** *A,* Simple manometry column used to measure CVP at the bedside. The stopcock is turned to direct the flow to the patient, bypassing the manometer. This is the position that is maintained to keep the catheter patent. The tubing is always flushed before connecting it to the patient's central venous pressure catheter. *B,* The stopcock is turned to fill the manometer to 25 cm $H_2O$. *C,* The stopcock is opened to the patient, and the column of water in the manometer is allowed to fall and stabilize before a reading is taken. Note that the zero mark is horizontally aligned with the tricuspid valve (midaxillary line in a supine patient).

**Figure 24-20** General configuration of an intravascular pressure transducer. A working understanding of these devices, particularly as regards proper setup, zeroing, and line debubbling, will maximize their effectiveness and accuracy.

is set so that the transducer is in continuity with the patient's venous catheter.

In spontaneously breathing patients, readings should be taken at the end of inspiration of a normal breath. If the patient is receiving positive-pressure ventilation, the CVP changes during the respiratory cycle are reversed, rising with inspiration and decreasing with expiration. In these patients, readings should be taken near the end of expiration. [141] Thus, during both normal and mechanical ventilation, the lowest reading is a useful estimate of the mean CVP.

A reading may be taken after proper assembly of the equipment and after accurate placement of the tip of the catheter has been established. To ensure optimal measurement, the patient should be in the supine position. Whenever the patient is repositioned, care must be taken to ensure that the transducer has been recalibrated to reflect the new position of the patient.

## ERRORS IN CVP MEASUREMENT

A number of extrinsic factors may alter the accuracy of the CVP reading (Table 24-6) . [26] In addition to the position of the patient, these include changes in intrathoracic pressure, [142] catheter tip malposition, obstruction of the catheter, and failure to calibrate or zero the line. Activities that increase intrathoracic pressure, such as coughing or

straining, may cause spuriously high measurements. The patient should be

<table>
<tr><td align="center">**TABLE 24-6** -- Faulty Central Venous Pressure Readings</td></tr>
<tr><td>Increased intrathoracic pressure (ventilator, straining, coughing)<br>Reference points in error<br>Malposition of catheter tip<br>Blocking or ball-valve obstruction of catheter<br>Air bubbles in circuit<br>Readings during wrong phase of ventilation<br>Readings by different observers<br>Vasopressors (presumed)</td></tr>
</table>

relaxed at the time of the measurement and breathing normally. Positive-pressure ventilation raises the CVP reading by only 1 to 3 cm $H_2O$. If disconnection from the ventilator can be tolerated, the patient should be temporarily disconnected to provide a more reliable measurement.

Another reason for faulty readings is malposition of the catheter tip. If the catheter tip has not passed far enough into the central venous system, peripheral venous spasm or venous valves may yield pressure readings that are inconsistent with the true CVP. [26]

If the catheter tip is passed into the right ventricle, a falsely high CVP is obtained. Recognition of a characteristic right ventricular pressure waveform on the patient's monitor should hopefully preclude this error. Such fluctuations may occasionally be seen in appropriately positioned CVP lines when significant tricuspid regurgitation or atrioventricular dissociation (cannon a waves) are present.

Inaccurate low venous pressure readings are seen when a valve-like obstruction at the catheter tip occurs either by clot formation or by contact against a vein wall. As mentioned above, wave damping due to air bubbles in the transducer or tubing also leads to faulty readings.

Using poorly zeroed lines may result in inaccurate measurements that may be interpreted as a change in the patient's status when none has actually occurred. The transducer should be zeroed to the same level for every measurement.

Some investigators mention a falsely elevated CVP in patients who are receiving vasopressors, but controlled data on this aberration are lacking. One animal study suggests that fluid can be infused into 1 lumen of a multilumen catheter without affecting the CVP reading at another lumen. [143]

**INTERPRETATION OF THE CVP MEASUREMENT**

Because determination of the CVP can aid the clinician in assessment of the critically ill patient, it is paramount that the clinician know the normal values and the variables that may affect these values and recognize the pathologic conditions that correlate with abnormal values.

Although early articles reported varying normal ranges for CVP measurements, recent cardiac catheterization studies have demonstrated that the normal range extends from -2 to +7 cm $H_2O$.

Gowen reports from his clinical experience that a reading of 7 cm $H_2O$ is the upper limit of normal, and a reading of 8 to 10 cm $H_2O$ is a borderline elevation, whereas levels >12 cm $H_2O$ are consistent with impending heart failure. [144] Weil and coworkers, however, described the normal CVP as ranging from 2 to 10 cm $H_2O$ and considered a high CVP to be >15 cm $H_2O$. [132] Knobel and associates noted that a CVP <6 cm $H_2O$ is rarely associated with a PCWP >12 cm $H_2O$ in septic patients. [145] The following guideline reflects the consensus of the literature:

*Low:* <6 cm $H_2O$

*Normal:* 6 to 12 cm $H_2O$

*High:* >12 cm $H_2O$

In the late stages of pregnancy (30 to 42 weeks), the CVP is physiologically elevated, and normal readings are 5 to 8 cm $H_2O$ higher in pregnant than in nonpregnant women.

A CVP reading <6 cm $H_2O$ is consistent with low right atrial pressure and reflects a decrease in the return of blood volume to the right heart. This may indicate that the patient requires additional fluid or blood. A low CVP reading is also obtained when vasomotor tone is decreased, as in sepsis, spinal cord injury, or other forms of sympathetic interruption. [132] [144] [145]

A CVP reading falling within a normal range is viewed in relationship to the clinical situation. A reading >12 cm $H_2O$ indicates that the heart is not effectively circulating the volume presented to it. This situation may occur in the case of either a normovolemic patient with cardiac decompensation or a patient with a normal heart who is overhydrated and overtransfused. A high CVP is also related to variables other than pump failure, which include pericardial tamponade, restrictive pericarditis, pulmonary stenosis, and pulmonary embolus. [144]

Changes in blood volume, vessel tone, and cardiac function may occur alone or in combination with one another; therefore, it is possible to have a normal or high CVP in the presence of normovolemia, hypovolemia, and hypervolemia. One must interpret the

specific CVP values with respect to the entire clinical picture: the response of the CVP to an infusion is more important than the initial reading.

**Fluid Challenge**

Monitoring of the CVP may be helpful as a practical guide for fluid therapy. [131] [132] Serial CVP measurements provide a fairly reliable indication of the capability of the right heart to accept an additional fluid load. Although the PCWP is a more sensitive index of left heart fluid needs (and in some clinical situations, PCWP measurement is essential), serial measurement of CVP can provide significant information.

A fluid challenge can help assess both volume deficits and pump failure. Although a fluid challenge can be used with either PCWP monitoring or CVP monitoring, only the fluid challenge for CVP monitoring is discussed here. Slight variations in methodology of fluid challenge are reported in the literature. Generally, aliquots of 50 to 200 mL of crystalloid are sequentially administered, and measurements of CVP levels are obtained after 10 minutes. [111] [144]

The fluid challenge is generally carried out in the following manner [132] : Fluid is administered by a route other than that used for monitoring. An initial CVP reading is taken, and fluid is infused at a rate of 20 mL/min over a 10-minute period. The infused volume is allowed to equilibrate for 10 minutes, and a reading is taken. If the CVP is >5 cm of $H_2O$ over the initial measurement, the fluid challenge is discontinued, and one assumes that the right ventricle is unable to handle an additional fluid load. Increases of between 3 and 5 cm $H_2O$ over the initial CVP value are equivocal, and additional measurements are taken over the next 30 minutes if this reading is obtained. Increases of <2 cm $H_2O$ over the original reading or a return of higher readings to this level within 30 minutes is indicative of volume depletion. The fluid challenge is repeated until measurements indicate that adequate volume expansion has occurred. The fluid challenge is discontinued as soon as hemodynamic signs of shock are reversed or signs of cardiac incompetence are evident.

**Cardiac Tamponade**

In cardiac tamponade, pericardial pressure rises to equal right ventricular end-diastolic pressure. The pericardial pressure encountered in pericardial tamponade characteristically produces an elevated CVP. The degree of CVP elevation is variable, and one must interpret measurements cautiously; CVP readings in the range of 16 to 18 cm $H_2O$ are typically seen in acute tamponade, [132] [146] but elevations of up to 30 cm $H_2O$ may be encountered. The exact CVP reading is often lower than one might intuitively expect, and it is not uncommon to encounter tamponade with a CVP of 10 to 12 cm $H_2O$. A normal, or even low, CVP reading may be seen if the tamponade is associated with significant hypovolemia.

An excessive rise in CVP following fluid challenge may be more important than a single reading in the diagnosis of pericardial tamponade. It is interesting to note that Shoemaker and associates [146] reported a decrease in CVP just before cardiovascular

collapse in patients with pericardial tamponade.

Excessive straining, agitation, pneumatic antishock garment inflation, positive-pressure ventilation, or tension pneumothorax may increase intrathoracic pressure, producing a high CVP reading, and may erroneously suggest the diagnosis of pericardial tamponade. Increases in vascular tone, as seen with the use of dopamine or other vasopressors, may also elevate the CVP, mimicking tamponade and complicating volume estimation.

**CONCLUSION**

CVP monitoring provides useful hemodynamic monitoring information in those individuals with a relatively normal cardiopulmonary system who do not otherwise warrant PCWP monitoring.

# Chapter 25 - Accessing Indwelling Lines

**John M. Howell**

Many therapies require the use of intravenous (IV) catheters that afford indwelling access to the central circulation. Indwelling central venous access is essential to those who require lengthy chemotherapy, [1] [2] hyperalimentation, [3] [4] extended antimicrobial therapy, [5] and hemodialysis. [6] [7] [8] An ever-increasing number of individuals care for these devices as outpatients. More than 500,000 indwelling venous access devices are placed annually in the United States. [9] These devices make indwelling lines available to emergency department staff to draw blood and administer medications, blood products, and IV fluids with minimal patient discomfort. Furthermore, an awareness of attendant complications should help minimize the risk of line sepsis and central vein thrombosis.

## BACKGROUND

### Historical Features

In 1973, after unsuccessful efforts with arteriovenous shunts, Broviac designed an IV catheter that afforded prolonged access for hyperalimentation patients. [3] This Silastic catheter was 90 cm long and passed through the subclavian vein to the mid-right atrium. Broviac's initial description fashioned a template for several products in use today.

Hickman altered Broviac's prototype in 1979 [1] for the treatment of leukemia patients receiving chemotherapy (Fig. 25-1) . His modified catheter was larger in diameter and lacked a surrounding sheath. This bigger lumen facilitated the drawing of blood as well as the infusion of chemotherapeutic agents, blood, and IV solutions. More recent approaches to the dilemma of prolonged access include multilumen external catheters [10] and surgically implanted subcutaneous infusion devices (e.g., Port-A-Cath, Infuse-A-Port, and MediPort). [11] These products are beneficial in that they obviate the need for multiple peripheral IV lines; however, their use may lead to complications. Wound sepsis and central venous thrombosis are among the more common difficulties encountered by patients tending their catheters at home. [5] [12] Timely recognition of these complications may avert morbidity in a population composed largely of immunocompromised individuals.

### Patient Populations with Indwelling Lines

Central venous and right atrial catheters are commonly placed in patients with cancer to deliver chemotherapy and to prevent tissue infiltration of these destructive chemicals. [11] [13] They are useful in treating both hematologic and solid malignancies and have been placed to facilitate daily radiation therapy of brain tumors in children. [14] Many patients opt for local care at home and return only periodically to a clinic or hospital for IV chemotherapy.

Otherwise healthy individuals with soft tissue or bony infections receive IV antimicrobials

in the same manner. Alternative indications include ambulatory heparin therapy [15] and parenteral hyperalimentation, [3] especially in those with short bowel syndrome and malnutrition. [4] McDowell and colleagues [7] and Schanzer and associates [8] reported good results using double-lumen, cuffed central venous catheters as an alternative to arteriovenous shunts in patients who need long-term hemodialysis.

Although the predominant indication for arteriovenous fistulas and shunts is hemodialysis, [15] these devices have also been used for parenteral hyperalimentation and chemotherapy. [16] However, right atrial catheters are more widely accepted for parenteral hyperalimentation and chemotherapy owing to their ease of insertion and the ability of patients to self-administer medications at home. Subclavian Uldall [17] [18]

**Figure 25-1** Hickman double-lumen catheter (12 Fr), injection caps (2), attached clamps (2), sheath introducer with vessel dilator, syringe, and 33-cm tunneler.

or Manhurkar catheters (also called *Quinton* catheters) are frequently positioned to deliver hemodialysis for up to 8 weeks while a peripheral fistula matures.

## INDWELLING CATHETER, SHUNT, AND FISTULA DEVICES

The majority of techniques designed to allow prolonged IV access form the following categories: long-term venous access catheters; implantable venous access devices; percutaneous multilumen catheters; and methods to deliver hemodialysis, including arteriovenous shunts, fistulas, and specific catheters.

### Long-Term Venous Access Catheters

Long-term venous access catheters are made by several manufacturers and include Broviac, Hickman, Leonard, Raaf, and Hermed catheters (see Fig. 25-1) . These Silastic catheters are between 65 and 90 cm long and have 1, 2, or 3 lumina. They are positioned in the right atrium either during an operation or at the bedside. [7] [11] [15] Access to the central circulation is achieved by various methods, including cephalic, [19] external jugular, [14] [15] saphenous, [15] internal jugular, [14] brachiocephalic, [20] subclavian, [15] [21] and translumbar [4] approaches. They are generally tunneled subcutaneously from the venous access point to a position on the anterior chest wall. Many kits include a 33-cm tunneler to pull catheters through subcutaneous (SQ) tissues. A Dacron cuff is generally positioned midcatheter to anchor and prevent the spread of infection. [15] CR Bard, Inc. (Salt Lake City, Utah) produces a catheter (VitaCuff) with two SQ cuffs to release silver ions that block the inward spread of microorganisms.

Two kinds of Silastic, long-term venous access catheters are neither tunnelled nor cuffed. [22] Long-term, peripherally inserted central catheters (PICCs) are placed in the brachium and terminate in the superior vena cava. They are positioned either at the bedside or under fluoroscopy. Short-line central venous catheters are placed directly into the subclavian vein. One study identified a $7 million annual savings for the insertion and maintenance of nontunneled Silastic catheters compared with tunneled

catheters. [22]

Manufacturers produce right atrial catheters in various lengths and diameters. [15] Single-lumen Broviac catheters are popular for children and vary in size from 2 to 6 Fr. Many Hickman catheters provide a double-lumen alternative up to No. 19 Fr. Weese and Trigg [23] produced a triple-lumen variation by placing Hickman and double-lumen Raaf catheters through the same venotomy. Individual lumen widths in double-lumen devices vary between 0.7 and 1.6 mm. Repair kits are available (Evermed, Inc.) that include silicone adhesive and a 12 Fr catheter segment (Fig. 25-2) .

Home care of long-term venous access catheters is generally performed on a daily basis. [15] The skin site is cleansed with alcohol and povidone-iodine before applying a sterile dressing (e.g., Tegaderm). Two to 3 mL of 100 units/mL heparin are then flushed to prevent catheter thrombosis. [15] [24] [25] The injection cap is changed weekly after first clamping the catheter. [15] Breaks in sterile procedure or overlooking the heparin flush may result in thrombosis or line sepsis.

Groshong catheters (CR Bard, Inc., Salt Lake City, Utah) are similar to Broviac and Hickman catheters in that Groshong

**Figure 25-2** Hickman catheter repair kit, with tube of adhesive, 18-ga needle, plastic syringe, injection caps (2), Luer-Lok caps (2), and external catheter segment with attached clamps.

lines are Silastic with an SQ cuff and up to three lumina. The catheter tip is closed, and there is a pressure-sensitive, two-way valve on the adjacent lateral wall (Fig. 25-3) (Figure Not Available) . There is no backflow of blood into the catheter because venous pressure maintains the valve in a closed position. Consequently, clamping and frequent heparin flushing are not necessary. The externalized part of the catheter is removable, making insertion and replacement simpler. Groshong catheters are three to seven times more likely than Hickman catheters to suffer valve-related complete catheter malfunction. [26]

**Implantable Venous Access Devices**

Implantable venous access devices (Port-A-Cath, Infus-A-Port, MediPort, MRI Port, and Norport) are surgically implanted

**Figure 25-3** (Figure Not Available) The Groshong catheter tip. The Groshong has a patented three-way valve that opens outward for infusion *(A)*, opens inward for aspiration *(B)*, and remains closed when not in use *(C). (Courtesy of Bard Access Systems, Inc., Salt Lake City, Utah.)*

circular, SQ chambers in continuity with a central Silastic catheter (Fig. 25-4) . [11] [15] [27] [28] All of these products use metallic chambers except the MRI Port, which is safe for magnetic resonance imaging (MRI) examinations. The major difference between this and other systems is that access to implantable venous access devices is obtained by inserting a tapered Huber needle through the skin into a diaphragm approximately 7 to 11 mm in diameter (Fig. 25-5) (Figure Not Available) . [15] The chamber is palpable as

well as visible on the anterior chest wall. Catheter widths are between 2.2 and 2.8 mm. [15]

In contrast to long-term venous access catheters, implantable venous access devices are accessed through a 19- or 22-ga tapered (Huber) needle to limit cylinder damage. [15] Although this technique allows approximately 2000 punctures, [15] it also hinders rapid delivery of blood and blood products. Implantable venous access devices are initially more expensive than long-term venous access catheters: however, they also require less frequent home care. Weekly or monthly [15] [24] flushing with 4 to 5 mL of heparin (1000 units/mL) prevents thrombosis.

## Percutaneous Multilumen Catheters

Triple-lumen catheters (Arrow) are inserted percutaneously into the central circulation to allow phlebotomy and simultaneous delivery of medication and blood products. They are approximately No. 7 Fr in caliber and deliver these substances through 16- and 18-ga lumina. Drawbacks include daily wound care, heparinization, and relative brevity of use. For a triple-lumen catheter, changing the line over a guide wire increases the risk of bacteremia, although moving the triple lumen to another site increases the frequency of mechanical malfunction. [29] Multilumen catheters are usually left in place <3 weeks, [10] [15] but extended use may approach several months. [15] In contrast, Hickman catheters have been left in place for 474 days, [30] and Port-A-Cath catheters up to 351 days. [27]

**Figure 25-4** Tapered Huber needle, Port-A-Cath reservoir, and catheter segment.

**Figure 25-5** (Figure Not Available) Port-A-Cath system. The Port-A-Cath system is accessed by inserting a Huber needle through the skin and portal septum. *(Courtesy of Pharmacia Nutech, Pharmacia Laboratories, Piscataway, New Jersey.)*

## Implantable Venous Access Pump Devices

Internal pump devices (Infusaid, Pfizer Hospital Products Group, Norwood, Massachusetts; SynchroMed, Medtronics, Minneapolis, Minnesota) deliver medications from the pump through a catheter into central veins, the spinal canal, and selected vessels near target organs or tumors (Fig. 25-6) (Figure Not Available) . Some devices are programmable for specific infusion rates; others are not. The catheter is accessed at its pump attachment for emergency infusion and blood drawing. Like a shunt or fistula, these devices are not used for routine, nonemergent indications.

## Arteriovenous Shunts, Fistulas, and Catheters

Arteriovenous shunts are external Silastic bridges between the arterial and venous circulations. When placed in extremities, they facilitate hemodialysis during acute renal insufficiency and failure. [15] However, subclavian and femoral vein access have recently subverted the use of these shunts in intensive care units during management of hemorrhagic shock and sepsis. In addition, central venous catheters are now used

frequently as a temporizing measure until permanent arteriovenous access sites mature. Consequently, emergency physicians are less likely to encounter arteriovenous shunts in their daily routine.

Peripheral arteriovenous fistulas, on the other hand, represent the procedure of choice for permanent vascular access in end-stage renal disease. [15] They are produced by anastomosing the peripheral venous and arterial circulations. In this way a superficial, dilated vein serves as a permanent access site for large-bore hemodialysis needles. Vessels commonly used for fistulas are the radial artery and cephalic vein or the ulnar artery and basilic vein. [15] Patent fistulas, generally located near the wrist, are easily discerned by their characteristic bruit and thrill. Pathologic arteriovenous

**Figure 25-6** (Figure Not Available) Implantable infusion pumps are available that allow for infusion of a medication at a constant rate or at a variable or programmable rate. They are normally placed in a subcutaneous pocket either in the abdomen or anterior chest wall. Two examples are shown. The Infusaid M400 Constant Flow Implantable Pump (Pfizer, Norwood, Massachusetts) *(A)* provides for a constant infusion rate of a medication placed transcutaneously into its drug chamber. It also has a sideport for direct access to the catheter for direct, percutaneous injections or IV therapy using the patient's central venous catheter attached to the device. The SynchroMed Infusion Pump (Medtronic, Minneapolis, Minn) *(B)* can be programmed by a computer through a radio transmitter for a wide variety of infusion rates. *(Courtesy of Pfizer Hospital Products Group, Norwood, Massachusetts).*

fistulas have been inadvertently produced by penetrating vascular injury [31] and superficial phlebotomy. [32]

Large-bore Uldall catheters [17] [18] are generally placed in the subclavian vein and provide hemodialysis access for up to 8 weeks while a peripheral arteriovenous fistula site matures. These double-lumen catheters are tunneled subcutaneously and lack the Dacron cuff present on most central venous access catheters. They are capped after each dialysis treatment with approximately 2500 units of heparin in each luminal arm.

Manhurkar (Quinton) catheters (Quinton Instrument Company, Seattle) are double-lumen lines inserted percutaneously into the subclavian or femoral vein for short-term dialysis access. Following insertion, each arm is flushed with 10 mL of normal saline. Subsequently, 1500 units of heparin (e.g., 1.5 mL of 1000 units/mL) are instilled into each arm.

## INDICATIONS AND CONTRAINDICATIONS

### Emergency Department Access

Indications for accessing right atrial and multilumen catheters in the emergency department include phlebotomy and administration of IV fluids and medications. In the emergency department, arteriovenous fistulas, shunts, and Uldall and Manhurkar catheters are accessed only in true emergency situations, because infection and shunt malfunction may result and necessitate additional surgical procedures to obtain access for hemodialysis. The emergency physician may be called on to free occluded lines or repair leaking external catheters; these procedures are discussed under Complications below. Right atrial and multilumen catheters should not be accessed in the emergency

department if patency cannot be demonstrated using the maneuvers described under Procedures.

**Medication Issues**

Medications that are known to be incompatible when mixed (e.g., calcium and bicarbonate) should not be administered concurrently, even through separate lumina of multilumen catheters (Arrow, personal communication, 1992). Medications should not be delivered through indwelling lines without intercurrent normal saline flushes. Arteriovenous fistulas, shunts, and Uldall catheters are used only in emergency situations in which more standard methods of IV access are not possible.

Taylor and Taylor [25] state that diazepam and diphenylhydantoin crystallize on contact with the silicone catheter walls of long-term venous access catheters and implantable venous access devices, thereby necessitating catheter replacement. In contrast, the manufacturers of these devices (i.e., Evermed, Pharmacia) hold that crystallization does not occur and that the silicone catheter wall absorbs these drugs without damage or the need for removal.

Adjacent skin cleansing with acetone or tincture of iodine products is injurious to catheter walls (Evermed, personal communication) and should not be used topically in the area of right atrial or triple-lumen catheters. However, povidone-iodine and alcohol are perfectly acceptable.

# EQUIPMENT FOR ACCESS

Access to indwelling lines requires sterile technique and appropriate precautions for prevention of exposure to bloodborne pathogens. The following items are recommended for each approach: sterile gloves, mask, and eyewear; povidone-iodine solution (10%); and sterile drapes. Additional items for specific catheter systems are identified below. For occluded catheters that do not respond to simple maneuvers (including a saline or a heparin flush), the use of thrombolytic agents as described below may be needed.

**Long-Term Venous Access Catheters**

For long-term venous access, clinicians should use catheters attached to implanted pumps only in emergencies. [25] Items needed are as follows:

1. One 10-mL syringe filled with normal saline
2. One 10-mL syringe containing 5 mL of heparin solution (100 U/mL) (5 to 20 mL of normal saline for Groshong catheters)
3. One catheter clamp or hemostat without teeth (do not clamp Groshong catheters)
4. Fluids and medications to be administered

**Implantable Venous Access Devices**

Implantable venous access devices [25] require the following:

1. One 10-mL syringe filled with normal saline
2. One 10-mL syringe containing 5 mL of heparin solution (1000 U/mL)
3. Two Huber needles with a 90° bend (19, 20, or 22 ga) or, in an emergency, a standard 19-ga needle
4. Extension tubing with a slide clamp
5. 4 × 4-inch gauze and 1-inch silk tape adequate to reinforce and stabilize the Huber needle
6. Fluids and medications to be administered

### Percutaneous Multilumen Catheters

The following items are needed with percutaneous multilumen catheters, [25] including Uldall and Manhurkar catheters:

1. One 10-mL syringe filled with normal saline
2. One 10-mL syringe containing 5 mL of heparin solution (100 U/mL) for Uldall catheters and one 10-mL syringe containing 3 mL of heparin solution (1000 U/mL) for Manhurkar catheters
3. Three standard needles (18, 19, or 20 ga)
4. Fluids and medications to be administered

### Arteriovenous Fistulas and Shunts

Arteriovenous fistulas and shunts, which are used for access only in emergencies, require the following:

1. Standard large-bore needles and IV catheters
2. Two 10-mL syringes
3. Fluids and medications to be administered

## PROCEDURES

### Accessing Long-Term Venous Access Catheters

Sterile technique is desired at all times when accessing indwelling lines. [24] The catheter (with the exception of Groshong catheters) is first clamped to prevent air embolism. Patients usually carry their own clamps; however, a hemostat without teeth will suffice. In an emergency, sterile tape or tubing wrapped around the teeth of a hemostat also protects the catheter. The cap is removed, and a 10-mL syringe of sterile water or normal saline is attached. Three to 5 mL of solution are injected and then withdrawn to ensure patency. More pronounced aspiration may be necessary to ensure patency of Groshong catheters. This procedure is done slowly with a syringe at least 10 mL in

volume to avoid damage to the cap area. [25]

Phlebotomy is accomplished by withdrawing dead space solution, reclamping, and using a separate syringe to remove the desired amount of blood. [25] Bolus medications are then injected and IV solutions infused through the catheter, which is clamped whenever unattached. A 5-mL normal saline flush should be delivered between medications. On completion, 3 to 5 mL of heparin (100 units/mL) are injected, the line is clamped, and the cap is repositioned. [15] [25] Groshong catheters should not be flushed with heparin, but instead should be flushed briskly with 5 to 20 mL of saline.

Difficulty in drawing blood from a long-term venous access catheter may be due to catheter position, catheter malfunction, or a fibrinous clot on the catheter tip. [25] Maneuvers to facilitate blood flow include the Valsalva maneuver, the reverse Trendelenburg position, slight tension on the catheter, IV hydration, and having the patient extend his or her arm above head level. [24] [25] Overzealous withdrawal on the syringe will collapse the catheter and make demonstration of patency difficult. [15] If these measures are unsuccessful, a fibrin sheath may be present on the catheter tip. Instilling 3- to 5-mL of heparin (10 or 100 units/mL) followed after several minutes by gentle aspiration is often successful in dislodging a clot. Haimov [15] reported no clinical pulmonary emboli after 70 attempts at declotting with a heparin flush. Streptokinase [15] [33] or urokinase [15] [25] are reasonable next steps. One mL of urokinase (5000 units/mL) may be injected and aspiration attempted after a 10-minute interval. [15] [25] This procedure may be repeated once. A urokinase flush successfully declots the line in up to 95% of cases. [15]

For implanted venous access pumps, access the catheter at its pump attachment for emergency infusion and blood drawing.

**Accessing Implantable Venous Access Devices**

The procedure for accessing implantable venous access devices is unique, because these devices are not external. Instead, a circular reservoir (cylinder) lies subcutaneously on the anterior chest wall. The cylinder is first palpated, and the overlying skin is prepared with povidone-iodine solution. A 10-mL syringe filled with sterile water or normal saline is attached to connecting tubing, which in turn is applied to a 19- to 22-ga, 90° tapered (Huber) needle. A clamp should be applied to the connecting tubing whenever the system is open. Air is expelled, and the Huber needle is inserted through the reservoir septum. The needle is inserted slowly and steadily through the diaphragm to the back of the reservoir. Although incomplete perforation of the septum will block flow, substantial pressure also may damage the back of the device. The clamp is removed slowly, and 5 mL of solution is injected to ensure patency. If patency is not easily demonstrated, the same measures described under Accessing Long-Term Venous Access Catheters may be used, including positioning, Valsalva maneuver, heparin flush, and low-dose thrombolytic therapy. [15] [25]

Once the solution has been injected, gentle negative pressure is applied to demonstrate the backflow of blood.

The Huber needle is then stabilized by building 4- × 4-inch gauze about the needle and further reinforcing with 1-inch silk tape. Phlebotomy is performed through the extension tubing after first removing 8 to 9 mL of blood with a separate syringe. IV solutions may also be delivered through extension tubing, although the rate of flow will be limited by the Huber needle's radius. A standard 19-ga needle may be used in emergency circumstances; however, one risks shortening the device's lifetime. A 5-mL normal saline flush should be delivered between medications. The procedure is completed with a 3- to 5-mL heparin (1000 units/mL) flush [25] and removal of the Huber needle.

## Accessing Percutaneous Multilumen Catheters

Percutaneous multilumen central catheters are used infrequently by outpatients. They are usually placed in the subclavian vein to facilitate patient comfort. One port is available for each lumen, and after povidone-iodine preparation is completed, access is gained by either inserting a needle or syringe into the protective cap or removing the cap entirely. A 5-mL normal saline or sterile water flush and verification of backflow precede all subsequent procedures. Phlebotomy is performed through the proximal 18-ga lumen to prevent mixture with medications being delivered through the other two ports. The proximal lumen port of the Arrow multilumen catheter is white and is the longest of the three tails. Tails attached to the more distal lumina are blue and brown. IV infusions are delivered in similar fashion, and a normal saline flush is injected between medications. The procedure is terminated with a 3- to 5-mL heparin (100 units/mL) flush.

## Accessing Arteriovenous Fistulas, Shunts, and Catheters

Arteriovenous fistulas, shunts, and Uldall and Manhurkar catheters are placed in patients who require hemodialysis and represent the sole access for that purpose. Consequently, routine use of these sites for phlebotomy and fluid administration is condemned. In fact, venipuncture of any type in the same extremity as a patent arteriovenous fistula is not recommended. When standard IV access cannot be obtained under emergency circumstances, however, fistulas, shunts, and catheters may all be used to administer IV solutions and medications. If possible, fistula patency should first be ascertained by noting a bruit and palpable thrill, although these signs may not be appreciable if the patient is in extremis.

Prepare the area with povidone-iodine solution and access the fistula with the smallest gauge needle appropriate to the task. [34] Puncture 1 to 2 cm from the anastomosis ends nearest the venous side, and avoid aneurysmal sites. [34] When complete, monitor the area for hemorrhage, and apply local pressure to avoid significant bleeding. Arteriovenous shunts are accessed similarly by placing the smallest needle possible into the catheter, bridging arterial and venous circulations. Local pressure should be applied for 5 minutes after completing the procedure. Uldall catheters are used in much the same way that multilumen central catheters are accessed. The retaining cap on each arm may be either removed or injected. Up to 5000 units of heparin are present within the two lumina, and so it is imperative that aspiration be performed before administering fluid or medications. Manhurkar catheters are accessed in a similar manner. After use,

flush each catheter arm with 10 mL of normal saline and instill 1.5 mL of heparin solution (1000 units/mL) into each catheter arm.

## AFTERCARE INSTRUCTIONS

Before release from the emergency department, each patient should be reminded of those aspects of catheter care that prevent complications and prolong the device's lifetime. [25] Patients with implantable venous access devices should avoid direct pressure on the reservoir and report local bruising or bleeding immediately. Although these patients may bathe and swim normally, heparin flushes are essential to prevent thrombosis in patients with these indwelling devices.

Long-term venous access catheters and multilumen catheters are sterilely dressed and flushed on a routine basis, [25] and patients should never force the delivery of heparin. Tunneled (e.g., Hickman) and nontunneled (e.g., PICC) catheters are flushed twice each week with 5 mL of heparin (10 units/mL). Implantable venous access devices are flushed every 4 weeks with heparin. Generally, Groshong catheters are flushed with 5 mL of saline once a week. Manhurkar and Uldall catheters are "flushed" during dialysis. Manhurkar catheters also are used for pheresis, in which case they are treated three times a week with normal saline and heparin as described above. An inability to flush any indwelling catheter should be reported immediately to a physician. All indwelling catheters must be observed daily for bleeding and signs of infection, which include fever, pain, redness, swelling, and purulent drainage.

## COMPLICATIONS (Table 25-1)

### Complications of Emergency Department Access

Line sepsis, discussed in more depth later, occurs when sterile technique is not strictly maintained. [24] [25] [35] Gloves, mask, and goggles should be donned before accessing indwelling lines and arteriovenous fistulas. Also, maintaining a closed system by clamping the catheter appropriately prevents the delivery of air into the venous circulation. Air embolism [36] is heralded by tachypnea, hypotension, and coma. When air embolism is suspected clinically, the patient should be positioned on the left side in the Trendelenburg

| **TABLE 25-1** -- Complications of Accessing Indwelling Lines in the Emergency Department |
| --- |
| Line sepsis |

| | |
|---|---|
| Pulmonary embolism | |
| Air embolism | |
| Perforation of externalized catheter | |
| Displacement of catheter | |
| Cardiac dysrhythmias | |

position, and supportive measures, including high-flow oxygen and secure IV access, should be undertaken.

The externalized portion of a right atrial catheter may be cut with scissors [5] or perforated during clamping, especially if an improper clamp is used. [36] The catheter should be reclamped immediately between the patient's skin and the location of the catheter injury (except for Groshong catheters). [25] At this point an alternate method of IV access may be sought. Repair kits (e.g., Evermed) for externalized catheters are available that contain silicone adhesive, plastic clamps, injection caps, and a 12 Fr catheter replacement segment (see Fig. 25-2) . Familiarity with this technique is necessary to avoid further problems (see below).

Embolization of catheter thrombi during flushing and injection of solutions is a concern. Anderson and colleagues [12] prospectively evaluated the size and frequency of catheter thrombi in 43 patients by aspirating after a urokinase flush. Clots were noted in 40 of 43 subjects and 153 of 508 total specimens. Thrombi varied in size from small fragments to 5 cm in length. No clinical sequelae were noted; however, the investigators discuss the potential for pulmonary embolism. Haimov [15] reported no clinical pulmonary emboli after heparin flushes in 70 patients; however, Zureikat and associates reported 1 case of pulmonary embolus associated with Broviac catheterization in a 2-month-old. [37] Emergency department personnel should be vigilant for symptoms of pulmonary embolus and should inject fluids with care.

Catheter displacement may occur when patients do not remain still during the procedure. Harvey and colleagues [30] reported two cases of Hickman catheters being withdrawn accidentally by patients. Chardavoyne and coworkers [38] described a similar

case in which an implantable venous access device (Infuse-A-Port) spontaneously withdrew from the central circulation after documented placement. Care must be taken when handling both long-term venous access catheters and implantable venous access devices, especially in active patients.

## General Complications of Indwelling Lines and Arteriovenous Fistulas and Shunts

### Infectious complications.

Emergency department personnel must remain vigilant for catheter sepsis in immunocompromised patients with indwelling lines and fistulas. The long-term venous access catheter infection rate in adults is between 1.5 and 19%, [5] [11] [30] whereas implantable venous access device sepsis is less frequent at 3%. [11] Among children, 2.8 Hickman catheter infections occur for every 1000 catheter-days, [39] and children between 1 and 4 years of age have a greater risk of multiple septic complications. [39] Insertion site inflammation occurs in 26% of PICCs and 2.6% of short subclavian catheters, although wound site cultures are usually negative. [22] Changing a triple-lumen catheter every 3 days does not decrease the infection rate, and changing such catheters over guide wires increases the risk of bacteremia. [29]

Catheter infections occur at the skin exit site, SQ tunnel, and skin overlying the catheter (venous) insertion site. [30] The SQ tunnel and exit site seem to be involved more frequently. [30] Clinical manifestations include local erythema, tenderness, fever, and purulent drainage [5] ; however, generalized immune suppression may mask these signs. [30] Some authors identified acute leukemia as a risk factor for line sepsis, especially during neutropenia. [22] [30] However, Couch and associates [5] noted a lower infection rate and no such correlation when considering the total white blood cell count at the time of insertion into osteomyelitis patients.

Staphylococci, streptococci, and diphtheroid organisms are cultured most frequently from infected indwelling catheters (Table 25-2) . [14] [21] [33] [39] *Staphylococcus aureus* and *Staphylococcus epidermidis*[5] [14] [40] [41] are common, as are *Streptococcus faecalis, Streptococcus bovis,* and viridans group streptococci. [41] Group C streptococci have also been implicated. [42] Gram-negative bacteria occur less frequently and may be associated with a neutropenic state. [40] These organisms include *Pseudomonas aeruginosa, Klebsiella* spp., *Acinetobacter* spp., and *Serratia* spp.; *Corynebacterium* spp., [40] [43] *Bacillus* spp., [44] and atypical mycobacteria [45] [46] have also been implicated.

Fungal infections occur less frequently but are nonetheless virulent. Premature infants requiring prolonged hyperalimentation seem to be at greatest risk [47] [48] ; organisms cultured include *Candida* spp., *Malassezia pachydermatis,* and *Malassezia furfur,* the causative agent of tinea versicolor. [43] [47] [48] [49] [50] These children present with low-grade fever, bradycardia, thrombocytopenia, apnea, and a predominance of immature polymorphonucleocytes. [49] Adults may develop primary cutaneous aspergillosis near a long-term venous access catheter exit site, with erythema, induration, and black cutaneous necrosis. [51] One adult with acute nonlymphocytic leukemia presented with a mycotic aortic aneurysm while receiving outpatient

chemotherapy through a Hickman catheter. [49] Appropriate antifungal therapy includes ketoconazole, itraconazole, flucytosine, and amphotericin B on an inpatient basis. [49] [51]

Management of indwelling catheter infections must be coordinated through the patient's primary physician (e.g., hematologist, oncologist). After supportive measures have been undertaken, the primary considerations are (1) the choice of antimicrobial, (2) the decision to remove the indwelling line, and (3) the decision to hospitalize. Selected patients may not require catheter removal. [5] [43] [45] [52] Couch and associates [5] removed only 5 of 105 Hickman catheters from infected sites before completion of antimicrobial therapy. Furthermore, a portion of those treated without catheter removal may be followed as outpatients. [5] Criteria for outpatient management of indwelling catheter infections include

| TABLE 25-2 -- Microorganisms Causing Indwelling Catheter Infection | |
|---|---|
| **Bacterial** | |
| Gram-positive cocci | *Staphylococcus aureus, Staphylococcus epidermidis, Streptococcus faecalis, Streptococcus bovis,* group C streptococci, and viridans streptococcus |
| Gram-negative bacilli | *Pseudomonas aeruginosa, Klebsiella* sp., *Acinetobacter* sp., *Serratia* sp. |
| Gram-positive bacilli | *Bacillus cereus, Bacillus laterosporus, Corynebacterium* sp. |
| Atypical | *Mycobacterium neoaurum, Mycobacterium fortuitum, Mycobacterium chelonae* |
| **Mycotic** | *Malassezia furfur, Malassezia pachydermatis, Aspergillus fumigatus, Aspergillus flavus, Candida albicans* |

(1) a competent immune system; (2) an intelligent patient with adequate outpatient support; (3) an infection localized to the skin exit site; and (4) an absence of fever, leukocytosis, granulocytopenia, tachycardia, and hypotension.

Cultures are obtained of local purulent discharge, blood drawn through the catheter, and blood drawn from a distant site. An oral antimicrobial should then be chosen that covers both staphylococcal and streptococcal organisms; semisynthetic penicillins and first-generation cephalosporins are adequate. Finally, the patient may be released home only after consultation with the primary physician and assurance of 24- to 48-hour follow-up to ensure clinical reassessment and review of culture results.

All patients with indwelling catheter infections who fall outside of this narrow spectrum are hospitalized. Those who are hemodynamically stable may have cultures taken and be treated with IV antimicrobials that are effective against gram-positive and gram-negative organisms. Vancomycin is effective [21] [30] [53] because of its gram-positive spectrum and efficacy against diphtheroids, which may be resistant to other antimicrobials. Aminoglycosides provide adequate gram-negative coverage [53] ; however, broad-spectrum triple antimicrobial therapy may be necessary in neutropenic patients. Strong consideration should be given to catheter removal when hypotension supervenes. Before an indwelling line is removed, cultures of blood drawn through the device, as well as the catheter tip itself, should be obtained. The decision to remove a catheter should be made in concert with the consultant, as these devices are both surgically implanted and expensive. Local thrombolytic therapy may remove fibrin sheaths that entrap pyogenic bacteria near catheter tips and possibly obviate the need for catheter removal, especially in children.

Arteriovenous shunts are seen less frequently in the emergency department owing to the increased use of femoral and subclavian Uldall and Manhurkar catheters for temporary hemodialysis. Localized shunt infections are generally caused by staphylococci or streptococci. [54] Gram-negative organisms are implicated less frequently, and antimicrobial coverage may be extended to include them. [55] Arteriovenous fistula infections are rare but, when present, may lead to thrombosis and massive bleeding. [15] Overlying cellulitis without involvement of the fistula itself may be treated on an outpatient basis with a single IV dose of vancomycin followed by an oral antimicrobial (e.g., cephalexin). Involvement of the fistula itself mandates hospital admission and observation for hemorrhagic complications.

**Noninfectious complications.**

Catheter thrombosis occurs more frequently in long-term venous access catheters (22%) than in implantable venous access devices (1%), [11] and pulmonary embolism associated with indwelling lines has been reported. [30] [50] [56] Flushing these catheters with heparin, urokinase, and streptokinase should be done with great care and vigilance. One commonly used approach is to inject a 1-mL, 5000-unit dose of urokinase, allow it to dwell for 3 to 5 minutes, and then attempt to aspirate blood. If

unsuccessful, repeat the procedure. Before replacing the line, consider admission for a 12-hour continuous urokinase infusion. [57]

A potentially fatal pulmonary embolus may be heralded by the sudden onset of chest pain, shortness of breath, tachycardia, and tachypnea. Continuous urokinase therapy has been effective in resolving pulmonary emboli in the setting of a large right atrial thrombus. [37] Inability to clear an indwelling line using the measures described under Procedures necessitates peripheral venous access and referral for possible catheter replacement.

Fractures of Hickman-type catheters occur either subcutaneously or in the externalized portion. [58] SQ fractures cause localized pain and swelling, and the involved line should be removed, because complete fracture with embolization can occur, albeit rare. Embolization of a catheter fragment is life threatening and causes acute dyspnea, palpitations, atypical chest discomfort, hypoxia, and atrial fibrillation. Embolized fragments are localized by chest radiography and removed either surgically or nonsurgically. [59] [60]

Repair of fractures of externalized catheter portions is done using kits made by the appropriate manufacturer. In general, the catheter is cut proximal to the fracture, the damaged portion removed (discarded), a new catheter segment inserted into the proximal catheter segment, and an overlying sleeve glued to connect and seal the two segments together (Fig. 25-7) . The repaired catheter is then secured in place and cleared with standard flush solution. Gryn and Sacchetti recommend securing the repaired catheter to a tongue blade using tape several cm on each side of the splice sleeve. [58] Repairs of externalized catheter fractures within 2 cm of the insertion site represent a greater risk for failure and embolization. These repairs should be done by personnel familiar with such repairs. [58] When a repair kit is unavailable, temporary (emergency) vascular access can be achieved by transecting the leaking externalized catheter as above and then inserting an IV catheter (frequently a 14-ga catheter will suffice) into the exposed lumen.

Additional complications of indwelling lines include mediastinitis, cardiac dysrhythmias, superior vena cava syndrome, SQ tunnel hematoma, central vein stenosis, and septic atrial thrombosis associated with Budd-Chiari malformation. [5] [56] Erosion of the SQ tissue by the Dacron cuff, causing a superficial ulcer, also has been reported. [61] In addition, pneumothorax, cardiac tamponade, lymphatic duct puncture, air embolism, and brachial plexus injury may worsen among outpatients if the result of catheter insertion is not recognized immediately. [62]

**Figure 25-7** Catheter repair. A hollow lumen connector and outer sleeve are placed to splice a replacement catheter to the remaining catheter segment (see text).

The most life-threatening complication of arteriovenous fistulas is bleeding caused by either trauma or infection. Massive blood loss is possible because the arterial system is violated; treatment includes local pressure, IV access, oxygen, supportive therapy, and immediate vascular surgery referral to obtain definitive control of the bleeding site. If

bleeding is substantial and not easily controlled, consider IV protamine in a dose of 0.01 mg per unit of heparin, or 10 to 20 mg for 1000 to 2000 units of heparin. [63] If possible, consult with the patient's nephrologist or vascular surgeon before giving protamine.

In addition to hemorrhaging, arteriovenous fistulas may thrombose, a complication usually related to dehydration (e.g., after hemodialysis), hypotension, recent surgery, local trauma, or a superficial ulcer. [15] Recombinant human erythropoietin use is a risk factor for thrombosis. [64] Also, thromboses occur more frequently among graft fistulas than among endogenous grafts. [65] Patients with the loss of a bruit or thrill in a previously functioning shunt or fistula should be referred to a plastic surgeon for possible revision. Thrombectomy may salvage the fistula once these precipitating factors have been addressed. Alternative venous access for hemodialysis (e.g., Manhurkar catheter) may be necessary.

Shunt and fistula infections can be difficult to detect, because fever and leukocytosis are frequently absent. [66] If a shunt or fistula infection is suspected, the clinician should first culture the blood and infected site. Most febrile patients are admitted, although some are given an IV dose of antimicrobial and discharged with 24-hour follow-up. [63] Vancomycin is an effective choice for the most common pyogenic bacteria ( *Staphylococcus* spp.).

The steal syndrome occurs when blood passes preferentially through a low-resistance arteriovenous fistula at the expense of distal arteries. [67] It generally occurs within 24 hours of fistula surgery in 1 to 3% of patients. [68] [69] Symptoms include pain, paresthesia, and weakness of the fingers and hand. [15] Again, surgical evaluation of the fistula is indicated. Venous hypertension distal to a forearm fistula may lead to painful hand swelling, stasis changes, and hyperpigmentation. The fistula may need to be closed if symptoms persist. Finally, venous aneurysms commonly develop but are not life threatening if the overlying skin remains intact.

## CONCLUSION

Numerous methods of vascular access are currently available to the emergency physician, including indwelling catheters and arteriovenous anastomoses. Facility with these latter devices enables emergency department staff to access the venous circulation of a high-risk patient population in a relatively safe and painless manner. However, vigilance during these procedures is necessary to avert potentially life-threatening complications. Knowledge and recognition of these complications among outpatients may prevent sepsis and life-threatening hemorrhage.

# Chapter 26 - Intraosseous Infusion

**Frank J. Cunningham Jr., William H. Spivey**

Establishing vascular access in a critically ill or injured pediatric patient can be one of the most difficult and frustrating procedures a physician has to perform. For example, in a review of intravascular access in pediatric cardiac arrest, the mean (± SD) time needed to establish venous access was a disappointing 7.9 ± 4.2 minutes. [1] Although peripheral percutaneous venous access was the fastest method (mean time, 3.0 minutes), it was successful in only 17% of cases. Success rate was highest for an intraosseous (IO) infusion (83%), followed by surgical cutdown (81%), and central venous catheterization (77%). The mean time required to obtain a functioning IO line was 4.7 minutes, followed by 8.4 minutes for a central line, and 12.7 minutes for a cutdown. Another study of emergency department pediatric arrests demonstrated failure to gain intravenous (IV) access entirely in 6% of the patients. [2]

Small peripheral vessels in children often collapse during shock, and the child's increased body fat makes peripheral cannulation time consuming and at times impossible. Central venous cannulation can be equally difficult and carries the risk of pneumothorax or arterial injury. Alternative routes for drug administration, such as the endotracheal and rectal routes, do not provide rapid, reliable drug absorption during cardiac arrest. For example, epinephrine administered by the endotracheal route, although effective in a normally functioning cardiovascular system, is poorly absorbed and produces minimal physiologic response when administered during cardiac arrest. [3] [4]

In light of the need for rapid vascular access in pediatric patients, the previously abandoned technique of IO infusion has been reintroduced. It provides a safe, reliable method of accessing the cardiovascular system for administration of fluids and drugs during cardiac arrest and other resuscitations.

## BACKGROUND

One of the earliest references describing the IO route was by Drinker and colleagues, who in 1922 examined the circulation of the sternum and suggested it as a site for transfusion. [5] The route was not used clinically until 1934 when Josefson, a Swedish physician, administered liver concentrate into the sternum of 12 patients with pernicious anemia and reported that all 12 improved. [6] Subsequently, the technique became widespread in the Scandinavian countries.

In 1940 the technique was introduced to American physicians by Tocantins, who described a series of animal and clinical studies that demonstrated fluid was rapidly transported from the medullary cavity of long bones to the heart. [7] [8] [9] Over the next two decades, thousands of cases of IO infusion of blood, crystalloid substances, and drugs were reported. [10] [11] [12] Relatively few complications were reported, considering the needles were often left in place for 24 to 48 hours. Heinild and coworkers in 1947 reviewed 982 cases of IO infusion and reported only 18 failures and 5 cases of

osteomyelitis. [12] None of the cases of osteomyelitis occurred in patients who received isotonic solutions.

With the introduction of plastic catheters and improved cannulation skills, the need for IO infusion as an alternative route of access diminished, and the technique was all but abandoned. It was not until the mid-1980s that the technique was reintroduced in response to the need for immediate vascular access during cardiopulmonary resuscitation. [2] [13] Use of the technique during cardiac arrest was supported by experiments that demonstrated sodium bicarbonate was effectively transported to the heart during cardiac arrest. [14] Since then, the technique has become widespread throughout the United States and is recognized as an accepted alternative to IV access in pediatric emergencies. The safety, ease, and effectiveness of the technique have led to the extension of its use for out-of-hospital emergency care. [15] [16] [17] [18] There is renewed interest in its use in adults as well.

## ANATOMY AND PHYSIOLOGY

Long bones are richly vascular structures with a dynamic circulation that is capable of accepting large volumes of fluid and rapidly transporting fluids or drugs to the central circulation. The bone, like most organs, is supplied by a major artery (nutrient artery). The artery pierces the cortex and divides into ascending and descending branches, which further subdivide into arterioles that pierce the endosteal surface of the stratum compactum to become capillaries. The capillaries drain into medullary venous sinusoids throughout the medullary space that in turn drain into a central venous channel (Fig. 26-1) . The medullary sinusoids accept fluid and drugs during IO infusion and serve as a route for transport to the central venous channel, which exits the bone as nutrient and emissary veins. [20] The medullary cavity functions as a rigid, noncollapsable vein, even in the presence of profound shock or cardiopulmonary arrest. [21] Radiographic studies have demonstrated that radiopaque dye spreads only a few centimeters in the medullary space before being transported to the venous system. [22] The richly vascular red marrow cavity of the long bones is gradually replaced by less vascular yellow marrow after 5 years of age. [23]

Almost every drug and fluid commonly used in resuscitation

**Figure 26-1** Schematic diagram illustrating the venous drainage from the marrow of a long bone with an intraosseous (IO) needle in place.

has been reported in clinical and preclinical IO studies. Medications and fluids that have been administered through IO infusion are listed in Table 26-1 (Table Not Available) . Crystalloid infusion studies in animals have demonstrated that infusion rates of 10 to 17 mL/min may be achieved with gravity infusion and rates as high as 42 mL/min with a pressure infusion. [24] [25] [26] IO crystalloid infusion has been shown to produce a significant increase in blood pressure in a hemorrhagic shock model in rabbits. [27] In small animals (7 to 8 kg) the size of the marrow cavity is the rate-limiting factor, whereas in larger animals (12 to 15 kg), the size of the needle determines the flow. [26] Blood

under pressure can be infused approximately two thirds as fast as crystalloid fluids. [26]

Comparisons of IO and IV infusion of drugs have demonstrated that the drugs reach the central circulation by both routes in similar concentrations and at the same time (Fig. 26-2) . [7] [28] This holds true even during cardiopulmonary resuscitation (CPR), where sodium bicarbonate has been shown to provide a greater buffering capacity when administered by the IO route than by the peripheral IV route.

## INDICATIONS AND CONTRAINDICATIONS

IO infusion is a means of achieving rapid temporary vascular access until a patient can be stabilized and traditional IV access obtained. It is indicated when fluid or drugs must be introduced into the circulation rapidly and venous access is not readily available. The primary indication is cardiac arrest in an infant or child. The route has been used in adults, [29] but vascular access is usually easily obtainable, and alternatives to IV access are not often needed. In pediatric patients it may be used as a first line of vascular access if peripheral vascular access does not appear to be readily obtainable. Other indications include shock, trauma, extensive burns, severe dehydration, and status epilepticus, or any situation in which the emergency administration of fluids or drugs is necessary but not feasible by other routes. [30] IO infusion into the tibia also has been used as a site for lower extremity venography. [31]

In addition to serving as a route for fluid administration, the IO needle may be used for obtaining blood for type, crossmatch, and blood chemistry determinations from the marrow cavity. Serum electrolyte, blood urea nitrogen (BUN), creatinine, glucose, and calcium levels are very similar to those in samples obtained from an IO aspirate. [32] [33] Blood gas values obtained from the IO site also are similar to those

**Figure 26-2** Serum diazepam levels (ng/mL, mean ± SE) graphed for the IO *(shaded area)* and intravenous (IV) *(blackened area)* groups as a function of time when injected during normal perfusion. Initially the IV drug level is slightly higher, but overall the difference between the two routes of administration is not significant.

obtained from central venous sites during steady- and low-flow states in one animal model and may be an acceptable alternative to judging central acid-base status during CPR. [34] A complete blood cell count may not be reliable, because it reflects the marrow cell count rather than the cell count in the peripheral circulation. Furthermore, the aspirated blood usually clots within seconds, even if it is placed in a tube that contains heparin. Brickman and colleagues demonstrated that bone marrow aspirates obtained from an IO needle in the iliac crest could be reliably used to type and screen blood for transfusion. [35]

Relatively few contraindications to IO infusion exist. Osteoporosis and osteogenesis imperfecta are associated with a high fracture potential; the procedure should be avoided when these diagnoses are known. A fractured bone must be avoided, because as fluid is infused, it increases the intramedullary pressure and forces fluid to extravasate at the fracture site. This may slow the healing process, cause a nonunion of

the bone, or lead to a compartment syndrome. A similar extravasation of fluid can occur through recent IO puncture sites placed in the same bone. Hence, recent prior use of the same bone for IO infusion represents a relative contraindication to IO line placement. Areas of cellulitis or infection or burns at or near the intended insertion site should be avoided if possible.

## EQUIPMENT AND SETUP

The only equipment necessary for establishing IO access is a sturdy needle with a stylet and a syringe for aspiration. Needles range in size from 20 to 13 ga and are made by several companies. Standard needles for drawing blood or administering medications are *not adequate* for IO infusions; they generally are not sturdy enough to penetrate bone. A cadaver study of IO puncture suggests that non-styletted needles (2.5 cm, 18-ga phlebotomy needles and 7.6 cm, 14-ga IV needles) enter the marrow space successfully about half the time. [36] In the past, an 18-ga spinal needle was commonly used for children younger than 12 to 18 months of age. This needle, although readily available in most emergency departments, often bends and is too long for rapid fluid infusion.

Several needles are currently used in emergency departments for IO infusion (Fig. 26-3) . Bone marrow aspiration needles such as the Rosenthal or Osgood can be used if available. They are large enough (16 ga) to be used on older children or adults and are good for fluid administration. The Illinois sternal/iliac aspiration needle (Monoject, Division of Sherwood Medical, St. Louis) is a 16-ga needle that has an adjustable plastic sleeve to prevent the needle from penetrating too deeply or through the opposite cortex. Its disadvantage is that it has a long shaft and cumbersome handle that make it vulnerable to dislodgment from the bone during transport or other procedures. The Cook IO needle (Cook Critical Care, Bloomington, Ind) comes in 18- and 16-ga sizes and has a detachable handle that decreases the likelihood of its being dislodged. A useful feature of this needle is a line located 1 cm from the tip of the needle to serve as a depth marker. The Sur-Fast needle (Cook Critical Care, Inc., Bloomington, Ind) has a threaded shaft to permit a more secure needle placement. The Jamshidi Disposable Illinois sternal/iliac aspiration needle (Baxter Healthcare, Valencia,

**Figure 26-3** Needles used for intraosseous infusion. *Left to right,* MedSurg Industries bone marrow aspiration needle, MedSurg Industries Illinois sternal/iliac aspiration needle, Jamshidi disposable sternal/iliac aspiration needle, Cook IO needle with 45° trocar, Sur-Fast IO needle.

Calif) comes in either 15- or 18-ga sizes and, like the larger Illinois sternal/iliac aspiration needle mentioned above, features an adjustable plastic sleeve. However, its shorter shaft length and smaller handle make it much easier to use. A 13-ga needle manufactured by MedSurg Industries (Rockville, Md) is good for fluid resuscitation. However, like the Illinois sternal/iliac aspiration needle, this latter product has a large handle that makes it cumbersome.

## TECHNIQUE

Originally the sternum was used as a site for IO infusion. After several cases of osteomyelitis and mediastinitis in children, the site was abandoned. [37] Other sites, such as the clavicle and humerus, have been used, but neither has gained popularity. Currently the site of choice is the proximal tibia, followed by the distal tibia and distal femur.

The tibia is popular because it is a large bone with a thin layer of subcutaneous tissue that allows landmarks to be readily palpated. It is the preferred site of IO insertion in infants and children <6 years of age, and its insertion does not interfere with airway management and cardiopulmonary resuscitation. On the proximal tibia, the broad, flat anteromedial surface is used, with the tibial tuberosity serving as a landmark. The site of IO cannulation is approximately 1 to 3 cm (2 fingerwidths) below the tuberosity on the medial or flat surface of the tibia (Fig. 26-4 *A*). This location is far enough from the growth plate to prevent damage but is in an area in which the bone is still soft enough to allow easy penetration of a needle. This site may be used in adults but is more difficult and requires a 16- to 13-ga needle to penetrate.

The patient's leg should be supported by placing a small sandbag or towel roll behind the knee. The site is prepared with a povidone-iodine solution or alcohol. Local anesthesia is usually unnecessary, since the majority of patients in whom IO is used have altered mental status from shock or are in cardiopulmonary arrest. However, if the patient is conscious, the skin and periosteum should be anesthetized. The thigh and knee above and lateral to the insertion site are grasped with the palm of the nondominant hand (Fig. 26-5) . The fingers and thumb are wrapped around the knee to stabilize the proximal tibia. To avoid self-puncture, care must be taken to ensure that the hand does not extend behind the insertion site.

The bony landmarks are palpated and the site identified. The needle should be grasped firmly in the palm of the hand and is directed either perpendicular (90°) to the long axis of the bone or slightly caudad (60° to 75°) to avoid penetration or injury to the growth plate. The needle is then advanced with a twisting or rotary motion to cut the bone and facilitate puncture of the cortex. Considerable resistance will be encountered, but once the cortex has been penetrated, there is a sudden decrease in resistance and a crunching feeling as the needle moves through the bony trabeculae into the marrow cavity. The distance from the skin through the cortex of the bone is rarely >1 cm in an infant or child, and penetration to this depth is usually adequate. A common mistake is to advance the needle through the opposite side of the bone. This can best be avoided by holding the needle in the palm of the hand with the index finger approximately 1 cm from the bevel of the needle to avoid pushing it past this mark. Another method is to use a needle with a preset plastic depth indicator on the shaft. [38] The stylet is then removed, and a 5- to 10-mL syringe is used to aspirate blood and marrow contents for confirmation of position. Many times, particularly during cardiac arrest, blood aspiration is not possible. Other signs of successful needle insertion include the needle's ability to stand upright without support and infused fluids that flow easily without evidence of swelling or extravasation.

If there is excessive resistance to fluid infusion, the needle may be pulled back a few

millimeters and another attempt made to infuse fluids. If continued resistance is met or evidence of extravasation exists, the needle should be removed and an attempt made on the other extremity. If the needle appears to be infusing properly, the test syringe is disconnected, and the needle is then connected to infusion tubing. The needle and tubing should be secured with tape and the extremity immobilized on a leg board (Fig. 26-6 A). The needle should be protected from accidental dislodgment by wrapping a stretch bandage around the extremity and placing a small plastic drinking cup around the needle (Fig. 26-6 B). The IO needle should be removed as soon as secure IV access has been obtained. A sterile dressing is placed over the puncture site, and pressure should be applied to the dressing for 5 minutes. [39]

The distal tibia, although the preferred site in adults, may be used as well in children. [29] [40] The cortex of the bone and the overlying tissue are both thin. The site of needle insertion is the medial surface at the junction of the medial malleolus and the shaft of the tibia, posterior to the greater saphenous vein (see Fig. 26-4 B). The needle is inserted perpendicular to the long axis of the bone or 10° to 15° cephalad to avoid the growth plate. [38]

The distal portion of the femur is occasionally used as an alternate site, but because of thick overlying muscle and soft tissue, it is more difficult to palpate bony landmarks (Fig. 26-4 C). If chosen, the needle should be inserted 2 to 3 cm above the external femoral condyles in the midline and directed cephalad at an angle of 10° to 15° from the vertical. [41]

In one study, similar rapid central circulation times of medications were noted when the distal tibia, proximal tibia, distal femur, and proximal humerus were compared to a peripheral IV site in a swine model, suggesting that adjustments in drug dosage may not be required for intraosseously administered medications

## COMPLICATIONS

As with any procedure, complications may arise when attempting to use the bone for vascular access. Complications may be divided into two categories: technical difficulties and latent soft tissue or bone problems. Technical difficulties are the most common, but these decrease as familiarity with the technique increases (Fig. 26-7) . The most common mistake is to place excessive pressure on the needle during insertion and force it through the bone (Fig. 26-8) . This may be avoided by placing the index finger against the skin to prevent the needle from going in too deeply. Also, attention to depth of the needle or use of a sheath on the needle to prevent excess penetration will decrease occurrences of this problem. Incomplete penetration of the bone may also occur, in which case blood will not be aspirated and fluid will extravasate if infused. Serum levels of medication are decreased when IO infusion is performed through bones with multiple cortical defects. [43] If several attempts are made to place a needle in the same bone, fluid may extravasate from previous puncture wounds. If extravasation occurs, the needle should be removed and pressure applied.

The needle may be blocked periodically by clots forming around the bevel or by bony spicules obstructing the flow of fluid. This complication may require that the line be

flushed with 3 to 5 mL of sterile saline every 10 to 15 minutes to keep it open. Pressure infusions usually do not require taking this step.

A major concern for any person receiving IO infusion is infection. This concern often leads physicians to shy away from using the bone and to continue searching for other methods of vascular access. Although the potential for infection is real, its actual incidence is low. A literature review of more than 4000 cases from 1942 to 1977 found a 0.6% incidence of infection. [2] Although most of the affected access sites were not placed under emergency conditions, the needles were often left in 1 to 2 days, thus increasing the likelihood of infection. A survey of more than 1000 U.S. and foreign medical schools found that the incidence of infection for IO needles placed in emergency conditions was <3%. [44]

The most common infection is cellulitis at the puncture site, which usually responds well to antibiotics. Osteomyelitis is less common, but it also usually responds to antibiotics. Heinild and colleagues reported three cases of osteomyelitis in 25 patients who received infusions of undiluted 50% dextrose in water ($D_{50}W$). [12] Undiluted $D_{50}W$ injected into rabbit femurs produces edema and pyknotic marrow nuclei that improve within 1 month. [45] In addition to infection, inflammatory reactions of the bone may be seen. These are most common when hypertonic or sclerosing agents are used and may produce an elevation of the periosteum with a positive bone scan (Fig. 26-9) . Unlike the clinical appearance of a patient with osteomyelitis due to bacteria, a child with a sterile inflammatory reaction does not look "toxic." One hypertonic sclerosing drug that is routinely used during cardiac arrest is sodium bicarbonate. Heinild and coworkers reported 78 cases of bicarbonate infusion with no complications. [12] Animal studies have reported a decrease in cellularity with edema and destruction of some cells, but these changes are temporary and completely resolve in a few weeks. [45] [46] [47]

Another complication that has been reported is necrosis and sloughing of the skin at the site of infusion. [48] This occurs if fluid or drugs extravasate from the puncture site into the surrounding tissues. Care should be taken when infusing drugs such as calcium chloride, epinephrine, and sodium bicarbonate to prevent dislodgment of the needle and extravasation into the tissue. It is best to infuse such drugs gently and not under pressure, because pressure frequently causes extravasation.

Injury of the growth plate and developmental abnormalities of the bone are ongoing concerns. These fears have not been supported in the literature, however. There have been no reports of growth plate damage or permanent abnormalities of the bone. One animal study specifically examined damage to the epiphysis; sodium bicarbonate was injected directly into the epiphysis, and no radiologic evidence of epiphyseal injury was found. [49] [50] By pointing the needle away from the joint space and using the previously mentioned landmarks for insertion, the danger of epiphyseal injury is remote.

Fat embolism is frequently mentioned as a potential complication. [2] [40] However, this condition is rare and has been reported only in adult patients. [51] Animal studies addressing this issue found no changes in blood gases during IO infusion and limited evidence of fat globule collection in the lungs. [52] [53] Because the marrow in infants and children is primarily hematopoietic, this potential complication is unlikely to occur. In one

animal study, no significant hemolysis was detected in the setting of pressurized IO blood infusion. [52]

Compartment syndrome following IO line use during cardiac resuscitation has been reported. [54] [55] Tibial fractures have also been reported. [56] [57] Follow-up radiographs of patients who have undergone IO needle placement or attempts at such placement are indicated. Cortical defects may be seen on radiographs for up to 40 days after injection.

## INTERPRETATION

The determination of whether an IO infusion is functioning may be made by assessing the ease with which fluid infuses and the clinical response of the patient. If the needle is properly placed, fluid should flow smoothly, with only occasional flushes necessary to keep it open. A clinical response to fluid or medications should be seen at approximately the same time and in the same magnitude as with IV administration.

## CONCLUSION

IO infusion provides a means of rapidly accessing the cardiovascular system in emergencies. The technique is not intended to replace traditional venous access; instead it should serve as a temporizing measure for resuscitation until venous access can be obtained. It is currently used widely throughout the United States and Canada and has been reviewed extensively in the emergency medicine and pediatric literature. The technique also is used by out-of-hospital emergency care personnel, who have demonstrated a high success rate in the field. [17] [59] The technique renders hope for those who infrequently care for critically ill or injured children, because this skill is easily mastered even if done infrequently.

Complications are most commonly related to technical mistakes. By carefully locating landmarks, staying away from the growth plate, and paying attention to the depth of the needle, complications can be minimized. Finally, as with any technique, practice on cadaver or animal leg bones (e.g., chicken, pig) greatly improves one's skill.

# Chapter 27 - Endotracheal Drug Administration

**James H. Bryan** , **J. Thomas Ward Jr.**

Endotracheal (ET) administration of selected medications is a simple, rapid, and effective method of drug delivery to the central circulation. However, this technique of drug administration should be reserved for use in situations in which a patient's condition warrants immediate pharmacologic intervention and when more conventional means of drug delivery, such as by intravenous (IV) or intraosseous (IO) access, are not readily available. Such circumstances occur infrequently, but when they do occur, knowledge of the appropriate drugs and dosages that can be delivered effectively by this route may prove to be life saving. It is important, therefore, that physicians be familiar with the concept and method of ET drug therapy.

Endotracheal drug administration dates to 1857, when Bernard demonstrated the ability of the lung to rapidly absorb an instilled solution of curare. [1] In this experiment, curare was instilled into the upper respiratory tract of dogs by way of a tracheostomy. After the dogs were tilted to an upright position, they died within 7 to 8 minutes. Bernard concluded that the alveoli must be permeable to curare. Over the following decades, other investigators expanded this work and demonstrated that solutions containing salicylates, atropine, potassium iodide, strychnine, and chloral hydrate were also absorbed rapidly from the lung and excreted

in the urine after injection of their aqueous solutions into the tracheas of experimental animals. [2]

In 1915, Kline and Winternitz provided experimental data suggesting that intratracheal therapy in pulmonary disease might prove to be an effective therapeutic route. [3] Further studies showed that moderate quantities of fluid could be insufflated into the lungs without marked changes in the lung architecture. [4] The use of intrapulmonary medication in the treatment of lung disease gained further acceptance when studies demonstrated that the inhalation of epinephrine mist dramatically relieved the symptoms of asthma. [5]

In the late 1930s and 1940s when certain chronic suppurative disorders of the lung failed to respond to parenteral antibiotics, drugs were administered by direct instillation into the lungs. During this period, several important observations were made concerning ET drug therapy: (1) penicillin delivered by the ET route demonstrated a depot effect, resulting in therapeutic blood levels that lasted twice as long as those noted with intramuscular injections [6] ; (2) various diluents mixed with penicillin affected both the rate and the degree of absorption from the lungs [7] ; and (3) higher serum drug levels were attained with direct ET drug administration than with aerosolized administration. [7]

In the 1950s, other important findings concerning the ET administration of drugs were established by investigators attempting to elucidate the mechanism responsible for adverse anesthetic reactions. This research demonstrated two important points. First, drugs delivered endotracheally were absorbed much more rapidly than those applied to

the posterior pharynx. Second, the rapid absorption of drugs applied locally to the larynx and trachea resulted in blood levels significant enough to cause adverse anesthetic reactions. [8]

In 1967, Redding and coworkers studied the use of ET administration as a route of drug delivery in a canine model of cardiopulmonary arrest. [9] They administered epinephrine by the IV, intracardiac, and intratracheal routes, and then evaluated its effectiveness in the resuscitation of dogs that had undergone both respiratory and circulatory arrest secondary to hypoxia. Their study revealed that all three routes of drug administration were equally effective in restoring the circulation of dogs in *hypoxia-induced* cardiac arrest, again demonstrating that the ET route of drug delivery provides an effective window to the systemic circulation. They concluded that whichever of these routes is most immediately available should be used.

A decade passed before further research was published concerning the use of ET drug therapy as an alternative means of systemic drug administration. In the late 1970s, Roberts and Greenberg and associates revived the study of ET drug delivery with a series of laboratory experiments and clinical uses of ET epinephrine. [10] [11] [12] [13] Since that time, a number of important animal and human studies, as well as a number of case reports, have been published dealing with various aspects of ET drug administration. These investigations have addressed (1) the appropriate dose of drug to administer; (2) the effect of drug solution volume; (3) the effect of different diluent solutions; (4) the role of different ET drug delivery techniques; and (5) the effects of hypoxia, hypotension, shock, and cardiopulmonary arrest on the absorption, distribution, and efficacy of endotracheally administered drugs.

## RECOMMENDATIONS REGARDING ENDOTRACHEAL DRUG DELIVERY

Although the American Heart Association's (AHA) Guidelines for Cardiopulmonary Resuscitation and Emergency Cardiac Care make specific recommendations regarding the use of ET drug delivery for cardiac resuscitation (Table 27-1) (Table Not Available) , [14] [15] [16] much of the literature remains controversial and is at times contradictory. It is likely, therefore, that many of these issues will continue to be the subject of future investigations. Specific studies related to each of these topics are discussed in the following paragraphs.

### Appropriate Dose

All investigators agree that the ET dose of a medication should be at least equal to the IV dose of the same drug given for the same indication. Most studies on the subject indicate that higher doses are needed endotracheally than intravenously. For Advanced Cardiac Life Support medications in adults, the AHA recommends a dose 2.0 to 2.5 times the usual IV dose. [14] This recommendation is supported by the results of a study of epinephrine administered immediately following intubation in the out-of-hospital setting. [17] There are, however, other studies that suggest that ET administration

requires higher doses.

In studies of endotracheally administered lidocaine, a 3 mg/kg dose was needed to obtain therapeutic serum levels. Studies and case reports of ET epinephrine have produced conflicting results. Some animal studies [20] and case reports [13] have shown positive effects and/or recovery from cardiovascular collapse when epinephrine was used in doses equal to recommended IV doses. In other animal [21] and human [22] studies, however, epinephrine in doses of approximately 0.02 mg/kg and 0.01 mg/kg, respectively, were shown to be unreliable in producing a physiologic response. In addition, studies using both normotensive and cardiac arrest canine models have shown that epinephrine doses of 0.01 mg/kg produce serum levels approximately one-tenth that produced when the same dose is given IV. [11] [23] [24] These studies recommend increasing the ET epinephrine dose to 0.1 mg/kg and are the basis for the AHA recommendation to use a 10-fold increased dose when administering ET epinephrine to pediatric patients. [15] However, ET drug delivery is associated with a depot effect. That is, when larger doses of drugs are administered via the ET route, the drug is "stored" and released slowly over time, similar to a continuous IV drip. This presumably occurs due to local vasoconstriction and/or lymphatic storage of the drug. [10] With epinephrine use, the depot effect produces a potential for postresuscitative arrhythmias, hypertension, and tachycardia, with resultant increased myocardial oxygen demand.

Given these data, it seems reasonable in adults to start with a dose 2.0 to 2.5 times the usual IV dose. If this appears ineffective, higher doses may be used on subsequent administration.

**Single-Dose Volume**

The need to increase the volume of the instilled medication to enhance effectiveness was recognized by Redding and associates during their early studies. [9] The goal is to use a volume that will enhance delivery and absorption of the drug while at the same time producing minimal deleterious effects on pulmonary gas exchange. The AHA recommends a total volume of 10 mL in adults [14] and 1-2 mL in pediatric patients and neonates. [15] [16] In studies with dogs, Mace compared undiluted lidocaine to diluted lidocaine (volume, 6.5 mL) and found significantly higher plasma lidocaine levels in the animals receiving diluted lidocaine. [25] There were no changes in the arterial blood gas values before and after ET drug administration. In another animal study, lidocaine diluted with normal saline to volumes up to 25 mL produced no changes in arterial blood gases or clinical condition, and no change was seen in lung gross anatomy or histology. [26] In contrast, a study comparing normal saline to distilled water revealed a decreased $Pa_{O2}$ for both solutions (water producing the greatest effect), but this study used large volumes (2 mL/kg) of solution. [27]

Studies of endotracheally administered lidocaine in human subjects revealed that dilution with distilled water to a total volume of 10 mL resulted in higher plasma lidocaine levels, but also produced a decrease in $Pa_{O2}$ of approximately 40 mm Hg that persisted for more than 1 hour. [28] A total volume of 5 mL yielded lower plasma levels but also produced a shorter period of hypoxemia. The authors concluded that a total volume of 5 to 10 mL (in agreement with the AHA guidelines) would produce optimal results. Volume

recommendations in the setting in which multiple doses of drug may be given (e.g., out-of-hospital arrest without peripheral venous access in a patient who may be facing long transport) are lacking. In this setting it may be appropriate to limit the diluent volume for an individual adult administration to 5 mL.

**Appropriate Diluent**

Due to their availability during emergency situations, both normal saline and distilled water have historically been the diluents of choice for ET drug administration. The AHA Adult Advanced Cardiac Life Support Guidelines recommend either diluent, noting that "endotracheal absorption is greater with distilled water than with normal saline, but distilled water has a more negative effect on Pa$_{O2}$ ." [14]

The use of saline is supported by the canine study of Greenberg and associates, which reported that administration of normal saline via the ET route produced fewer detrimental effects on arterial blood gases than did distilled water. [27] The safety of endotracheally administered normal saline is further supported by a study in which no changes in pulmonary status (arterial blood gas, oxygen saturation, gross anatomy, or histology) were observed in dogs given lidocaine diluted with normal saline to total volumes of between 6 and 25 mL. [26] In contrast, Hahnel and colleagues studied absorption of lidocaine when administered endotracheally in either normal saline or distilled water and found that saline impaired Pa$_{O2}$ more than distilled water. [29] In addition, they reported higher serum levels when lidocaine was diluted with distilled water. They concluded that distilled water is probably a better diluent than normal saline. Finally, a study of ET epinephrine diluted with either normal saline or distilled water found no difference in arterial blood gases between either the solution or the control. [30] In this latter canine cardiac arrest model, survival rates for ET epinephrine in either diluent were equal to the rate with IV epinephrine. Hence, the optimal diluent is controversial; saline and distilled water both can produce pulmonary dysfunction when administered in excess.

**Technique for Endotracheal Drug Delivery**

Multiple techniques have been used to deliver medications via the ET tube, including direct instillation into the proximal end of the ET tube, ET administration via a catheter that extends just beyond the distal tip of the ET tube, deep endobronchial administration using a longer catheter, administration via ET tube monitoring ports, and injection through the side of the ET tube with a needle. The AHA Advanced Cardiac Life Support Guidelines for adults and children and the Neonatal Resuscitation Guidelines recommend the use of a catheter that extends beyond the distal tip of the ET tube to minimize drug adherence to the ET tube and to enhance alveolar delivery. [14] [15] [16] However, several studies have indicated that this practice may not be necessary.

Greenberg and Spivey instilled radiopaque contrast material directly into the proximal end of the ET tube and compared its distribution to contrast instilled via a catheter extending out the distal end of the tube. [30A] Their study revealed that both techniques were equally effective in distributing

the contrast agent to the peripheral lung fields as long as instillation was followed by five rapid manual hyperventilations. Although some studies have suggested that drug absorption with direct instillation into the ET tube is inconsistent during cardiopulmonary arrest, [19] [22] at least one case report has shown successful resuscitation using this method. [13] In addition, using a porcine cardiopulmonary arrest model, Jasani and coworkers showed no difference in resuscitation rates or physiologic responses between epinephrine administered by direct injection into the ET tube, via a catheter extending out the distal end of the ET tube, or via a monitoring lumen built into the sidewall of the ET tube. [31] In a related study, no difference was detected in resuscitation rates or plasma epinephrine levels when epinephrine was instilled during apnea vs instillation during the ventilator inspiratory cycle. [32]

However, in studies in which subjects had normal perfusion, conflicting results have been reported. In one study using female volunteers, no difference was found in plasma lidocaine levels when the drug was administered directly into the proximal end of the ET tube vs deeper administration into the trachea or lungs. [33] However, a later report by the same group demonstrated significantly higher plasma lidocaine concentrations when the drug was administered directly into the proximal end of the ET tube. [34] In direct contrast, when administered to dogs, significantly higher plasma epinephrine levels were obtained via the deep endobronchial route vs direct ET tube instillation. [35]

Given these conflicting studies, use of a catheter to enhance deep pulmonary delivery seems reasonable. However, if a catheter is not readily available, alternative routes appear justified.

**Effects of Hypoxia, Hypotension, and Cardiopulmonary Arrest**

Several authors have examined the effect of cardiopulmonary arrest on the absorption of medications administered via the ET route. Although concern existed that medication absorption might decrease in states of hypoxia or low blood flow, studies reveal the opposite to be true. In a hemorrhagic shock model, Mace demonstrated that higher plasma lidocaine levels were obtained via the ET route during shock than during nonshock states. [36] In a lamb model, when epinephrine was administered endotracheally, higher plasma epinephrine levels were achieved during hypoxia-induced low pulmonary blood flow than during baseline, normal pulmonary blood flow. [37] Finally, higher plasma lidocaine levels were initially observed when lidocaine was administered endotracheally to dogs with hypoxemia than to dogs that were not hypoxemic. [38] However, this study did not find a difference in the pharmacokinetics of lidocaine in hypoxemic and nonhypoxemic dogs when the drug was administered via the ET route.

Despite evidence indicating that cardiopulmonary arrest does not inhibit absorption of endotracheally administered medications, other studies have indicated that ET drugs are unreliable during cardiopulmonary arrest. [19] [21] [22] The latter studies serve to emphasize that ET drug administration should not be used in lieu of attempts to obtain definitive access to the systemic circulation. Therefore, ET drug administration should not be performed when another more direct means of accessing the central circulation

is available.

| |
|---|
| **TABLE 27-2** -- Endotracheally Administered Drugs Shown to Be Effective Experimentally and Clinically |
| Atropine |
| Diazepam * |
| Epinephrine |
| Lidocaine |
| Naloxone |

* See text--effective, but produced pneumonitis in one animal model.

## INDICATIONS

Endotracheal drug therapy is indicated whenever there is a need for emergent pharmacologic intervention and other access, either IV or IO, is not readily available. This most frequently occurs in patients with poor veins (e.g., patients receiving chemotherapy, dialysis patients, drug abusers, elderly patients), in patients with small or obscured veins (e.g., pediatric patients, burn patients, obese patients), or during cardiovascular collapse (e.g., cardiac or traumatic arrest).

Specific indications for the delivery of a drug endotracheally are the same as those for IV and IO administration. However, there are only a limited number of emergency drugs that can be given safely and effectively by the ET route (Tables 27-2 and 27-3) . Medications that have been administered endotracheally and found to be safe and effective in both experimental animal models and human studies or case reports include epinephrine,  atropine,  lidocaine, and naloxone.

Diazepam also has been shown to be effective. [46] [47] However, in one animal model, diazepam produced pneumonitis when 0.5 mg/kg was administered via the ET route. [48] Because diazepam is sparingly soluble in water, it is available only in a solution of

propylene glycol, ethanol, and benzyl alcohol. It is unknown if the reported pneumonitis was due to the direct effects of the diazepam or to that of the diluent.

In one case report, 5 mg of diazepam (i.e., approximately 0.1 mg/kg) was administered via a tracheostomy to an adult female, resulting in cessation of seizure activity within 2 minutes. [47] In this report, no change in arterial blood gas values or chest x-rays was noted over the ensuing 5 days. Whether the apparent lack of adverse pulmonary effects in this patient implies that diazepam can be safely used via the ET route is still speculative. The lower dose used (0.1 mg/kg) may have been a factor in reducing pulmonary damage. A further issue, given the low solubility of diazepam in water, is the ability to dilute stock diazepam solutions to a total volume of 10 mL (as is recommended for ET drug administration). A volume of 10 mL of propylene glycol or ethanol used as diluent may further compromise pulmonary function. Additional studies are needed to resolve these issues, but some authors have removed diazepam from their

| **TABLE 27-3** -- Endotracheally Administered Drugs Shown to Be Effective Experimentally but Not Proven Clinically |
|---|
| Midazolam |
| Metaraminol |
| Propranolol |

list of medications that can be given safely via the ET route. [49] [50]

Experimental studies of midazolam, [51] propranolol, [41] and metaraminol [52] in animal models suggest that these medications also may be effective when administered endotracheally, but no clinical studies have been conducted to date to verify these findings. It is of interest to note that in the study of midazolam, no pathologic changes were seen in lung sections after midazolam administration. [51] In addition, midazolam is available commercially in aqueous solution and can, therefore, be diluted with normal saline or sterile water for ET administration. Although clinical studies are needed for confirmation, midazolam may prove to be a viable alternative to diazepam when IV or IO access is not obtainable.

## CONTRAINDICATIONS

At present, the only true contraindication to the ET delivery of an appropriate drug is the presence of another form of access to the systemic circulation through which the needed drug can be delivered rapidly and effectively. The pharmacokinetics and pharmacodynamics of ET drugs delivered during various states of cardiovascular or pulmonary compromise are to a large extent unknown, and for that reason, more conventional routes of rapid and effective drug administration should be used when available.

No drug should be delivered by the ET route without experimental or clinical evidence to support its effectiveness and safety. A complete list of drugs that are contraindicated relative to delivery by the ET method is not available. Specific emergency medications that have been shown to be ineffective or unsafe when given via the ET route include sodium bicarbonate, isoproterenol, and bretylium. In a study using a canine model, sodium bicarbonate was shown to inactivate lung surfactant. [53] Isoproterenol, even when given in doses ten times the IV dose, failed to produce significant changes in arterial blood pressure or heart rate. [41] Studies of bretylium also indicate low serum levels after ET administration, even when administered at doses of 20 mg/kg.

## EQUIPMENT

Patients in need of ET drug therapy first require management of the airway, usually in the form of tracheal intubation. Once this procedure has been performed (see Chapter 2) , little other equipment is needed for the ET delivery of a drug. In the Procedure section that follows, several different techniques for ET drug administration are described. The equipment listed below is that required to perform any of the four different techniques described. This equipment is suggested for the ideal situation and/or technique. *At no time should drug delivery be delayed while searching for the "perfect" piece of equipment.*

1. Manual bag-valve ventilation device capable of delivering an $F_{IO_2}$ of at least 50%. In circumstances in which ET drug delivery is indicated, the patient's condition almost always warrants supplemental oxygen. Although the technique may not result in any significant deterioration in respiratory function, it is still advisable to administer additional oxygen after drug delivery. The bag-valve ventilation device also is used to deliver several rapid insufflations immediately after drug delivery to assist delivery of the drug distally, where it may be absorbed more rapidly and effectively. [30A]
2. A fine-bore catheter or special ET tube to deliver the drug at or beyond the distal end of the ET tube. The ideal catheter is at least 8 Fr in size and at least 35 cm (14 in.) in length. The most effective length or diameter of the catheter is unknown, but it is recommended that the catheter be long enough to protrude past the distal end of the ET tube. The diameter of the catheter should be large enough to allow rapid delivery of a 10 mL volume of solution. Several different types of tubes and catheters commonly available in the emergency department can be used for this

purpose:

A 16-ga central venous pressure or cutdown catheter. Most of these are only 30 cm in length, however, and the proximal end of the ET tube requires shortening if the catheter is to protrude past the distal end of the ET tube.

An 8 or 10 Fr polyethylene pediatric feeding tube (e.g., Argle, St. Louis). These tubes are much longer than needed and should be cut to the desired length to reduce dead space. Luer-Lok-type syringes and IV adapter locks both fit nicely onto the proximal end of the tube.

An 8 Fr (or larger) pediatric pulmonary suction catheter without the control port. Because this catheter is designed to extend past the tip of the ET tube, it is an ideal length. However, with some brands, it is difficult to attach a syringe or IV adapter lock after the suction control port is removed.

Alternatively, two ET tubes with built-in ports are available that allow ET instillation of drugs without removing the bag ventilation device. Unless these special tubes are used for all patients requiring intubation, the major drawback is the obvious need to decide prior to intubation if IV access is expected to be a problem. In these cases, the patient can be intubated using one of these tubes in anticipation of the need for ET drug administration. The available tubes include the following:

Endotracheal tubes designed for bronchoscopy (e.g., Hi-Lo Jet Tracheal Tube, Mallinckrodt Medical, St. Louis). These tubes have two additional ports, one used for monitoring or irrigation (opaque lumen) and one used for jet ventilation (transparent lumen). They are available in adult and pediatric sizes (cuffed and uncuffed). In a porcine cardiopulmonary arrest model, successful resuscitation using this tube was comparable to resuscitation using other forms of ET drug administration. [31] The major disadvantage to this ET tube is the need to be familiar with the specific ports prior to use. Determining which is the irrigation port if one has never seen the tube previously could prove to be time consuming. In addition, the port requires placement of an IV adapter lock to use prefilled syringes (in which most emergency medications are now supplied).

The EMT tracheal tube (Mallinckrodt Medical, St. Louis). This tube is designed specifically for ET drug administration. The instillation lumen opens into the tube at the eye, approximately 1 cm from the end of the tube. The injection port has an IV adapter lock, which makes it amenable to use with prefilled syringes.

The EMT tube is not available in pediatric sizes, and although its design is theoretically sound, no studies have been completed at this time that demonstrate an advantage over ET drug delivery using other techniques with standard ET tubes.

1. An IV adapter lock. This can be placed as needed onto the proximal end of the ET tube or catheters described previously to convert them for use with prefilled

syringes. This adapter is usually unnecessary if a standard syringe is used.
2. A 10 to 20 mL syringe, preferably a Luer-Lok type, big enough to deliver the desired volume of drug solution plus an additional 5 mL of air. Unfortunately (for ET drug therapy), most of the medications now prescribed for emergency situations come in prefilled syringes with built-in needles (Fig. 27-1) . This type of apparatus usually does not allow one to draw up an additional volume of air to empty the catheter of solution. Also, the needle on the prefilled syringe requires an IV adapter lock in order to utilize most catheters for injection.
3. Desired diluent solution. As previously described, the drug should be delivered in a final volume of 10 mL for adults and 1 to 2 mL for neonates and children. An adequate volume of diluent, usually normal saline, must be available. Distilled water is an acceptable alternative.
4. Desired medication to be instilled (see Tables 27-2 and 27-3) .
5. An 18- or 19-ga needle for use in drawing up the drug solution and/or for injecting the solution. Use of an 18-ga, 8.9 cm (3.5 in.) spinal needle is recommended for direct instillation of medications into the proximal end of the ET tube.
6. Alcohol wipes for cleaning vials and/or injection ports.
7. Gloves, mask, and eye protection. After instillation, the solution often refluxes out of the ET tube, making blood and body fluid precautions of paramount importance.

## PROCEDURE

Four procedures are described below. They are listed in order of generally accepted use, but definitive studies have not

**Figure 27-1** Example of a prefilled syringe, which is frequently used in endotracheal therapy.

clearly identified the best technique. Most important, the procedure of choice is the method that will deliver the medication to the patient in the most timely fashion. As stated previously, one should not delay drug administration to find the perfect piece of equipment or diluent (normal saline or distilled water are both acceptable in most cases).

It is assumed for all procedures that vials and IV adapter locks will be cleaned with alcohol wipes prior to use and that universal precautions will be practiced at all times. In addition, all patients require prior intubation (see Chapter 2) , and the ET tube should be secured to prevent the tube from being expelled if the patient coughs. If present, the cuff of the tube should be inflated.

**Use of a Catheter**

Begin ventilations with the bag-valve ventilation device with supplemental oxygen while the required drug dose is drawn up in either a 10- or a 20-mL syringe. Dilute the drug with normal saline as needed to attain a total volume of 5 to 10 mL (for adults) or 1 to 2 mL (pediatric and neonates). Draw back the plunger to add 5 mL of air to the liquid in the syringe. If the drug to be delivered is in a prefilled syringe, place an IV adapter lock on the catheter. The syringe can be attached to the catheter at this time or once the

catheter has been placed within the ET tube (Fig. 27-2) .

Interrupt the connection between the proximal end of the ET tube and the bag-valve ventilation device. Place the catheter within the lumen of the ET tube, ideally in such a manner that the distal end of the catheter extends 1 cm beyond the distal end of the ET tube. For the catheter to reach deep enough, the proximal end of the ET tube may need to be cut to a shorter length. Hold the proximal ends of the catheter and the ET tube at all times during the procedure. If it has not already been done, attach the syringe to the catheter. If external cardiac compressions are being performed, *they should be interrupted during drug delivery.* Inject the drug solution rapidly and forcefully through the catheter into the trachea followed by the 5 mL of air needed to flush the catheter of any remaining drug solution. Promptly remove the syringe and catheter from the ET tube. If the patient makes an effort to cough, place a thumb over the opening of the ET tube to prevent expulsion of the solution. As soon as possible after drug delivery, reconnect the bag-valve ventilation device with supplemental oxygen to the ET tube. Deliver five rapid ventilations to promote distal dispersion of the drug. Resume chest compressions if necessary.

**Direct Instillation into the Endotracheal Tube**

When special ET tubes or catheters are not available, the drug may be instilled directly into the proximal end of the ET tube. Studies using this technique have diluted the medication with normal saline and also flushed the tube with normal saline after the drug is instilled. [31]

While the patient is being ventilated, draw up the desired drug into a syringe (or use a prefilled syringe). Because no catheter is used, there is no need to draw up an additional volume of air into the syringe. Dilute the drug to a final volume of 5 mL (adults) or 1 mL (neonates and children) with normal saline. Attach an 18- or 19-ga needle (some authors recommend using an 8.9 cm [3.5 in.] spinal needle).

**Figure 27-2** Use of a central venous catheter for endotracheal drug delivery.

In a second syringe, draw up an equal volume (i.e., 5 mL adults or 1 mL neonates or children) of normal saline alone and attach an 18- or 19-ga needle. This will be used to flush the ET tube after drug instillation.

Interrupt the connection between the proximal end of the ET tube and the bag-valve ventilation device. Insert the needle of the first syringe (drug) into the proximal opening of the ET tube. If not using a Luer-Lok-type syringe, hold the proximal end of the needle with one hand to prevent loss of the needle into the tube. Discontinue cardiac compressions, and inject the drug solution rapidly and forcefully. Flush the tube immediately with the diluent in the second syringe. If the patient makes an effort to cough, place a thumb over the opening of the ET tube to prevent expulsion of the solution. Reattach the bag-valve ventilation device and deliver five rapid insufflations.

Resume chest compressions if needed.

**Use of Endotracheal Tubes with Irrigation/Drug-Delivery Lumens**

Unless these ET tubes are used for every intubation, their usefulness in emergent ET drug delivery is dependent on the provider's recognition prior to intubation that IV access will be problematic. If this is recognized, intubate the patient using an ET tube with monitoring lumen (e.g., Hi-Lo Jet ET tube) or with an EMT tracheal tube. While ventilating the patient with supplemental oxygen, draw up the required drug dose in either a 10- or a 20-mL syringe with an 18- or 19-ga needle attached (or use a prefilled syringe). If necessary, dilute the drug with normal saline or distilled water to attain a total volume of 5 to 10 mL (for adults) or 1 to 2 mL (pediatric and neonates). Draw back the plunger to add 5 mL of air to the liquid in the barrel of the syringe. Attach the syringe or insert the needle into the IV adapter lock on the ET tube monitoring/irrigation lumen (Hi-Lo Jet tube) or on the drug-delivery lumen (EMT tube). Discontinue chest compressions and rapidly and forcefully inject the drug solution during the inspiratory phase of ventilation (i.e., when the bag is squeezed). Deliver five rapid hyperventilations with the bag-valve ventilation device and resume chest compressions if necessary.

The advantage of this method of drug delivery is that it does not require interruption of the connection between the bag-valve ventilation device and the ET tube. In addition, it offers the theoretical advantage of allowing drug delivery during the inspiratory phase of ventilation, although at least one study has shown that this is not an important factor. [32]

**Injection Through the Endotracheal**

Tube Wall

This method of drug delivery has not yet been evaluated scientifically but has been used clinically. [55] [56] While the patient is being ventilated, draw up the desired drug into a syringe with an attached 18- or 19-ga needle (or use a prefilled syringe). No additional air needs to be added to the syringe. Insert the needle into the side of the ET tube proximally (Fig. 27-3) . Discontinue chest compressions and rapidly and forcefully inject the drug solution into the ET

**Figure 27-3** Method of drug injection through the endotracheal tube wall.

tube during the inspiratory phase of ventilation (i.e., when the bag is squeezed). Deliver five rapid hyperventilations with the bag-valve ventilation device and resume chest compressions, if necessary. As with ET tubes with drug-delivery lumens, this technique requires no interruption of the connection between the bag-valve ventilation device and the ET tube. In addition, placing an IV adapter lock on the needle allows it to be left inserted in the ET tube for use with additional medications.

# COMPLICATIONS

Reported complications of ET drug therapy are rare, due in part to the infrequent use of this technique. In addition, because most or all of the patients who receive ET drug therapy are in cardiopulmonary arrest or are otherwise critically ill, it is difficult to ascertain if an adverse outcome is the result of the therapy or the patient's underlying condition.

With regard to the techniques of ET drug administration, no serious complications have been reported. A theoretical complication is the loss of a needle or catheter down the ET tube, but this can be prevented by specifically holding the catheter or needle while instilling the drug. Thus the techniques of ET drug administration seem to provide a safe method of drug delivery.

Following ET drug administration, the administered emergency drugs may produce adverse effects. Epinephrine administered during CPR has been noted to produce prolonged hypertension, tachycardia, and arrhythmias after the return of a perfusing rhythm in both animal models [23] and human case reports. [13] It appears that this side effect is related to the depot effect, in which larger doses of drugs administered endotracheally are released slowly over time (similar to an IV drip). In addition to epinephrine, atropine and lidocaine also exhibit a depot effect when administered endotracheally. [53] No serious long-term sequelae have been reported, however, due to this effect.

A second area of potential concern with ET drug therapy is a transient decrease in arterial oxygen content during or after drug delivery. As discussed previously, if total volumes are maintained between 5 and 10 mL in adults, the effect on pulmonary function appears minimal. It should be remembered, however, that any decrease of $Pa_{O2}$ in a critically ill patient, regardless of how small or how transient, may have deleterious effects. The potential for this adverse effect should always be considered when administering drugs by the ET route, and supplemental oxygen should always be administered in an effort to improve oxygenation and offset any transient drop in arterial oxygen content that might develop.

## CONCLUSION

It is important that physicians be familiar with ET drug therapy because it may be a life-saving procedure in some emergent situations. The great variability in the effectiveness of this procedure, however, mandates that it be used only when IV or IO access is not available. Little information is available on the pharmacokinetics, pharmacodynamics, and effectiveness of medications administered via the ET route during cardiopulmonary arrest. Consequently, the ideal dose, diluent, volume, and technique have yet to be determined. Despite these limitations, ET drug therapy has been shown to be beneficial for some patients. If other access is not available, appropriate medications should be delivered via the ET route using the procedure that will ensure delivery to the patient in the shortest period of time.

# Chapter 28 - Autotransfusion (Autologous Blood Transfusion)

**Thomas B. Purcell**

Among the various afflictions that may jeopardize human life and well-being, traumatic injury affects men and women during their most productive years. [1] This fact, coupled with increasingly efficient and rapid emergency transportation systems, has resulted in growing numbers of these victims arriving at emergency facilities in potentially salvageable condition. The ensuing urgent demand for blood has often exceeded the immediately available supplies of homologous banked blood. Successful approaches to this problem have included earlier hemostasis (i.e., definitive surgery), use of volume expanders (crystalloid, colloid), and autotransfusion. Approaches currently under investigation include the use of hypertonic saline and manufactured blood substitutes.

Autotransfusion may be defined as "collection and reinfusion of the patient's own blood for volume replacement." [2] Emergency autotransfusion most often involves collection of shed blood from a major body cavity, usually the pleural space (hemothorax) and occasionally the peritoneal space. Autotransfusion in the emergency department is generally limited to acute hemothorax with clinically significant hypovolemia. The following discussion examines the advantages and potential complications of emergency autotransfusion, patient selection, available equipment, and procedural technique for several widely used devices.

## BACKGROUND

Autotransfusion has a relatively extended tradition in the Western medical literature. An early report was published in 1818 by Blundell, [3] an English practitioner who reflected on the possibility of reinfusion of shed blood after witnessing a woman exsanguinate from uterine hemorrhage. His subsequent work with autotransfusion of shed blood in dogs suggested the clinical feasibility of the procedure. In 1886 Duncan [4] used the technique without notable ill effects while reinfusing blood shed during an amputation. In 1914, the use of the technique in ectopic pregnancies was popularized by Thies, [5] and 3 years later Elmendorf [6] published a description of the first case of autotransfusion from traumatic hemothorax. Also in 1917, Lockwood [7] used the procedure for the first time in the United States during a splenectomy. In 1922 Burch [8] reviewed 164 cases of autotransfusion published over the preceding 8 years in the European medical literature, primarily out of Germany.

The discovery of ABO blood typing at the turn of the century and the institution of blood banks in the 1930s led to the almost exclusive use of homologous blood up to and following World War II. Interest in autotransfusion concomitantly declined, and only sporadic reports appeared in the literature during this period. During the 1960s and 1970s, cardiopulmonary bypass surgery generated extensive data regarding intraoperative retrieval of large quantities of blood for reinfusion. Concurrently, the Vietnam War created tremendous new demands for readily available blood in areas

remote from conventional reserves of homologous bank blood. Thus, revitalized interest, coupled with growing experience, generated the early publications of such investigators as Dyer and associates, [9] Klebanoff and colleagues, [10] [11] and Symbas and coworkers, [12] which initiated the "new era" of autotransfusion.

## ADVANTAGES

The advantages of autotransfusion over banked blood transfusion in patients who are hypovolemic from traumatic blood loss include the following:

1. There is rapid availability to the patient (collection and initiation of reinfusion can be accomplished within minutes).
2. Blood compatibility is not a problem, which avoids both untoward transfusion reactions and the problem of crossmatching uncommon blood types.
3. There is immediate reinfusion of normothermic autologous blood and consequent reduction in the life-threatening complications of hypothermia created by the administration of room-temperature fluids. [13]
4. The risk of indirect patient-to-patient transmission of infectious diseases (e.g., hepatitis, malaria, cytomegalovirus, or human immunodeficiency virus) is eliminated. [14]
5. Levels of 2,3-diphosphoglycerate (2,3-DPG) have been found to be significantly higher in autotransfused red blood cells (RBCs) than in stored homologous cells. [15] [16]
6. There are no direct complications of hypocalcemia or hyperkalemia. [17] [18]
7. There is less risk of inadvertent circulatory overload and adult respiratory distress syndrome (ARDS). [19]
8. Autotransfusion allows preservation of limited stores of banked blood, thereby ensuring their availability for other uses. [14]
9. Autotransfusion lowers the cost of medical care. [14] No blood drawing, typing, or crossmatching is required; thus, time, money, and personnel expenditures may be conserved. Davidson [20] reported the cost of autologous blood to be three- to eight-fold cheaper per unit compared with banked blood.
10. It may be acceptable to those patients whose religious convictions prohibit transfusions with homologous blood. Techniques for intraoperative or extraoperative collection of autologous blood that involve *blood storage* or *reinfusion of shed blood* are objectionable to Jehovah's Witnesses. Nonetheless, salvage when extracorporeal circulation is uninterrupted may be acceptable to many members of that religion.

## PATIENT SELECTION

### Indications

In general, all victims of severe trauma, whether blunt or penetrating, should be considered potential candidates for

autotransfusion. More specifically, Reul and colleagues [22] have described three

categories of patients for whom emergency autotransfusion is suitable. First, the ideal candidate is the patient who has sustained blunt or penetrating chest trauma, with an acute chest tube collection of 1500 mL or more of blood. A second category is the patient with immediate need but with <1 whole-body blood volume loss for whom no homologous blood, or only limited quantities, are available because of the urgency of the situation, a blood bank shortage, or a difficult crossmatch. Under these circumstances, Reul and coworkers used autotransfusion regardless of the type of injury or degree of contamination. A third category is the patient with massive blood loss (less than one whole-body blood volume) for whom autotransfusion can serve as a supplement to homologous replacement. O'Riordan [18] adds a fourth category: the trauma patient who urgently requires blood transfusion and whose religious convictions prohibit homologous transfusion. Barriot and colleagues reported their experience with hemothorax autotransfusion by the emergency medical services of the Paris Fire Brigade and demonstrated that this technique may be effectively used in the out-of-hospital setting.

It seems clinically reasonable to consider use of autotransfusion in all suitable patients who have a hemothorax and require even minimal blood replacement. In situations in which the need for homologous blood transfusion is borderline, autologous blood can be readily reinfused without the risk of complications associated with the use of banked blood. In our own emergency department, we have simplified the *indications* for initiating collection for possible autotransfusion to the following:

1. Blunt or penetrating chest trauma with significant hemothorax (500 mL or more) as suggested on a chest film
2. Multiple trauma with shock of uncertain etiology in a patient for whom immediate (prior to chest film) tube thoracotomy is contemplated
3. Emergency thoracotomy
4. Any hemothorax when there is an urgent need for blood and the patient's religious beliefs prohibit homologous transfusions

**Contraindications**

Reul and coworkers [22] also suggested the following 4 general contraindications to the use of emergency autotransfusion:

1. The presence of malignant lesions in the area of traumatic blood accumulation.
2. Known renal or hepatic insufficiency.
3. Wounds older than 4 to 6 hours (because of the theoretic problem of bacterial overgrowth).
4. Gross contamination of pooled blood, usually as a result of trauma of the gastrointestinal tract. They added, however, that "the presence of any of these contraindications was occasionally overruled by the lack of available (banked) blood."

Huth and colleagues [25] found that major combined pancreatic-proximal intestinal injury also may constitute a contraindication owing to the potential for systemic reinfusion of

large amounts of pancreatic enzymes.

On the other hand, the National Blood Resource Education Program Expert Panel [26] has limited contraindications to active infection or gross contamination and the possibility of malignant cells in the salvaged blood. Investigation has confirmed that even when using systems that separate and wash cells, tumor cells will be resuspended and reinfused to the patient. [27]

Several investigators believe that the reinfusion of limited amounts of possibly contaminated blood from the peritoneal cavity may be accomplished with an acceptable risk, [28] [29] [30] but the current consensus is that exsanguinating hemorrhage is the only acceptable indication for autotransfusion when there is recognized intestinal contamination. Klebanoff, [31] on the other hand, believes that autotransfusion has "no place" when there is extensive fecal or urinary contamination of the pooled blood. Thus, the advisability of autotransfusing possibly contaminated blood from the peritoneal cavity remains controversial (see Complications).

## EQUIPMENT AND MATERIALS

### Autotransfusion Units

Symbas and associates [32] described a simplified collection system using standard materials available in any emergency department. After insertion of a chest tube, drainage is established into a standard chest tube bottle containing 400 mL of normal saline, maintaining a suction of 12 to 16 mm Hg. (Many of the disposable plastic thoracostomy collection devices now have the ability to act as reservoirs for autotransfusion in case the need arises.) If autotransfusion is required, the collected blood in the chest bottle is reinfused in one of two ways:

1. The chest bottle may be disconnected from the pleural drainage tube and simply inverted on an IV stand for reinfusion through a filter into the patient. During infusion, a second sterile chest bottle with saline is connected to the chest tube for continuing collection.
2. After disconnection from the pleural drainage tube, the chest bottle may be connected to a standard blood collection bag and the salvaged blood transferred to this bag for subsequent reinfusion in the conventional manner. Symbas [32] reported on more than 400 patients autotransfused by this method since 1966, with no adverse effects attributable to the procedure.

Von Koch and associates [33] reported their experience with the Sorenson unit (now the Abbott Receptal system) in autotransfusing an average of 1000 mL of salvaged blood in each of 30 trauma patients. They found this unit could be assembled quickly and was easily operated, and its use resulted in minimal air-blood interfacing (a source of hemolysis). They also described the unit as "efficacious, inexpensive, cost effective and safe." Davidson [20] described in detail the step-by-step use of this system in the emergency department and characterized it as "simple to use, efficacious, cost effective and safe" for the emergency setting. Autotransfusion using this device is described in

the following section.

A unit introduced more recently, the Pleur-Evac autotransfusion system (Deknatel, Inc., Fall River, Massachusetts), is similar in concept to the Abbott Receptal system but is designed to be installed directly in series with a standard Pleur-Evac underwater seal drainage system. A disadvantage of this unit is the smaller collecting capacity (1200 mL vs 1900 mL in the Abbott Receptal system). Advantages include an overflow accommodation, which sends blood in

excess of the 1200-mL collection bag capacity automatically into a spillover collection chamber of the main Pleur-Evac unit, and an ability to monitor the patient for air leaks during collection without changing systems.

Other units suitable for use in the emergency department have recently entered the market and are described below in the Procedure section.

The Haemonetics Cell Saver (Haemonetics Corp., Natick, Mass) aspirates the patient's blood into a reservoir and brings the blood, after it has been anticoagulated, through a special suction line to a centrifuge. The centrifuge spins off the supernatant fluid, which consists of plasma that contains hemolyzed cells, free hemoglobin, fat cells, electrolytes, anticoagulant, and contaminants. When the hematocrit of the remaining blood approaches 55 to 65%, normal saline washes it clear of hemolyzed cells, and the packed and washed cells are then reinfused. Major disadvantages of the device, in the opinion of Mattox, [34] are its complexity, requiring a specially trained technician for its operation, and its cost. Use of the Cell Saver system offers the theoretical advantage of avoiding reinfusion of activated clotting factors. Brewster and colleagues believe that "this advantage is achieved at the expense of an earlier dilutional decrease of coagulation factors, which are totally lost with discarded plasma and wash fluid." [35] Therefore, the system may require the use of fresh frozen plasma and additional colloid, such as albumin, to replace the discarded plasma volume, although this is generally not required with lower infusion volumes. [36] This device is generally not suited for use in the emergency department.

Two highly simplified techniques have been described. Schweitzer and coworkers [37] reported successful autotransfusion in dogs by means of a chest tube connected to a Heimlich flutter valve (Bard-Parker, Rutherford, NJ). The valve is connected in turn to a 1900-mL Abbott Receptal blood collection bag. Drainage is entirely by gravity; no suction is applied. Similarly, Barriot and colleagues [23] [24] described the European experience with a device called a *Hemotraum* adapted for prehospital autotransfusion. Blood from the chest tube fills a 750-mL sterile bag by gravity via a 120-mum micropore filter. When full, the bag is clamped and disconnected, and the collected blood is reinfused through a 50-mum micropore filter. While this blood is being transfused, a second sterile bag is connected to the chest tube. No anticoagulant is used. Although neither of these techniques has undergone clinical trials in the United States, they do serve as indications of possible future trends in emergency autotransfusion in the battlefield, the small rural hospital, or the prehospital care setting.

**Blood Filters**

*Some form of in-line filtration is advisable during reinfusion of blood products* to reduce the danger of microembolization and resulting pulmonary insufficiency. Controversy continues regarding the relationship between the presence of microaggregates and the development of ARDS [38] ; however, most investigators advise some form of micropore filtration during emergency autotransfusion. Pore size seems to be the only issue, and recommendations range from 20 to 170 mum. [40] [41] The majority of investigators believe that a pore size of 40 mum (microns) minimizes the risk of microembolization without undue elevations in filtration pressures.

**Vacuum Suction**

*The level of vacuum suction used should be limited to minimize RBC hemolysis*. [35] Reul and colleagues [22] found that 5 to 10 mm Hg avoided hemolysis, but higher levels of suction could be used. Von Koch and associates [33] used 10 mm Hg, Davidson [20] used 20 to 40 mm Hg, Noon [40] used 30 to 60 mm Hg, and Brewster and colleagues [35] and Dyer and coworkers [9] found that levels below 100 mm Hg kept hemolysis to a minimum.

Suction pressure of 60 mm Hg or less has been preferred by most researchers for aspiration of hemothorax or hemoperitoneum, but in the operating room, adequate suction to maintain a bloodless surgical field may require a pressure well over 100 mm Hg. With most commercial systems, the manufacturer's recommendations specify a suction limitation of 150 mm Hg. [43] A second vacuum source of 300 mm Hg should be available for occasions when high-flow suction is required. [44] To minimize hemolysis, reducing the air-blood interface is as important as limiting the vacuum level. Therefore, suctioning should be from pooled blood whenever possible, with the suction tip kept well below the surface of the blood.

**Anticoagulation**

Anticoagulation of the aspirate during autotransfusion has been accomplished using several different methods. These include both local and systemic heparin, acid-citratedextrose (ACD), citrate-phosphate-dextrose (CPD), and normal saline (i.e., no anticoagulant). Local heparinization of the tubing and reservoir may lead to the formation of platelet microaggregates on the filter and in the line and systemic heparinization could lead to further life-threatening hemorrhage in an already bleeding patient.Therefore, *the use of heparin as an anticoagulant during emergency autotransfusion of the trauma patient is discouraged by most investigators*.

In several early studies, ACD was used as an alternative to heparin. [17] Raines and coworkers [41] found no clinical or laboratory evidence of intravascular coagulopathy after autotransfusion using ACD, even in patients who received more than 8000 mL of autologous blood. More recent studies report the use of CPD for extracorporeal anticoagulation. Some advantages of CPD are that it avoids the complications of

heparinization, [22] necessitates less volume as an anticoagulant, and results in less acidosis than does ACD. Reul and colleagues [22] found CPD to be well tolerated, even in large amounts.

Investigators have recommended a range of 25 to 70 mL of CPD per 500 mL of collected blood. A 1:7 ratio of CPD to blood has also been suggested. [45] [51] This compares favorably with the standard 67 mL of CPD per unit of banked donor blood. [22] Klebanoff [31] stated that CPD is the safest method of anticoagulation for autotransfusion and that the use of CPD avoids the problem of clot formation on the blood filter, thus maintaining higher platelet counts in reinfused blood.

Davidson has noted that for the average chest wound, added anticoagulant may not be required. [20] Blood retrieved from pleural and abdominal cavities frequently will not clot because it is devoid of fibrinogen, [53] likely because moderate rates of bleeding allow time for defibrination by contact with

serosal (pleural and peritoneal) surfaces and by mechanical agitation from respiratory and cardiac movements. Dog studies have documented pleural deposition of fibrin following experimental hemothorax, further substantiating this mechanism as the cause of hypofibrinogenemia in collected blood. [54] Others [12] [24] [55] report corroborating clinical findings and recommend simple reinfusion through a filter without any anticoagulant.

However, wounds of the great vessels may bleed at a rate that allows coagulable blood to enter the collection reservoir, clotting off the entire system. [20] [33] [51] In such an instance, an anticoagulant, specifically CPD, is appropriate. Thus, accepted guidelines generally include the use of CPD, which itself undergoes such rapid metabolism that anticoagulation is confined, to a large degree, to blood in the autotransfusion apparatus. Rarely, excessive use of CPD can cause citrate intoxication because of chelation of calcium and subsequent cardiac dysrhythmias. Use of insufficient or outdated CPD may result in clotting of collected blood.

# PROCEDURE

Mattox [34] set forth the properties of the ideal autotransfusion device: (1) easy and quick assemblage, (2) cost-effectiveness, (3) easy operation, (4) in-line microfiltration, (5) minimization of air-fluid interfaces, and (6) simple anticoagulation technique. Several devices currently being marketed conform to these specifications, and these are discussed in detail.

### Abbott Receptal Disposable Suction Liner System

The Abbott Receptal autotransfusion system (Abbott Laboratories, Abbott Park, IL 60064) consists of a closed, rigid, nonsterile plastic canister into which a gas-autoclaved plastic bag is placed for blood collection. The canister can be mounted on an IV pole and connected to a vacuum regulator valve for control of negative suction pressure. The collection bag is placed in-line with disposable collection tubing that has a separate inlet

valve for admixture of anticoagulant and aspirated blood. This inlet is connected via sterile tubing to a bottle of CPD (Figs. 28-1 (Figure Not Available) and 28-2) (Figure Not Available) .

**Collection**

1. To collect autologous blood from a hemothorax, first open the included "Trauma Drainage Tubing Set" containing one 36 Fr chest tube, latex drainage tubing ( *B* and *C*, Fig. 28-1) (Figure Not Available) , and a male-to-male connector *(D)*.

    While tube thoracostomy is being performed in the usual manner, the burette set *(A)* is connected to the CPD bottle, and the burette is filled with 150 mL of CPD.

2. Connect the yellow-tipped *(E)* end of the latex drainage tubing (the end with the side port) to the inlet port *(F)* of the red liner cap attached to the collection canister (see Figs. 28-1 (Figure Not Available) and 28-3) (Figure Not Available) . Then remove the protective cap from the side port and connect the anticoagulant (CPD) administration line (see Fig. 28-2) (Figure Not Available) . Prime the liner with 50 mL of CPD from the burette.
3. Connect the downstream suction hose ( *H*, Fig. 28-1) (Figure Not Available) to wall suction and turn wall suction to maximum. Be sure that the regulator on the autotransfusion stand does not exceed the preset 60 mm Hg during collection (100 mm Hg in special situations such as post-thoracotomy); otherwise, excessive hemolysis of RBCs may result.
4. When the chest tube is in place, connect the latex drainage tubing and begin collection. During collection, stay ahead of the accumulating blood volume with the CPD in 50-mL increments. Always keep the ratio no less than 1 part CPD to 10 parts blood (1:7 ratio of CPD to blood is recommended by the manufacturer); otherwise, the collected blood may clot, especially with massive ongoing hemorrhage. Do not overfill the liner bag; it will overflow, spilling blood into the regulator valve.

**Reinfusion**

1. Prepare a standard Y-type blood infusion line with a high-capacity 40-mum in-line filter, prime the line and filter with normal saline, and connect it to a large-bore IV access (14-ga or larger) (Figs. 28-4 (Figure Not Available) and 28-5) (Figure Not Available) .
2. When the liner bag is full, temporarily clamp the chest tube, discontinue suction, and remove the yellow end of the latex drainage tubing *(E)* from the red liner lid. The liner lid tubing connector *(J)* is now removed from the canister tee *(K)* (Fig. 28-6) (Figure Not Available) and connected to the inlet port *(F)* of the liner cap, thus sealing the top of the collection lid (Fig. 28-7) (Figure Not Available) .
3. Remove the liner assembly from the canister by pushing upward on the thumb tab (Fig. 28-7) (Figure Not Available) , lift out the liner bag, invert the bag, and unscrew the protective cap *(N)* over the bottom stem of the liner. Now insert the free recipient arm ( *L*, Fig. 28-4) (Figure Not Available) of the Y-tube infusion line into the stem of the collection bag (see Fig. 28-4) (Figure Not Available) , and

hang the liner bag on the IV stand by the attached tab (M) (see Fig. 28-5) (Figure Not Available) . Before infusion, briefly disconnect the liner lid tubing connector ( *J*, Fig. 28-5) (Figure Not Available) , *vent all air from the bag* (to eliminate the possibility of air embolism), and then reconnect it to the inlet port.

4. Gravity flow, manual squeezing of the liner bag, or an in-line roller pump may be used to hasten reinfusion. Although some reports [20] [33] mention the use of encircling pneumatic blood pumps during reinfusion, Abbott cautions that such devices may damage the pump or rupture the liner bag.

5. During reinfusion, autologous blood collection may be continued with a second liner bag. Be sure that the new liner bag is fully extended before placing it into the canister (Fig. 28-8) (Figure Not Available) . If the bag is crumpled at the top of the canister, blood may be sucked directly into the regulator valve. Insert the new liner into the canister, and snap the lid in place with the thumb tab directly over the canister tee (Fig. 28-9) (Figure Not Available) . The unit is now ready for collection assembly as previously outlined.

## Boehringer Autovac System

A unique advantage of the Boehringer Autovac (Boehringer Laboratories, Inc., Norristown, PA 19401) is the optional integral vacuum regulator built into the cap. This feature allows direct attachment to a wall vacuum without an external regulator. It automatically limits applied suction to a

**Figure 28-6** (Figure Not Available) Preparation for reinfusion. Removal of liner lid tubing connector (J) from canister tee (K). Inlet port (F) is shown. *(Courtesy of Abbott Laboratories, Abbott Park, IL 60064. Reproduced by permission.)*

maximum of 100 mm Hg, thereby minimizing RBC hemolysis. The collection line has been enlarged to ¼-inch in diameter, which decreases the surface area to which RBCs are exposed and decreases blood velocity as it travels through the line (thereby further limiting hemolysis). The number of connections on the collection line has been reduced to 1, and the male connector has a skirt shield, reducing the chances of inadvertent contamination through contact with nonsterile surfaces. The collection bag is prepackaged within a disposable rigid plastic canister and will hold up to 1000mL of collected blood. Inside the collection chamber is a shutoff system consisting of a hydrophobic filter which, upon contact with blood or any liquid, quickly occludes preventing overflow into any downstream component (e.g., wall suction regulators).

### Collection

1. First, the vacuum line is attached to the connector on the canister (Fig. 28-10) (Figure Not Available) . Models not equipped with the optional integral regulator must be attached to a wall regulator that has been set at no greater than 100 mm Hg vacuum.

2. The canister is anticoagulated by injecting anticoagulant through a port on top of the canister. The Boehringer Company recommends (and offers for sale) anticoagulant citrate dextrose solution A (ACD-A), a solution containing 0.73 g of citric acid, 2.2 g of sodium citrate and 2.45 g of dextrose per 100 mL. Initially 40

mL is injected into the bag, and as collection proceeds, ACD-A is added to maintain a proportion of 1:5 to 1:10 ACD-A to blood.
3. During collection, the canister is periodically agitated to facilitate mixing of blood and anticoagulant.
4. When full, blood will come into contact with the hydrophobic filter at the top of the canister, automatically shutting off the system.

**Reinfusion**

1. Engage both slide clamps at the top of the canister.

   Both collection and vacuum lines are now occluded, creating a closed system.

2. Disconnect the canister from suction and collection lines. If further collection is desired, another canister can now be attached to these lines.
3. The flexible blood bag is now removed from the rigid canister by first removing the white safety tape and then popping the lid from the lower portion of the canister.
4. While keeping the bag upright, the slide clamp over the vacuum connector is now temporarily loosened and the bag gently squeezed to expel any residual air out through the filter shutoff mechanism. Then the slide clamp is re-engaged.
5. The bag may now be attached to a blood administration set in the usual fashion (Fig. 28-11) (Figure Not Available) . The manufacturer recommends the use of an in-line microaggregate filter (20 to 40 mum).
6. An external pneumatic cuff around the bag may be used to increase flow rates, but cuff inflation pressures should not exceed 300 mm Hg.

**Figure 28-10** (Figure Not Available) Boehringer Autovac System, collection configuration. *(Courtesy of Boehringer Laboratories, Inc., Norristown, Pa. Reproduced by permission.)*

## Pleur-Evac Autotransfusion System

The Pleur-Evac autotransfusion device (Deknatel, Inc., 600 Airport Road, Fall River, MA 02720) consists of a sterile, single-use, disposable, rectangular-shaped, flexible polyvinyl

**Figure 28-11** (Figure Not Available) Boehringer reinfusion apparatus. *(Courtesy of Boehringer Laboratories, Inc., Norristown, Pa. Reproduced by permission.)*

chloride blood collection bag supported by a rigid metal frame. The bag and frame are designed to attach in series directly to a standard Pleur-Evac underwater seal drainage unit. On a newer version of the drainage unit, the water suction control chamber is replaced by a dry suction control with a dial that may be set to regulate suction between 0 and 40 cm $H_2$ O, regardless of the amount of source suction applied. Inside the collection bag in line with the incoming drainage tube is a 200-mum nylon mesh filter. Attached to the collection bag are two latex tubes, one (red tipped) for collecting shed blood from the drainage site and one (blue tipped) for connection to the Pleur-Evac drainage unit. An injection port is provided on top of the autotransfusion bag for the

addition of anticoagulant to collected blood.

**Collection**

1. The Pleur-Evac underwater seal drainage unit (Fig. 28-12) (Figure Not Available) is first prepared in standard fashion and connected to a source of suction. Slide the metal hanger over the patient drainage tubing port on the right-hand side of the unit (Fig. 28-13) (Figure Not Available) , and pull it down flush with the top and side of the drainage unit.
2. Unwrap a replacement autotransfusion system (ATS) bag and fit it into the wire frame provided (Fig. 28-14) (Figure Not Available) . Close both white clamps on the bag tubing, and place the frame and bag on the wire hanger.
3. Clamp the drainage tubing coming from the patient.
4. Remove the red protective cap from the collection tubing ( *B*, Fig. 28-12) (Figure Not Available) on the ATS replacement bag and connect it to the patient chest drainage tubing *(C)* using the red connector.
5. Remove the blue protective cap from the suction tubing *(D)* on the ATS replacement bag and connect to the underwater seal drainage unit using the blue connector.
6. Using a syringe and an 18-ga (or smaller) needle, inject anticoagulant through the rubber diaphragm *(E)* on the ATS bag cap. The manufacturer of the Pleur-Evac unit does not offer recommendations regarding specifics of anticoagulation of blood collected in the unit; however, the method outlined for the Abbott Receptal system may be applied to this unit as well (see step 4 under Abbott Receptal System--Collection).
7. Open all clamps; make sure all connections are airtight. The system is now operational.

**Reinfusion**

1. Prepare a standard Y-type blood infusion line with a high-capacity 40-mum in-line micropore filter, prime the line and filter with normal saline, and connect it to a large-bore IV access (14-ga or larger) (see Fig. 28-5) (Figure Not Available) .
2. When the ATS bag is full, use the negative-pressure relief valve (located on top of the drainage unit) (see Fig. 28-12 (Figure Not Available) *F*) to reduce excessive suction in the unit, and close the white clamps on the chest drainage tubing and ATS bag.
3. Disconnect all connections to the autotransfusion bag.
4. Attach the male (blue) and female (red) connectors on top of the autotransfusion bag to each other, and remove the bag from the drainage unit by spreading and disconnecting each metal support arm.
5. Slide the autotransfusion bag out of the wire frame and invert the bag so that the spike port ( *G*, Fig. 28-12) (Figure Not Available) points upward. Remove the protective cap. Insert the free recipient arm of the Y tube infusion line into the spike port using a constant twisting motion.
6. Invert the autotransfusion bag and suspend it from an IV pole (Fig. 28-15) (Figure Not Available) .
7. Reinfusion using the Pleur-Evac autotransfusion bag may be assisted using a

pneumatic pressure blood pump, not to exceed 150 mm Hg infusion pressure. However, if a pneumatic pump is used, all air in the bag must be removed before infusion to minimize the risk of air embolism. To accomplish this, the red and blue connectors may by disconnected temporarily, one clamp opened, and the bag slowly squeezed until all air is out of the unit. Then the clamp is closed again, and the red-to-blue connection reestablished.

8. During reinfusion, autologous blood collection may be continued with a second ATS bag, repeating steps 2 through 7 under Collection.

**Continuous Reinfusion**

The Pleur-Evac system may be set up for continuous collection and simultaneous reinfusion of collected blood as follows:

1. Set up a blood administration set, microaggregate filter, and IV pump.
2. Prime the filter, drip chamber, and administration set with normal saline and then remove the bag of saline.
3. Spike the autotransfusion collection bag and connect it to the blood administration set.
4. Use the IV pump to prime the filter, drip chamber, and infusion line with blood. Make sure no air is in the line.
5. Attach the primed administration set to the patient's IV line.
6. To prevent air from entering the system, keep at least 75 mL of blood in the collection bag at all times. If this is not done, air embolism may result. (When an infusion pump is used in this fashion, air embolism is a significant risk--see Nonhematologic Complications below).
7. If continuous reinfusion is used, maintenance of proper proportions of anticoagulant to collected blood may become problematic.

## Thora-Seal-III Autotransfusion Chest Drainage System

The layout of the Thora-Seal-III system (Sherwood Medical, St. Louis) (Fig. 28-16) (Figure Not Available) is similar to that of the Pleur-Evac system. Suction is limited by a water seal chamber to no more than 25 cm $H_2$ O (equivalent to 18 mm Hg). However, occlusion of the suction control portal ( *C*, Fig. 28-16) (Figure Not Available) above the chest drainage unit will create a closed system, allowing direct connection to wall suction and attainment of slightly

higher suction levels when desired. It should be noted that if this is done, suction must not exceed 40 mm Hg, since the reinforced collection bag *(D)* will begin to collapse above this level. Reinfusion may be assisted with a standard infusion cuff, but the manufacturer advises that reinfusion pressures be limited to no more than 150 mm Hg.

This system also may be set up for continuous reinfusion in a fashion similar to that described above. Caveats regarding the danger of air embolism and difficulty of maintaining proper proportions of anticoagulant to collected blood similarly apply.

**Thora-Klex Chest Drainage System with Autotransfusion**

The Thora-Klex system (Davol, Inc., a subsidiary of C. R. Bard, Inc., Cranston, RI 02920) (Fig. 28-17) (Figure Not Available) has an in-line configuration with a standard chest drainage system similar to the Pleur-Evac system. The Thora-Klex does not incorporate a water seal chamber. Instead, there is a suction control dial (which reads centimeters of water rather than millimeters of mercury) built into the top of the unit (Fig. 28-18) (Figure Not Available) . A negative pressure indicator within the system indicates at all times the degree of suction inside the chamber. The suction may be set up to 40 cm $H_2$ O (29 mm Hg) and a built-in suction relief valve limits absolute suction to a maximum of 50 cm $H_2$ O (37 mm Hg).

Design of the unit allows unimpaired collection even if the unit is accidently tipped over. The autotransfusion bag has a capacity of 1000 mL, and like the Thora-Seal unit, the system may be set up for continuous collection/reinfusion (the caveats mentioned above regarding potential for air embolism and difficulties with maintaining blood/anticoagulant proportions apply similarly to this system).

The collection bottle is used for reinfusion through a blood administration system (Fig. 28-19) (Figure Not Available) . The in-line filter should be changed when the blood flow into the collection bottle slows at maximum vacuum levels (Fig. 28-20) (Figure Not Available) .

**Additional General Autotransfusion Information**

1. Use each liner bag only once.
2. After reinfusing a total of 3500 mL, or 7 units, of autologous blood, it has been suggested that 1 unit of fresh frozen plasma be given for every 2 units (1000 mL) of autotransfused blood. [41]
3. To minimize risk from bacterial overgrowth, collected blood must not be allowed to stand for prolonged periods of time before reinfusion. Some authors [45] [51] [56] advise a limit of no more than 4 hours between collection and reinfusion. The age of collected blood should be calculated from the time of injury, and reinfusion of blood older than 4 to 8 hours should be considered hazardous. Because one is performing the procedure for significant hypovolemia in the emergency department, the collected blood should generally be transfused as soon as the collection bag is full.
4. If some or all of the collected blood becomes clotted in the liner bag, the blood should be discarded.
5. The blood filter used during reinfusion is changed as needed (usually after each 1000- to 2000-mL transfusion). In practice, the need for a new filter will become apparent as perfusion pressures increase and flow rates visibly slow down.

## COMPLICATIONS

Complications from autotransfusion, which generally are clinically insignificant if the

proper technique is followed and if <3000 mL of blood is reinfused, may be categorized as hematologic and nonhematologic (Table 28-1) .

## Hematologic Complications

The most frequent hematologic consequence of autotransfusion is thrombocytopenia. Samples taken from collected autologous blood generally show very low platelet counts. However, the number of platelets found in this blood is still significantly greater than that found in banked blood. [55] Until patients receive more than 3500 mL of autologous blood, in vivo platelet counts, although depressed in all patients, do not drop below 60 × 10 [9] /L [23] [32] [35] ; above this level, trauma surgery can be satisfactorily performed. [56] Although those platelets collected from autotransfusion reservoirs function abnormally when tested in vitro by aggregation or serotonin uptake and release, postinfusion samples drawn from patients aggregate normally. [41] However, at least one study conducted with dogs indicates that significant in vivo platelet

| **TABLE 28-1** -- Potential Complications of Autotransfusion |
|---|
| **Hematologic** |
| Decreased platelet count |
| Decreased fibrinogen level |
| Increased fibrin split products |
| Prolonged prothrombin time |
| Prolonged partial thromboplastin time |
| Red blood cell hemolysis |

| |
|---|
| Elevated plasma-free hemoglobin level |
| Decreased hematocrit level |
| **Nonhematologic** |
| Bacteremia |
| Sepsis |
| Microembolism |
| Air embolism |

dysfunction may appear when autotransfused volumes exceed an amount equivalent to 1 total blood volume. [16] Thus, platelet counts should be followed and significant thrombocytopenia treated with platelet infusion.

The most common coagulation factor abnormality after autotransfusion is hypofibrinogenemia, especially when the volume of autologous blood used exceeds 4000 mL. Because of the liver's capacity to replenish fibrinogen rapidly, the low postautotransfusion levels have not proved to be clinically significant. [22] [24] [51] Yet some investigators [22] believe that hepatic insufficiency is a relative contraindication to autotransfusion unless fibrinogen is supplemented.

Symbas and colleagues extended their work with laboratory dogs to the clinical study of victims of traumatic hemothorax. They found no clinical evidence of coagulopathy following autotransfusion as long as the volume collected and reinfused remained equal to or less than one half the patient's total blood volume. In those few patients who required a larger volume autotransfused, a proportional decrease in platelets and fibrinogen occurred, requiring subsequent correction with fresh frozen plasma and platelet packs. Other investigators [17] [20] have confirmed these findings and have shown that both platelet and fibrinogen levels return to normal by 48 to 72 hours, without replacement therapy. Similarly, elevations in prothrombin and partial thromboplastin

times, which were encountered routinely, were not clinically significant. These coagulation abnormalities also self-corrected within 48 to 72 hours. On the other hand, Schonberger and colleagues found that intraoperative autotransfusion of more than 800 mL shed blood may provoke a significant derangement of hemostasis. [58]

Hemolysis occurs with autotransfusion in part because of prolonged exposure of the cells to serosal linings of the traumatized body cavities. [59] Hemolysis also results from mechanical factors during collection and reinfusion, such as high vacuum pressures during aspiration, roller pump trauma, or excessive exposure to air-fluid interfaces. [22] An elevated plasma-free hemoglobin is a consistent finding in patients who have received autotransfusions.

Most samples of blood taken from the autotransfusion reservoir have free hemoglobin levels of <1.0 g/L, but some levels have been reported to be as high as 13.9 g/L. [50] Clinically, patient plasma-free hemoglobin values immediately after autotransfusion range from 0.1 to 1.0 g/L. When the binding capacity of haptoglobin is saturated and the threshold of tubular resorption of hemoglobin is exceeded, hemoglobinuria is seen. This threshold corresponds to a plasma-free hemoglobin concentration of 1.0 g/L. [60]

In the past it was believed that elevated levels of free hemoglobin following hemolytic transfusion reactions caused renal failure by precipitation of hemoglobin in and obstruction of renal tubules. However, more recent evidence suggests that the mechanism of renal failure in this setting is independent of free hemoglobin. Rather, it is a result of an antigen-antibody-induced intravascular coagulation that, compounded by vasoconstriction and hypotension, leads to renal ischemia. [61] Indeed, it has been shown that "massive hemoglobinuria may follow the transfusion of immunologically compatible hemolyzed RBCs with minimal symptoms and a benign outcome" and that isolated free hemoglobin levels of up to 130 g/L may be tolerated without renal compromise. [62] Even though renal failure as a direct consequence of autotransfusion has not been reported, [31] transient elevations in serum creatinine do occur, [18] and in the presence of shock and systemic acidosis, acute tubular necrosis remains a potential complication. [60] Some researchers [22] believe that renal insufficiency is only a relative contraindication to autotransfusion and may occasionally be overruled by the lack of available blood.

Finally, the hematocrit falls in direct proportion to the quantity of blood transfused, averaging an approximate drop of 10 to 20%. [22] [32] [29] However, nontraumatized RBC survival has been reported to be normal in all cases studied.

In general, although coagulation problems should be anticipated, they have not proven to be clinically important when volumes of autotransfused blood remain below 2000 mL in adult patients. [51] However, when reinfused volumes exceed 3500 mL, [41] laboratory evidence of a dilutional coagulopathy may become evident. When volumes of autotransfused blood are greater than the patient's total blood volume, animal studies suggest that there is increasing risk of a true consumptive coagulopathy. [16]

Recommendations regarding those infused volumes of autologous blood that should trigger replacement therapy of fresh frozen plasma or platelets range from 25% of total

blood volume (about 1250 mL in a 70-kg adult) [54] to 3500 mL. [41] Others advise reliance on laboratory tests and clinical findings rather than a set protocol based on volume exchanged. [51] Prudent clinical judgment dictates application of the more liberal guidelines for replacement therapy in those patients with extensive hepatic injury, intractable shock, or ongoing losses requiring immediate surgical intervention.

## Nonhematologic Complications

The theoretic risk of sepsis after the administration of potentially contaminated blood always exists within the nonsterile surroundings of the typical emergency department resuscitation area. Experience has shown this risk to be minimal after competent autotransfusion of an isolated hemothorax, [11] [17] [31] and there is *no* evidence to suggest that routine prophylaxis with systemic antibiotics is beneficial in this situation.

The issue of whether to autotransfuse shed intraperitoneal blood, however, is somewhat more complex than in the case with hemothorax. Recovery and reinfusion of hemoperitoneum per se has proved to be relatively safe, and Klebanoff and associates have concluded that "for contaminant-free conditions at least, as in ruptured ectopic pregnancies, ruptured spleen and liver, traumatic hemothorax, and vascular surgery, autotransfusion can be performed readily to reduce the need for homologous blood replacement." [11]

Reinfusion of autologous blood with possible enteric contamination is still considered by most investigators to be ill advised in all but the most desperate of circumstances, such as in the patient who will exsanguinate before homologous blood can be made available. If contaminated blood is infused, systemic antibiotics should be given. [30] [51] [63] Several small series describing collection and reinfusion of intestinally contaminated hemoperitoneum have been reported. [28] [63] [64] [65] [66] The overall mortality rate among this selected group approximates 20%, not unexpectedly high considering the severity of associated injuries. Experimental studies with dogs also show that autotransfusion of hemoperitoneum contaminated by intestinal contents, urine, or bile is tolerated. [11] [29]

Another complication, microemboli secondary to platelet microaggregation or fat emboli, has largely been eliminated by the use of micropore filters. [22] [35] [41] In most instances, during reinfusion of collected blood, there is a mild increase in screen filtration pressures, indicating the formation of microemboli trapped by the filter. [35] There has been no clinical evidence of pulmonary insufficiency or unexplained elevation of the alveolar-to-arterial oxygen gradient that might be attributed to the passage of microemboli beyond the micropore filter systems. [22]

Air embolism has been reported sporadically as a complication of autotransfusion. [17] [64] [67] [68] This uncommon but often fatal complication has been associated, in all cases reviewed, with autotransfusion systems using automated roller pump units in which the aspirate reservoir was inadvertently allowed to run dry. Air embolism with gravity or with a manually assisted technique is rare.

Available data indicate that although autotransfusion is not free of complications, the risk-to-benefit ratio weighs heavily in its favor in the resuscitation of selected trauma

victims. Klebanoff and coworkers reviewed the evidence as of 1970 and determined that "in over 1000 documented cases of autotransfusion in the Western literature, not a single death or major complication was attributed directly to the transfusion." [11] Symbas [32] reported autotransfusing more than 400 patients with traumatic hemothorax without any significant morbidity related to the procedure. Mattox and associates [17] reported autotransfusing 69 patients an average of 3.9 units each, with only 1 death (from air embolism) directly attributable to the procedure.

## CONCLUSION

Autotransfusion, a technique more than 100 years old, has become a subject of renewed interest in the emergency setting. The previously feared complications of hematologic or metabolic embarrassment and sepsis have not proved to be of clinical significance when appropriate patient selection and careful technique are followed. In addition, the use of

autologous blood has several advantages over the transfusion of stored homologous blood in the emergency patient, including ready availability of compatible blood, homeostasis of core temperature, higher levels of 2,3-DPG in RBCs, and cost-effectiveness. Autotransfusion has been endorsed by the Council on Scientific Affairs of the American Medical Association as a procedure that has been found to be "effective, safe and cost effective for many trauma and surgical patients" when used by experienced health care providers. [14] Although the technique is not totally free of complications, the benefits to be gained from autotransfusing the selected trauma patient outweigh the relatively limited risks.

# Chapter 29 - Transfusion Therapy: Blood and Blood Products

**Douglas A. Propp**

Transfusion of whole blood or its components (red cells, white cells, platelets, whole plasma, or plasma fractions) is indicated to compensate for certain deficiencies manifested by patients. The indications for the transfusion of *whole* blood have virtually vanished, as recent technical advances have made specific component replacement more feasible. Appropriate administration of blood products may be life saving in some emergency circumstances; it may also be indicated as a prophylactic measure in less urgent situations. By law, transfusions can be given only by a physician; potential life-threatening complications can result, as is true of any intravenous (IV) infusion.

## BACKGROUND

### Blood Groups

In the 17th century, radically daring physicians were experimenting with the transfusion of blood from animals into humans to treat a variety of ills. Around the beginning of this century it became obvious that only human blood was fit for humans. Landsteiner found that most human serum contained naturally occurring substances that reacted with the red blood cells of some, but not all, other humans, thereby discovering the ABO red cell antigen-antibody system.

### Red Cell Antigens and Antibodies

An integral part of the red blood cell (RBC) membrane is a series of glycoprotein moieties, or antigens, which give the cell an individual identity. Two different genetically determined antigens, type A and type B, occur on the surface of RBCs. The RBCs of any individual may have one, both, or neither of these antigens. Because the type A and type B antigens on the cell surface make the RBC susceptible to agglutination, these antigens are termed *agglutinogens*. The presence or absence of the agglutinogens makes up the ABO blood group classification. If neither the A nor the B agglutinogen is present, the blood group is O. When only type A agglutinogen is present, the blood is group A, and when only type B agglutinogen is present, the blood is group B. When both A and B agglutinogens are present, the blood is group AB. The relative frequencies of the different blood groups are listed in Table 29-1 (Table Not Available) .

Genes on adjacent chromosomes determine the presence or absence of agglutinogens and hence the individual's specific ABO blood group. These allelomorphic genes can be only one of the three different types--that is, A, B, or O--allowing for six possible combinations of genes (OO, OA, OB, AA, BB, and AB). There is no dominance among the three different allelomorphs; however, the type O is basically functionless, in that it causes such weak agglutination that it is normally insignificant. The different combinations of genes signify the individual's genotype, and each person is one of six

different genotypes. The resultant blood groups for the various genotypes are listed in Table 29-2 (Table Not Available) .

When type A agglutinogen is *absent* on a person's RBC, antibodies known as anti-A spontaneously develop in the plasma. Likewise, when type B agglutinogen is *absent*, anti-B antibodies develop in the plasma. When the blood is group O, both anti-A and anti-B antibodies develop. These antibodies are termed *agglutinins*. It follows, then, that group AB blood, which contains the agglutinogens A and B, contains no agglutinins at all in the plasma. Immediately after birth, the quantity of agglutinins in the plasma is near zero, but titers begin to develop in the first year of life and reach their maximum titer when the individual is between 8 and 10 years of age. This titer gradually declines throughout the remaining years of life.

The agglutinins are gamma-globulins of the IgM and IgG types and are probably produced by exposure to agglutinogens in food, bacteria, or exogenous substances other than blood transfusions. The antibodies (agglutinins) in the plasma of one blood type react with the antigens (agglutinogens) on the RBC of another blood type. This initiates the agglutination and hemolysis that are encountered in a transfusion reaction. Many other antigenic proteins (as many as 300 of different potency) are present in the RBCs of different persons. Some are of academic or legal importance, whereas others are important for their ability to produce transfusion reactions.

Clinically, the importance of antibodies directed against RBC antigens is determined by their frequency and whether they can cause RBC destruction in the circulation. The ABO system is the most important. With the first transfusion of ABO- *incompatible* blood, severe, potentially fatal reactions can occur. The Rh system is likewise very important, because there is a high likelihood (30 to 50%) that an Rh(D)-negative person will form antibodies after exposure to Rh-positive RBCs; these antibodies are then capable of causing severe hemolysis when RBCs containing the antigen are transfused a second time. Of the 40 antigens in the Rh system, D is the most antigenic, but others can also stimulate the production of antibodies in recipients lacking the antigen (e.g., E), thus complicating future transfusions. Other antigen systems in which antibodies could potentially cause hemolytic reactions are the Kell (K and k alleles), Duffy ($Fy_a$ and $Fy_b$ ), Kidd ($Jk_a$ and $Jk_b$ ), and MNS (M and N; closely linked S and s) systems. Other antigen systems are very rarely important in transfusion therapy.

## Crossmatching

Compatibility testing, or *crossmatching*, is the procedure by which the RBCs and serum of the donor unit of blood are mixed, respectively, with the serum and RBCs of the recipient to identify the presence of any antibodies and, hence, the potential for a transfusion reaction. These antibodies, after attaching to the appropriate RBC surface antigen, have the potential to cause agglutination and hemolysis of either donor or recipient RBCs. This hemolysis may be immediate or delayed. "Major" and "minor" crossmatch procedures are outlined in Table 29-3 . The end point of all crossmatches is the presence of RBC agglutination (either gross or microscopic) or hemolysis. Testing is performed immediately after mixing, after incubation at 37 °C for varying times, and with

and without an antiglobulin reagent to identify surface immunoglobulin or complement. Each unit of blood product, when properly crossmatched, can be administered with the expectation of safety.

## Transfusion Reactions

*Most serious transfusion reactions occur because of patient-blood product misassignment.* When incompatible blood is given, the result to the patient may range from no effect to death. If the recipient does *not* have antibodies (naturally occurring or acquired) directed against the foreign RBC antigen received, there will be no immediate reaction, but antibodies to the infused blood may develop within weeks, which will limit the safety of subsequent transfusions from the same donor or same antigenic type. If the recipient's serum *has* preformed antibodies directed against the donor RBCs (incompatibility in the *major* crossmatch), within seconds or minutes the recipient will begin to hemolyze the transfused (donor) cells.

In most cases of major crossmatch reactions, RBCs of the *donor* blood are agglutinated and hemolyzed. It is very rare that the transfused blood ever produces agglutination of the recipient's cells. Donor blood is affected because the plasma portion of the donor blood immediately becomes diluted by the plasma of the recipient, thereby diluting the titer of the infused agglutinins to a level too low to cause agglutination. Because the recipient's plasma is not diluted to any significant degree, the recipient's agglutinins can still agglutinate the donor cells. Mismatched blood groups eventually cause hemolysis of the RBCs. Occasionally, antibodies cause immediate hemolysis. More often, the cells first agglutinate, then are trapped in peripheral vessels; over a period of hours to days they become phagocytized, releasing hemoglobin into the circulatory system.

Clinical manifestations of acute hemolysis are chills, fever, tachycardia, abdominal pain, back pain, hypotension, fainting, and an anxious "feeling of impending doom." From the liberation of intracellular material associated with hemolysis, vasoactive substances may aggravate a preexisting hypotension and cause shock; other substances may precipitate disseminated intravascular coagulation, and high-output cardiac failure, or anoxic acute renal failure may result. Hemolytic transfusion reactions are estimated to occur once per every 6000 blood units transfused, with a fatality rate of 1 per every 100,000 units transfused.

An incompatibility in the *minor* crossmatch usually causes no serious reaction, although the recipient's (patient's) red cells could be hemolyzed if the titer of the antibody were sufficiently large. Even when major and minor crossmatch compatibility indicates the safety of a transfusion, a delayed hemolytic transfusion reaction can occur days to weeks later. Usually seen in multiply transfused patients (or in multigravida women), these reactions may be unavoidable without complete RBC antigen typing, a procedure occasionally indicated for recipients of numerous repeated transfusions. Fortunately, 90% of transfusions are now given as packed RBCs

| | **Major Crossmatch** | **Minor Crossmatch** |
|---|---|---|
| | TABLE 29-3 -- Crossmatch Procedures | |
| Donor | Red cells | Serum |
| Recipient | Serum | Red cells |
| End point | Agglutination or hemolysis at 37° C | |

that contain a small volume of plasma, minimizing the chance for a transfusion reaction due to donor sensitization.

Additional antibodies *not* caused by sensitization from transfused RBCs include autoantibodies (both cold- and warm-reacting) and various agglutinins. Autoantibodies can be "cold," reacting with red cells *more strongly at 4 °C* than at 37 °C. These antibodies are common and are usually harmless; however, they may be associated with disease states in higher titers (e.g., anti-I in *Mycoplasma* infections). If active at higher temperatures (up to 28 to 32 °C), pathologic cold antibodies may cause hemolysis or may even lead to enough RBC agglutination to cause obstruction of blood flow through the small vessels of the hands and feet on exposure to cold. These would also be present and identifiable at 37 °C. The primary significance of cold antibodies stems from their ability to complicate crossmatching procedures in the blood bank.

Warm antibodies, reacting *more strongly at 37 °C* than at lower temperatures, can be harmless or can be responsible for a hemolytic anemia of variable severity. Characteristics of the IgG antibody itself determine its significance to the patient. Usually harmless warm autoantibodies that can occasionally cause hemolysis are seen in patients taking alpha-methyldopa. Harmful warm autoantibodies are encountered in approximately 80% of patients with autoimmune hemolytic anemia.

Additional problems in pretransfusion testing may occur with antibodies directed against various substances that can attach themselves to the RBC surfaces and can cause

agglutination of the "innocent bystanders." Examples are the fatty acid-dependent agglutinins; penicillin and cephalosporin antibodies; bacterial polysaccharides; and nonspecific agglutination associated with a high erythrocyte sedimentation rate, caused by high levels of the acute-phase reactants fibrinogen, alpha2 -macroglobulin, and IgM. A delay in pretransfusion testing may occur when the blood bank has to undertake procedures to identify various proteins on RBC surfaces to ascertain their clinical significance.

## Miscellaneous Transfusion Problems

Pyogenic transfusion reactions, such as fever and chills, are rather common and result from the presence in the donor plasma of proteins to which the recipient is allergic. Full-blown anaphylactic reactions occasionally result.

Theoretically, citrate salts, which are the usual anticoagulants in donor blood, may combine with ionized calcium in the plasma, producing hypocalcemia. In clinical practice, the hemodynamic consequences of citrate-induced hypocalcemia are minimal, although the Q-T interval may be prolonged on the electrocardiogram (ECG) with citrate infusion. Supplemental calcium administration is usually not necessary even during massive blood replacement as long as circulating volume is maintained, because the liver is able to remove citrate from the blood within a few minutes. Alterations in this recommendation may be necessary in the presence of severe liver disease.

## Infectious Complications of Transfusions

Sepsis is of theoretical concern in blood component therapy because 0.1% of all transfusions may transmit virulent bacteria. However, sepsis is an uncommon occurrence because both citrate preservative and refrigeration kill most organisms. Concern over sepsis has governed the decision to complete transfusions within 4 hours and to return unused blood products to the blood bank refrigerator for future use only if they have been out of the refrigerator for <30 minutes. The virulent pathogens involved are most commonly gram-negative organisms.

Most blood products retain the ability to transmit hepatitis. Ninety percent of the time, the etiologic agent is hepatitis C. The customary incubation period for this agent following infusion is 2 to 12 weeks, although 80% of patients are anicteric. The likelihood of developing post-transfusion hepatitis ranges from 1 per 100 to 1 per 3000 units of blood administered. Up to 20% of patients developing chronic hepatitis ultimately develop liver failure. Since 1991, blood donors have been tested for hepatitis C, although the test is relatively inaccurate and has a positive predictive value of only 40%. In 1992, the FDA approved the use of interferon alpha2b for the treatment of selected cases of chronic hepatitis C infection.

Although syphilis may theoretically be transmitted by transfusion, *Treponema pallidum* does not survive when refrigerated or when placed in a citrated medium. Only fresh blood or platelet transfusions are of concern. The incubation period is 4 weeks to 4 months, and the initial clinical manifestation is a rash. Although controversial because of high rates of false-positive and false-negative results, many blood banks still test for

positive *T. pallidum* serology.

Both cytomegalovirus and Epstein-Barr virus can be transmitted through transfusions. A mononucleosis-like syndrome occurring 2 to 6 weeks after a transfusion is suggestive of the presence of either of these agents. Blood products with negative serologic findings for both of these agents should be used for seronegative recipients in high-risk groups, such as pregnant females, premature or low-birth-weight newborns, marrow or organ transplant recipients, and immunosuppressed patients.

The likelihood of transmission of malaria through blood products is minimal because of routine preventive measures. Prospective blood product donors who have been to an endemic region within 6 months or treated with malarial prophylaxis within 3 years are not allowed to donate blood products.

The acquired immunodeficiency syndrome (AIDS) epidemic has affected transfusion therapy. In the United States, 3% of AIDS cases have been linked to blood products. In addition, 65% of hemophiliacs in the United States have been exposed to the virus. Voluntary deferment by high-risk groups was encouraged in 1983, and formal screening of all blood products began in 1985. Given that any units that test positive for the human immunodeficiency virus (HIV) antibody are routinely discarded, concern over transmission of the virus centers around the 6- to 8-week "window," during which a donor might be antigen-positive (HIV-infected) but temporarily antibody-negative. It has been estimated that the likelihood of transmitting HIV through blood products is 1:493,000 per unit of blood donated. [1] If a blood recipient's antibody level remains negligible 6 months after transfusion, it is extremely unlikely that the disease will develop. Zidovudine has not been shown to protect patients receiving blood infected with the AIDS virus.

**Directed Donations**

A system of " *directed donations*," a system by which friends or family members may give blood to be transfused to a given individual, has been proposed to answer the concern over the transmission of HIV. Some believe that the blood products derived from a relative or a friend have a lower likelihood of testing positive for an HIV infection. At this time, directed donation systems are in place in some institutions, but the practice has not been widely supported. It is feared that blood products will be less safe because social pressures may result in no self-deferment of high-risk donors and more clerical errors will be made because of the increased complexity of this system. Finally, there is concern that the directed donation plan will disrupt the normal anonymous blood donor system, leaving fewer units available for other needy patients.

**Crystalloid vs Colloid Solutions**

In the case of a hypotensive patient suffering from acute hemorrhage, continued debate surfaces regarding whether it is most appropriate to administer crystalloid or colloid solutions before the institution of blood component therapy. [2] Crystalloid solutions are promoted as being ideal to replace the interstitial fluid deficit that occurs during bleeding. Unfortunately, these balanced salt solutions freely cross capillary membranes,

thus providing less oncotic pressure and less prolonged plasma volume expansion than is desired. Thus, only 10 to 20% of the administered crystalloid solution remains intravascular, perhaps for only 2 hours.

Colloid solutions contain large, oncotically active molecules that remain within the capillary membrane. They are efficient blood volume expanders because fluid deficits can be replaced faster with two to four times less volume than that needed with crystalloid solutions. [3] Unfortunately, colloid solutions cost 20 to 30 times more than a comparable amount of crystalloid solutions.

Three colloid preparations are commercially available. Albumin is manufactured from donor plasma as either a 5% or 25% solution dissolved in normal saline. Volume expansion typically lasts 24 hours. There is a 0.5% incidence of urticaria, fever, or chills when using this fluid. Hetastarch is a synthetic amylopectin available as a 6% solution dissolved in normal saline. It has an osmolality of 310 mOsm and a pH of 5.5. Volume expansion lasts 24 to 36 hours. Minor adverse effects on clotting have been documented. The rate of adverse reactions is the same as that seen with albumin. Dextrans are high-molecular-weight polysaccharides, which have been known to interfere with typing and crossmatching as well as with platelet function.

There are data to support either viewpoint in the crystalloid versus colloid debate. Unfortunately, the information is confusing and frequently conflicting. Although fluid resuscitation can be adequately accomplished with the use of crystalloids, when severe blood loss is replaced with crystalloids alone, marked overexpansion of the extracellular fluid volume occurs. Although colloids typically offer no obvious clinical advantage and it would be imprudent to use them routinely in hemorrhagic fluid resuscitations, they are more efficient plasma volume expanders.

Human studies are now being reported in which the prehospital use of hypertonic saline (4 mL/kg of 7.5% sodium chloride) have proven beneficial in restoring trauma victims' blood pressure and survival compared with conventional isotonic solutions. The addition of dextran to the hypertonic saline seems to add nothing to the positive outcomes.

## USE OF BLOOD PRODUCTS

Blood products are divided into components and fractions. Blood components, such as fresh frozen plasma (FFP), packed RBCs, granulocytes, cryoprecipitate, and platelets, are prepared from a single donor and are separated by physical means and transfused as single units.

The availability of autotransfusion devices in most emergency departments provides an opportunity to preserve blood bank resources that might otherwise be needed. The autotransfused blood harvested from the patient's intrathoracic cavity is fresh, warm, compatible, inexpensive, immediately available, and unlikely to cause a transfusion reaction. Once the blood is collected, it is passed through a filter that removes extraneous debris, and then it is reinfused through a standard IV line. Although controversial, most authorities recommend administering citrate-phosphate-dextrose (CPD) with the whole blood. Potential complications include air embolization,

coagulopathy if large volumes of blood are administered, and sepsis if contaminated blood is disseminated.

Although of limited clinical applicability to emergency transfusions, autologous donations are commonplace. It has been suggested that up to 10% of the blood supply could be provided through this mechanism. Most appropriate applications at this time include elective cardiac, gynecologic, orthopedic, and vascular surgical cases. Benefits of this system include avoidance of exogenous bloodborne disease and sensitization. The individual can donate 1 unit of blood weekly, until 3 days before the surgical need. As blood can be stored up to 35 days, the donations usually begin 5 weeks before they will be needed. The blood donor will require iron supplements and must maintain a hemoglobin >11 g/dL. Table 29-4 lists some characteristics of blood and its components.

**Whole Blood**

Individuals normally have 70 to 80 mL/kg of whole blood. Whole blood provides a source of red cells for oxygenation, proteins for coagulation factors and oncotic pressure, and volume for rapid restoration of hypovolemia. Whole blood is indicated only for massive transfusion or exchange transfusion and usually is appropriately used only in treatment of patients with a decreased RBC oxygen-delivering ability and hypovolemic shock, such as after multiple trauma. Whole blood is *not* the indicated treatment for hypovolemic shock that can be treated effectively with crystalloid (e.g., lactated Ringer's solution, 0.9% sodium chloride) or colloid (e.g., plasma protein, albumin) solutions; it is *not* indicated for correction of thrombocytopenia, replacement of coagulation factors, or treatment of anemia that can be treated with replacement iron, vitamin $B_{12}$ , or folic acid. Most blood banks currently do *not* stock significant quantities of whole blood; instead they substitute packed erythrocytes for indications previously assigned to whole blood.

| TABLE 29-4 -- Characteristics of Blood and Its Components | | | |
| --- | --- | --- | --- |
| **Component** | **Volume** | **Shelf Life** | **Requirements for Transfusion** |
| Whole blood * | 450 mL blood | 21 days at 4° C | Crossmatched |

| | | | |
|---|---|---|---|
| ACD | 63 mL anticoagulant and preservative | | |
| CPD | | | |
| CPD-A | 35-40% hematocrit | 35 days at 4° C | |
| Packed red cells concentrate, washed | 280 mL 70% hematocrit | Same as for whole blood | Crossmatched |
| | 250 mL 70% hematocrit | 1 day at 4° C | Crossmatched |
| Frozen-thawed red cells | 250 mL 70% hematocrit | ? years when frozen, 1 day after thawing | Crossmatched |
| Platelet concentrate | 30 mL $10^{10}$ platelets | 5 days at 22° C | Type-specific if possible, but not essential, not crossmatched |
| Fresh frozen plasma | 200-250 mL | 1 year at - 18° C, 24 hr after thawing | ABO-compatible; random donor, not crossmatched |
| Cryoprecipitate | 10-25 mL per bag 60-120 units of factor VIII | 1 year at - 18° C, 6 hr after thawing | ABO-compatible; random donor, not crossmatched |

| Factor IX or prothrombin concentrate | 25 mL per vial | Check label | None required |
|---|---|---|---|
| Granulocyte concentrate | 400 mL 10$^{10}$ leukocytes | Transfuse within 24 hr at 22° C | Specific donors for each patient, crossmatched |

* ACD, acid-citrate-dextrose; CPD, citrate-phosphate-dextrose; CPD-A, citrate-phosphate-dextrose-adenine.
Special order--few hospitals have facility inhouse.
Use immediately to correct deficiency of coagulation factors.

Whole blood is collected from donors into plastic bags containing 63 mL of citrate-phosphate-dextrose (CPD) anticoagulant and preservative in a total volume of 515 ± 50 mL and resultant hematocrit of 35 to 40%. If >24 hours old, whole blood is essentially devoid of normally functioning platelets and other clotting factors, especially the labile clotting factors V and VIII. In addition, whole blood contains antigenic leukocytes and serum proteins, which may produce allergic reactions (a risk of 1%).

Because blood products are stored for up to 35 days, various "storage lesions" occur. Some blood banks now use ADSOL (adenine, dextrose, saline, mannitol, and water) as a preservative, which increases storage life up to 49 days. Cell metabolism continues to occur while blood is being stored, so a small degree of acidosis commonly develops. However, this acidosis is buffered effectively by the bicarbonate derived from the metabolism of the citrate used as a preservative, assuming normal hepatic function. Even in massive transfusions, acidosis is usually due to the disruption of normal physiologic function rather than to the blood products themselves. During storage, levels of 2,3-diphosphoglyceric acid (2,3-DPG) also decrease. This decrease results in the shift of the oxygen-hemoglobin dissociation curve to the left. The shift is of small clinical significance, however, because the level soon rises and is usually normal in the recipient within 24 hours of infusion. In addition, potassium commonly leaks out of the cells during storage because of a less efficient sodium-potassium adenosine triphosphatase (ATPase)-dependent pump. Fortunately, most of the potassium is either absorbed by the remaining blood cells, excreted by the kidney, or shifted back into the cells owing to the alkalosis produced by metabolism of the citrate in the preservative. Hyperkalemia is clinically relevant only in newborns and patients with renal impairment.

The incidence of transfusion reactions following transfusion with whole blood is approximately 2.5 times greater than the incidence of reactions following transfusion with packed RBCs. [5] Although it is certainly true that patients bleed whole blood, not packed cells, it is often recommended that even acute blood loss be treated with packed cells as opposed to whole blood. [6] One unit of whole blood raises the hematocrit approximately 3%. The plasma of whole blood is no more effective than 5% albumin as

a volume expander.

**Packed Red Blood Cells**

Packed RBCs provide oxygen-carrying capacity and volume expansion. Packed RBCs are prepared by centrifugation and removal of most of the plasma from citrated whole blood. Packed RBCs that have been grouped and have been assayed for Rh factor should be the most common blood component used for treating anemia not amenable to nutritional correction. Hazards of metabolic derangements, donor antibodies, volume overload, and (possibly) hepatitis are lessened with packed RBCs as compared with whole blood. Patients with severe or chronic anemia or heart disease or those who otherwise require fluid restriction can receive packed RBCs more safely than whole blood. Furthermore, to prevent circulatory overload in susceptible patients, a rapid-acting diuretic, such as furosemide or ethacrynic acid, can be administered IV at the beginning of the transfusion. Criteria for transfusion of packed RBCs vary. A hemoglobin level <10 g/dL is one criterion commonly suggested for transfusion

prior to surgery. The National Institutes of Health suggest that prophylactic transfusion should not be done on anemic patients with a hemoglobin >7 g/dL (hematocrit 21%). Healthy individuals sustaining acute blood loss may have no significant physiologic impairment with hemoglobin levels as low as 6 to 8 g/dL (hematocrit, 18 to 24%). The patient with circulatory shock and a hemoglobin concentration of 8 g/dL or greater may not benefit from a transfusion if volume-resuscitation has already been undertaken. [7]

One unit of packed RBCs contains the same red cell mass as 1 unit of whole blood at approximately half the volume and twice the hematocrit (70 to 80%). One unit of packed RBCs raises the hematocrit approximately 3% in an adult or increases the hemoglobin level of a 70-kg individual by 1 g/dL. In children, there is an approximate rise in hematocrit of 1% for each mL/kg of packed cells. For example, if 5 mL/kg of packed RBCs is transfused, the hematocrit will rise by approximately 5%. Actual changes are dependent on the state of hydration and the rate of bleeding.

When washed to remove leukocytes, platelets, microaggregates, and plasma proteins, packed RBC transfusions cause fewer transfusion reactions than do whole blood transfusions. RBCs are not routinely washed before transfusion, but washing reduces the titer of anti-A and anti-B, permitting safer transfusion of type O packed RBCs in non-O recipients. Washing does not totally eliminate the risk of hepatitis. Washed RBCs are prepared in the blood bank by centrifugation, filtration, or use of sedimenting agents or by washing the unit of whole blood or packed RBCs.

Frozen deglycerolized RBCs likewise are free of platelets, plasma, and white blood cells, having been washed after an indefinite period of frozen storage in glycerol. Frozen RBCs and fresh RBCs function similarly; frozen RBCs provide normal levels of 2,3-DPG. Washed or frozen preparations should be given to patients who have had febrile (nonhemolytic) reactions to previous transfusions as a result of leukocyte antibodies or IgA sensitization. Blood bank procedures require that these be prepared to order, with routine crossmatching. Considerable delay (6 hours) may occur if the

transfusion service does not have the capability of washing RBCs.

Packed RBCs contain less sodium, potassium, ammonia, citrate, and antigenic protein and fewer hydrogen ions than does whole blood. This may offer an advantage in patients with reduced cardiovascular, renal, or hepatic function. The rate of urticaria is still relatively high at 1 to 3% of transfusions, but the incidence of adverse reactions to packed cells is approximately one third that noted with whole blood.

Many physicians use packed RBCs during surgery and for replacement treatment of acute blood loss of any cause. As is true of whole blood, packed RBCs can be stored up to 21 days by law, although newer preservatives may allow 49-day storage. Red cell viability decreases approximately 1% per day.

**Fresh Frozen Plasma**

Fresh frozen plasma should be given to patients with a hereditary or acquired deficiency of coagulation factors, provided that a preparation of the specific deficient factor is not available. Each unit of FFP has a volume of approximately 200 to 250 mL and is prepared by freezing the plasma separated from RBCs and platelets of single-donor whole blood, and stored at 18 °C or below within 8 hours of collection. Plasma should be compatible in terms of the recipient's ABO group. Rh compatibility is not considered essential. [8]

Fresh frozen plasma contains all soluble coagulation factors of the intrinsic and extrinsic clotting systems, including the labile factors V and VIII. Fresh frozen plasma also contains fibrinogen, although not as much as does cryoprecipitate. Fresh frozen plasma has a shelf life of up to 1 year, and plasma stored for 3 months retains approximately 60% of the normal factor VIII activity. Fresh frozen plasma contains no platelets.

Fresh frozen plasma is indicated for the clotting factor deficiencies resulting from the diluting effect of massive blood replacement. However, pathologic hemorrhage following massive transfusions is often caused by thrombocytopenia rather than by a depletion of clotting factors. One unit of FFP per 5 to 6 units of packed cells or whole blood is a reasonable replacement formula if specific clotting tests are not rapidly available, but plasma replacement is best dictated by evaluation of prothrombin time (PT) and partial thromboplastin time (PTT). Fresh frozen plasma is indicated for rapid reversal of serious acute bleeding from warfarin (Coumadin) anticoagulants or for prophylaxis prior to surgery or an invasive procedure. In an emergency situation, 5 to 10 mL/kg of FFP will effect a rapid reversal of the vitamin K-dependent factors II, VII, IX, and X. As a rough guide, 1 unit of FFP increases all coagulation factor levels by 2 to 3% in the average-sized adult. In *life-threatening* hemorrhage from warfarin excess, factor IX concentrate (Konyne 80, Proplex, Mononine) may be used, but such therapy should not be routine because of the high incidence of hepatitis and the possibility of thrombosis with these products.

Fresh frozen plasma may be valuable in patients with other clotting abnormalities, such as a congenital deficiency of factor II, V, VII, X, XI, or XIII, von Willebrand syndrome, hemophilia A (factor VIII deficiency) and hemophilia B (factor IX deficiency), or

hypofibrinogenemia; however, the effectiveness is limited in severe clotting abnormalities because of the large volume that is generally required. For example, FFP may be successful in the treatment of hemarthrosis or other minor bleeding tendencies in hemophilia, but specific factor replacement is preferred. Fresh frozen plasma is also used to treat the acquired deficiency of multiple factors such as that seen in severe liver disease, disseminated intravascular coagulation, or vitamin K depletion, and for plasma exchange in thrombotic thrombocytopenic purpura or hemolytic uremic syndrome. Fresh frozen plasma should not be used for volume expansion or to enhance wound healing.

Reactions to FFP include fever, chills, allergic responses, HIV infection, and a risk of hepatitis that is similar to the risk with whole blood. Fresh frozen plasma should be infused rapidly and given immediately after thawing because of the rapid loss of labile clotting factors.

The usual starting dose is 2 bags of FFP if the PT is >1.5 times normal or the activated PTT (aPTT) is >1.5 times the top normal value. If the PT is <22 seconds or the aPTT is in the 55 to 70 second range, one bag of FFP may be sufficient to bring the deficit into the hemostasis range (Table 29-5) . Each 5 to 6 units of platelets contain the equivalent of unit of FFP, so concomitant platelet infusions may lower FFP requirements.

| TABLE 29-5 -- Transfusion of Blood Products | | | |
|---|---|---|---|
| **Blood Product** | **Waiting Time to Receive in Emergency Department** | **Initial Amount to Transfuse** | **Expected Response in 70-kg Adult** |

| | | | |
|---|---|---|---|
| Un-crossmatched Rh+ or Rh- RBCs | 5 min | 2-10 units, 10 to 20 mL/kg/ hr or as needed based on clinical condition | Stabilize patient in shock |
| Un-crossmatched type-specific whole blood | 15 min | | Change in hemoglobin/hematocrit depends on hydration and rate of bleeding |
| Typed and screened whole blood | 25 min | | Approximate rise of 1 g dL hemoglobin per unit |
| Crossmatched whole blood | 1¼ hr | | |
| Packed RBCs | 1½ hr | | Each unit raises hematocrit 2-3% |
| Frozen RBCS | 4-6 hr (if not prepared inhouse) | | In children, each mL/kg of packed cells raises hematocrit by 1% |
| Platelet concentrate | 5 min if available | 1 unit per 10 kg, usually 6 to 10 units per transfusion in an adult | Rise of 5000 to 10,000 platelets per mm3 per unit; 6 units usually sufficient to stop bleeding |
| Cryoprecipitate | 20 min | 1-2 bags per 10 kg (7-15 bags) 10-min push, or 20-50 units/kg | Rise of 3% in factor VIII level per bag (40-100% activity desired) |

| Factor IX or prothrombin concentrate | Immediately available (reconstituted powder) | 10-50 units/kg | 30-100% rise in factor IX activity |
|---|---|---|---|
| Fresh frozen plasma | 40 min | 1 bag per 7 kg (4-10 bags for adult) 10-min push, *3-10 mL/kg, depending on clinical condition | Correction in coagulation status; 1 unit raises all coagulation factors by 2-3% in average-sized adult |

Also consider thrombocytopenia as a cause of bleeding from massive transfusion.
* Administer 1 bag per 4 to 6 units of blood transfused to replace diluted and inactivated coagulation factors.

**Cryoprecipitate**

Cryoprecipitate is used specifically to correct a deficiency of coagulation factor VIII (in hemophilia A and in von Willebrand syndrome), factor XIII, or fibrinogen. The precipitate is prepared from single-donor plasma by gradual thawing of rapidly frozen plasma, which yields an undissolved protein that is collected and stored at very low temperatures. Cryoprecipitate is a plasma product and as such requires ABO and Rh compatibility, but crossmatching is not necessary.

Cryoprecipitate contains approximately 30 to 50% of the original plasma content of factors VIII and XIII and fibrinogen. Each 15- to 25-mL bag of cryoprecipitate contains 60 to 120 units of factor VIII, 125 to 250 mg of fibrinogen, and an unknown amount of von Willebrand factor. Cryoprecipitate is of no value in the treatment of factor IX deficiency (hemophilia B).

Once spontaneous bleeding has occurred in hemophilia or von Willebrand syndrome, it will usually *not stop until the deficient factor is replaced*. It is best to treat early to prevent minor bleeding from developing into a significant hemorrhage. The goal of therapy is to achieve at least 50% of normal factor VIII activity. In spontaneous intracranial hemorrhage, one should seek 100% activity. The amount of cryoprecipitate required to correct coagulation defects ranges from 10 to 20 units/kg for minor bleeding, such as hemarthrosis, to 50 units/kg for bleeding control in surgery or trauma, but specific replacement should be guided by laboratory assay of factor VIII activity. One bag of cryoprecipitate per 5 kg of body weight will raise the recipient's factor VIII level to approximately 50% of normal. The half-life of factor VIII in plasma is 8 to 12 hours. Mild deficiencies of factor VIII are considered to exist at 10 to 30% of normal, and severe deficiencies exist at <3% of normal activity. Many patients know their level of factor VIII,

and such levels remain relatively constant.

Rarely, cryoprecipitate may be required to correct significant hypofibrinogenemia (<100 mg/dL). Fresh frozen plasma may also be used to treat mild degrees of hypofibrinogenemia.

**Factor VIII Concentrate**

Factor VIII concentrate is a product for the treatment of classic hemophilia A. It is derived from a large donor pool and can be stored for up to 2 years in a home refrigerator. The product is significantly more concentrated than cryoprecipitate or FFP. The known factor activity is listed on every bottle. Administration of 1 unit per kg of body weight should increase the factor VIII activity by 2%. Minor episodes of bleeding are usually treated with 10 to 20 units/kg, whereas major episodes are treated with 20 to 30 units/kg, with a

repeat dose 12 hours later. Antibodies develop in up to 15% of factor VIII recipients. Various techniques have been used to overcome this problem. Administration of massive doses of factor VIII has been shown to be somewhat beneficial in overwhelming the endogenous antibody response. In addition, immunoadsorbent techniques to remove the antibody have met with some guarded success. The general use of immunosuppressives and plasmapheresis has had limited success. Activated prothrombin complex has been effective, but concern over the cost of preparation, the significant hepatitis risk, and the thrombogenicity associated with its use limits its application.

Because factor VIII concentrate is derived from large donor pools, the likelihood of transmission of hepatitis and HIV infection is higher. Different forms of chemical treatments have been attempted to minimize this threat. These techniques include heat treatment, pasteurization, use of organic solvents and detergents, and immunoaffinity chromatography. Since the gene for factor VIII production was discovered in 1984, research into recombinant genetics has been aimed at providing a safer product that theoretically will be more readily available and less expensive to produce. Two recombinant-DNA derived factor VIII preparations (Recombinate, Kogenate) were approved by the FDA in 1993. Although these genetically engineered products have hemostatic activity equivalent to plasma-derived factor VIII and are thought to be free of viral contaminants, they remain much more costly than other products. [9]

A synthetic analogue of pituitary vasopressin, 1-deamino-(8-D-arginine)-vasopressin (DDAVP), has been found to stimulate the endogenous production of factor VIII in a subset of mild hemophiliacs. The exact mechanism is unknown, but treatment with 0.3 mg/kg IV over 15 minutes has been recommended when avoidance of the inherent risks of the factor VIII concentrate is desired.

**Factor IX Concentrate (Prothrombin Complex)**

Prothrombin complex concentrate, or factor IX concentrate (Konyne or Proplex), is

prepared from pooled human plasma and is available as a lyophilized powder. Factor IX concentrate contains the liver-synthesized, vitamin K-dependent factors: II (prothrombin), VII, IX, and X. The actual factor IX activity of each vial is stated on the label. Each vial is reconstituted to a volume of 25 mL and contains approximately 500 units of factor IX, 300 units of factors VII and X, and 200 units of factor II. The use of this product carries a very high risk of hepatitis transmission (almost 100%); because of this, it is rarely used. The risk of HIV infection is analogous to that seen with the use of factor VIII concentrate. Post-treatment hyperthrombosis may also occur.

Factor IX concentrate is used almost exclusively in the treatment of hemophilia B, because cryoprecipitate is effective only for the treatment of hemophilia A (factor VIII deficiency). Patients with hemophilia should always be asked which deficiency they manifest, because the treatment of each type is different. Although factor VIII deficiency is much more common, the routine treatment of all patients with "hemophilia" with cryoprecipitate is not warranted. Factor IX concentrate may theoretically be used instead of FFP in the rare instance in which volume must be kept at a minimum. The use of prothrombin complex is also warranted when there is a possibility of *life-threatening* hemorrhage, such as intracranial bleeding, in patients with *warfarin-induced* hemorrhage. Vitamin K and FFP, however, are definitely preferred in the noncritically ill patient with warfarin-induced bleeding. A new monoclonal antibody purified factor IX preparation, safe from the risks of hepatitis and thrombogenicity, is now available.

In hemophilia B, the aim of therapy is to achieve 20 to 30% of normal values of factor IX. Higher levels are desired for the treatment of intracranial bleeding. Most patients know the level of their deficiency, and the deficiency remains relatively constant. Treatment of factor IX-deficient patients with 15 to 30 units of factor IX concentrate per kilogram of body weight, once or twice a day, usually results in normal hemostasis, but individual responses to therapy may vary. Minor bleeding (soft tissue, joints) may be controlled with 10 to 20 units/kg of body weight. It is a common error to assume that a minor spontaneous bleeding episode will be self-limited in patients with either form of hemophilia. Once spontaneous bleeding occurs, however, it rarely stops spontaneously, and treatment with replacement factors is necessary.

**Platelet Concentrates**

Platelet concentrates are prepared by rapid centrifugation of platelet-rich plasma, which is obtained by slow centrifugation of freshly collected whole blood to separate the RBCs. Platelets are obtained by single-donor apheresis or from random donor whole blood units. HLA-matched platelets may be used when patients develop HLA antibodies from repeated random donor platelet transfusions. Platelet concentrates contain most of the platelets from 1 unit of blood in 30 to 50 mL of plasma; they are given to raise a patient's platelet count and to correct bleeding from thrombocytopenia. One unit (pack) of platelets per 7 kg of body weight will raise the platelet count by 50,000/mm3 in the absence of antibodies; therefore, 1 unit of platelet concentrate raises the platelet count by 5000 to 10,000/mm3 . The usual adult dose given is 6 to 10 units of platelet concentrate, depending on the clinical condition. Assuming a zero platelet level, 6 units given to a normal-sized adult should increase the platelet count to >50,000 per mm3 . If

there is no evidence of platelet consumption, this transfusion should be adequate for 3 to 5 days. In cases of severe platelet consumption the transfusion may be required every 6 to 24 hours. Some hospital blood banks prepare platelet concentrates regularly; in some cities a central blood bank service, such as the American Red Cross, prepares platelet concentrates regularly and delivers units on an "as-needed" basis within 1 to 2 hours of the request. Platelet concentrates are viable for 5 days when kept at *room temperature* and gently agitated at intermittent periods or when kept in motion. They should not be refrigerated.

This issue of prophylactic platelet transfusion remains controversial. Spontaneous bleeding rarely occurs if the platelet count is above 10,000 to 20,000/mm3 . Even in the event of surgery or trauma, excessive bleeding is uncommon in patients whose platelet count exceeds 50,000/mm3 . It is generally recommended that active hemorrhage be treated with platelet transfusion if the platelet count is below 50,000/mm3 , but prophylactic transfusion may be safely withheld until the platelet count is <20,000/mm3 . Patients with idiopathic thrombocytopenic purpura (ITP) should not

receive platelets prophylactically, but they may be transfused if life-threatening bleeding occurs.

Crossmatching is unnecessary for platelet transfusion, but the donor and the recipient should be ABO- and Rh-compatible. Note that platelet concentrates contain enough RBCs to sensitize an Rh-negative individual. There may be a diluting effect to the platelet count that results in thrombocytopenia with massive blood transfusions. When >8 to 10 units of blood is transfused, the platelet count must be routinely evaluated, and platelets must be replaced accordingly. Clinically significant platelet depletion rarely occurs if <15 units of blood (or 1.5 to 2 times blood volume) have been transfused.

Each 5 to 6 units of platelets will contain 250 to 350 mL of plasma (about 1 bag of FFP), which includes coagulation factors that may reduce the requirements of FFP. Platelets may be infused rapidly (1 unit/10 minutes), using specialized platelet filters.

**Granulocyte Transfusions**

Granulocyte transfusions are given in those unique instances in which a severely neutropenic patient has a suspected or proven bacterial infection that does not respond to appropriate treatment. They are rarely given in an emergency department. White blood cell transfusions require prior arrangements with a large blood bank service that has the capabilities of collecting granulocytes from a suitable donor; the collection procedure takes 4 to 6 hours on a continuous-flow cell separator. Transfusions need to be repeated frequently (every 12 hours) to provide a sufficient number of white blood cells to help the patient.

**Blood Products for Jehovah's Witnesses**

There are more than 1.5 million Jehovah's Witnesses in the United States. Based on the religious belief that the Bible prohibits blood or blood product transfusion (Acts

15:28-29), members of this religion do not accept transfusions of whole blood, packed cells, white blood cells, platelets, or plasma or autotransfusion of predeposited blood. Some may permit infusion of albumin, clotting factor solutions, or dextran or other plasma expanders and intraoperative autotransfusion. [10] Although no guidelines for administration of blood products to Jehovah's Witnesses are absolute, certain recommendations can be made. Even though a transfusion may be necessary to save a patient's life and would otherwise be considered standard care, the administration of blood and/or blood products in the face of refusal after informed consent can be legally considered as battery or a violation of a patient's right to control what is done to his or her body. In the awake and otherwise competent adult, courts have ruled that physicians cannot be held liable if they comply with a patient's directive and withhold life-saving blood administration following specific and detailed informed consent of the consequences of such an omission of treatment. The issue becomes clouded when patients are incompetent, unconscious (most Jehovah's witnesses carry cards informing medical personnel of their religious beliefs), or minors.

In the absence of specific directives to the contrary it is prudent to administer blood products to patients who are unconscious or are judged to be incompetent adults or are minors. Although case law often upholds the patient's wishes, actual damages against physicians are difficult to document in the United States. When done under documented life-threatening circumstances, significant physician liability would be extremely unusual. Pregnant females and significant providers for dependents have been deemed appropriate recipients of blood products against their wishes. Explicit documentation of the intent of the physician to preserve life coupled with an accurate description of the discussion of the issue with the patient or the family and a clarification of the patient's mental capacity is mandatory. Furthermore, emergency legal assistance (such as court orders, appointment of a temporary guardian) should be sought immediately with rapid judicial resolution. Various clinical techniques to maximize oxygen delivery and minimize oxygen consumption should be used. Examples include limited blood draws, the use of erythropoietin and nutritional support, hypothermia, volume expansion, sedation, and oxygen.

## ADMINISTRATION OF BLOOD COMPONENTS

When it has been decided that a patient needs a transfusion and the patient's condition is stable enough, the physician should question the patient or the patient's relatives concerning any previous transfusion reactions and whether the patient abides by any religious prohibitions to transfusions. A tube of blood (approximately 2 mL for every unit of blood product to be crossmatched) should be drawn from the patient and put into a red-topped, non-anticoagulated tube. The tube must *not* contain a serum separator gel. The label should be signed by the physician. This identifying signature will be used in the blood bank's crossmatching procedures.

### Emergency Transfusion

In an emergency or life-threatening situation, three alternatives to fully crossmatched blood exist. The preferred substitute is type-specific blood with an abbreviated crossmatch. The abbreviated crossmatch includes ABO and Rh compatibility. In

addition, the recipient's serum is screened for unexpected antibodies, and an immediate "spin" crossmatch is performed at room temperature. This abbreviated crossmatch requires approximately 30 minutes, and many institutions are now using this procedure as their standard crossmatch for most patients. The safety and utility of the type-specific abbreviated crossmatch have been demonstrated repeatedly, and transfusion reactions should occur only rarely. [11]

The second preference for an alternative to fully crossmatched blood is type-specific blood that is only ABO- and Rh-compatible, without screen or immediate spin crossmatch. The patient's ABO group and Rh factor can be determined within 2 minutes, and, in an emergency, typing of the blood group and the Rh factor is all that is necessary before transfusion. Type-specific blood that is not crossmatched has been given in numerous military and civilian series without serious consequences. While the type-specific blood is being transfused, the antibody screen and the crossmatch are carried out in the laboratory; the transfusion should be stopped if an incompatibility is found.

Ideally, type-specific blood should be similar in Rh factor as well as in ABO group; however, blood with the opposite Rh factor may be used in an extreme emergency or in

times of disaster or blood shortage. The patient may develop sensitization to the Rh factor. [12] This may affect a subsequent pregnancy if an Rh-negative woman is given Rh-positive type-specific blood. A male patient may likewise be sensitized to subsequent Rh-incompatible transfusions.

The third preference for an alternative to fully crossmatched blood for an emergency transfusion is group O blood. [13] In general, type-specific blood is preferable to group O blood. There is rarely a situation in which a few minutes cannot safely be expended to allow the blood bank to release type-specific blood. Nevertheless, exceptions may occur, in which case type O blood may be required. Such exceptions would include a trauma victim or a patient with a ruptured aneurysm or ruptured ectopic pregnancy who has not responded to crystalloid resuscitation in the field.

When type and Rh determination creates an unacceptable delay in transfusion, group O blood (either as whole blood or as packed cells) is transfused. Packed cells are preferred over whole blood. Group O negative *whole* blood was in the past designated the "universal donor" blood, because a recipient's naturally occurring antibodies (anti-A and anti-B) will not react with donor group O RBCs. Nonetheless, some donor serum may have a high titer of naturally occurring anti-A and anti-B antibodies capable of hemolyzing the recipient's (patient's) RBCs if large quantities of blood are transfused. True universal donor blood is low in anti-A and anti-B titer. Because group O donors are not regularly screened for unsafe levels of anti-A and anti-B titers, the use of even small amounts of group O *whole blood* that is not crossmatched is potentially dangerous. The significance of varying titers of anti-A and anti-B antibodies in the donor whole blood may be essentially eliminated if *packed cells* are used instead of whole blood. Other RBC antigens on type O RBCs may sensitize the patient or may cause antibody production, complicating future crossmatching or possibly causing future hemolytic

transfusion reactions.

Approximately 25% of patients receiving a transfusion of 5 or more units of type O whole blood develop hyperbilirubinemia suggestive of a minor hemolytic reaction. Large amounts of group O whole blood may cause the patient to acquire significant amounts of anti-A and anti-B antibodies that have been passively transfused; hemolysis of RBCs may then occur when the recipient's original blood group is subsequently transfused. In a resuscitation, one should continue to use group O blood if large amounts (i.e., >2 units) of *whole blood* have already been given.

One may transfuse both Rh-positive and Rh-negative group O packed cells in patients who are in critical condition. It is a common *misconception* that patients who are Rh-negative will have an immediate transfusion reaction if given Rh-positive blood. There is no particular advantage in the Rh factor determination because preformed, naturally occurring anti-Rh antibodies do not exist. Theoretically, individuals who are Rh-negative may become sensitized either through pregnancy or by previous transfusions, resulting in a delayed hemolytic transfusion reaction if Rh-positive blood is transfused. However, this scenario is very rare and is of no great clinical significance when compared with life-threatening blood loss. Many advise the routine use of *O Rh-positive* packed cells in all patients for whom the Rh factor has not been determined, except in females of childbearing age, for whom future Rh sensitization may be an important consideration. Once resuscitated with Rh-positive blood, patients may receive their own type without a problem. Because individuals with O Rh-negative blood represent only 15% of the population and the blood may be in short supply, it is reasonable to save O Rh-negative blood for Rh-negative females of childbearing potential and to use *group O Rh-positive packed cells routinely as the first choice for emergency transfusions*. In a study of emergency blood needs, Schmidt and colleagues reported 601 units of Rh-positive type O blood transfused to 193 patients, including 8 Rh-negative women, before blood type was determined. No acute hemolytic reaction occurred, and no women were sensitized. Rh immune globulin prophylaxis is recommended only for Rh-negative women with childbearing potential receiving Rh-positive blood.

If non-crossmatched blood is transfused, the laboratory should receive a plain (without a serum separator) red-top tube of venous blood as soon as possible to begin a formal crossmatch procedure. Brickman and coworkers have demonstrated that bone marrow aspirates obtained by an intraosseous needle can be used for crossmatching. [14]

Rh immune prophylaxis with human immune globulins (RhoGAM) is indicated for Rh-negative pregnant women who may be bearing Rh-positive children and may have fetomaternal transplacental hemorrhage. These events include bleeding in early pregnancy, such as spontaneous or elective abortion, ectopic pregnancy, and other potential causes of antepartum hemorrhage such as trauma. The indications for Rh immunoglobulin in *threatened abortions* is less clear but is advocated by some. The product suppresses the immune response of Rh-negative women to Rh-positive RBCs, and it is effective when given up to 72 hours after exposure to fetal erythrocytes. Dosing of Rh immunoglobulin is 50 mug IM for first-trimester bleeding and 300 mug IM for later bleeding. [15] In the setting of significant fetal-maternal transfusion (usually only in the

third trimester), doses may be increased. Rh immunoglobulin is prepared in the blood bank and the correct dose is suggested on an individual basis, following confirmation of Rh status, evidence of prior sensitization, and testing for fetal erythrocytes in the mother's blood.

## Transfusion Coagulopathy

Within the past 10 years it has been appreciated that pathologic hemostasis occurs following massive blood transfusions. [16] [17] [18] The exact cause of the transfusion coagulopathy is poorly defined and poorly understood. Although such abnormalities rarely develop within the time frame of the initial resuscitation in the emergency department, an understanding of the problem leads to a more intelligent approach to transfusion practices and the anticipation of potential problems. The term *massive transfusion* is loosely defined but is usually considered to be the transfusion of >10 units of blood to an adult (equivalent to one blood volume) within 24 hours. In patients who are given a transfusion that is equal to two blood volumes, only approximately 10% of the original elements remain. Considering the significant alteration in blood and blood products that occurs during storage, one can readily appreciate the underlying problem associated with such massive transfusions. The development of transfusion coagulopathy is multifactorial and in large part is related to tissue injury and duration of shock. [19]

Abnormalities in platelets and plasma clotting factors also play a role.

### Platelets

Transfusion coagulopathy is related partly to a diluting effect of the transfusion of blood deficient in platelets. Disseminated intravascular coagulopathy plays a secondary role in post-transfusion bleeding.

Banked whole blood and packed cells are devoid of functioning platelets. Dilutional thrombocytopenia is a well-recognized complication of massive transfusion, and a platelet count should be obtained routinely if >5 units of blood is transfused. As a general guideline, platelet therapy should be considered after the first 10 units of blood has been given, although the most useful parameter for estimating the need for platelet transfusions is the platelet count.

### Plasma Clotting Factors

Factors V and VIII are labile in stored blood and absent in packed cells. Fibrinogen is relatively stable in stored blood but is absent in packed cells. A deficiency of most clotting factors, especially factors V and VIII and fibrinogen, occurs with massive transfusions. This deficiency probably occurs on a "washout" (i.e., dilutional) basis, although the dynamics are poorly understood. The replacement of these factors may be required. Specific assays for the individual factors are available, but it is more practical to measure aPTT, PT, and fibrinogen levels. Fresh frozen plasma has been used to correct clotting factor abnormalities secondary to dilution from massive transfusions, but

its effectiveness has not been firmly established. Cryoprecipitate has also been used to replace factor VIII and fibrinogen, but it is rarely required, because FFP contains some fibrinogen. Fresh frozen plasma should be infused to correct the coagulopathy as indicated by clotting studies, but as a general guide, 1 to 2 units of FFP may be given empirically for each 5 to 6 units of blood in the massively traumatized or bleeding patient. Cryoprecipitate may be required if fibrinogen levels fall below 100 mg/dL and are not adequately supplemented with FFP.

## ORDERING OF BLOOD

Ordering a type and crossmatch procedure on a blood product implies that the decision has already been made to administer a transfusion. A "type and hold" or "type and screen" (no crossmatch) request alerts the blood bank to the *possibility* that a blood product will be required for the patient, so appropriate units can be acquired and kept on hand. A type and crossmatch procedure takes 45 minutes and restricts a unit of blood to a specific patient. This limits a valuable resource and should not be requested lightly. In the emergency department, a crossmatch procedure should be requested for a blood product only if the adult patient (1) manifests shock, (2) has *symptomatic* anemia (usually associated with a hemoglobin <10 g/dL) in the emergency department, (3) has a documented loss of 1000 mL of blood, or (4) requires a blood-losing operation immediately (e.g., thoracotomy). [20] A type and hold can safely be requested for all other situations in which a blood transfusion is considered possible during the patient's care; a desirable ratio of units crossmatched to units transfused can thus be achieved.

The number of units to be requested for a crossmatch procedure is determined by the size of the patient, the response of the patient to the injury and subsequent emergency treatment, and the presence of ongoing blood losses (e.g., arterial or massive gastrointestinal bleeding). In the majority of fatalities from massive hemorrhage, the patients die from hypovolemia rather than from lack of oxygen-carrying capacity. Specific guidelines for the administration of blood components are given in Table 29-5 .

Red blood cell preparations for transfusion are not routinely tested for the presence of sickle hemoglobin. Donors with sickle trait are not excluded, and blood with sickle trait can be safely given to almost every patient, because occlusion of blood flow caused by intravascular sickling would occur only in extreme conditions of acidity, hypoxia, or hypothermia that are unlikely to be compatible with life. Nonetheless, when transfusion is being performed in infants and patients with known sickle cell anemia, the blood bank should be alerted, and a "sickle prep" should be requested for donor blood to avoid the infusion of sickle-trait blood into such patients. Rare instances have occurred in which blood from a donor with a mild variant, such as SSC disease, caused massive intravascular sickling and death in a sick, hypoxic, acidotic infant. [21]

### Blood Request Forms

The most important part of ordering blood components for a patient is proper identification of the patient and the intended unit of blood. Transfusion of an incorrect unit is a potentially fatal error. *Most transfusion mistakes are clerical errors.* Several identification systems have been established to minimize the risk of improper

transfusions (Fig. 29-1) (Figure Not Available) . Just before administering the blood, the nurse or physician checks the identity of the numbered labels. In addition, the blood bank laboratory slip should identify the patient by name and number and should also contain the identification number of the unit of blood. Because most serious transfusion reactions are due to misidentification issues, one cannot be overcautious in adhering to these identification procedures (Fig. 29-2) (Figure Not Available) .

Usual procedures require a separate *blood bank request form* for each unit of RBCs or whole blood that is ordered. A number of units of FFP, cryoprecipitate, and platelet concentrates may be ordered on one form with proper identification (depending on individual blood bank procedures). When the blood bank indicates that the units ordered are ready, the person picking up the blood, along with the blood bank technician, checks the notation on the *blood release form (transfusion form)* to verify the identity of the patient (name, hospital number) and to ensure that the blood unit has been prepared for that patient (blood group and type, unit number). Immediately before administering the blood to the patient, the nurse or physician should check the release form, blood unit, and patient tag for identity as well as for the expiration date of the unit (Fig. 29-3) (Figure Not Available)

## INTRAVENOUS ADMINISTRATION

One should not open the unit of blood until and unless a free-flowing IV access line has been established in a large-bore vein. A 14-ga IV catheter is preferred, both to minimize hemolysis and to ensure rapid infusion of fluid for the treatment of hypovolemia or hypotension. When large amounts of blood must be given rapidly, administration by means of a high-flow infusion system is preferred. Standard central venous pressure lines are generally too small for adequate volume resuscitation in patients whose condition is critical. Likewise, the purpose of a large-bore infusion line is defeated if blood is piggybacked with an 18- to 20-ga needle through a side port in the infusion tubing. For an elective transfusion, however, blood may be given through a smaller needle. No significant hemolysis occurs when small-gauge (21-, 23-, 25-, and 27-ga) short needles are used for transfusion of fresh blood or packed cells in infants and children and when the maximum rate of infusion is <100 mL/hour. [22] For rapid infusion, however, the blood administration tubing is connected directly to the infusion catheter. The infusion site should be monitored for infiltration, infection, or local reactions. Antiseptic technique is essential (Figs. 29-4 (Figure Not Available) through 29-8 (Figure Not Available) ).

If the patient already has a suitable IV line in place, a solution of 0.9% normal saline only should be used to flush the system before administering the blood. Other IV fluids are *not* to be used because of the risks of hemolysis or aggregation (with 5% dextrose in water) or clotting (with lactated Ringer's solution). [23] No medications can be placed into the unit of blood or added to the infusion line for the same reasons.

### Filters

All blood and blood products should be infused only through an appropriate filter, such as those supplied in-line in the blood administration tubing sets. In the past, filtration

was required merely to keep the IV line from becoming blocked by clots, but the adverse consequences to the patient that result from infusing unfiltered blood products have now been recognized. Debris consisting of clots and aggregates of fibrin, WBCs, platelets, and intertwined RBCs (ranging in size from 15 to 200 mum) will accumulate progressively during storage of the blood unit from the first day of collection. The usual filter, made of a single layer of plastic with multiple 170-mum pores, traps larger particles and still allows for the rapid infusion of blood for 2 to 3 units before flow is greatly obstructed. Purified *components* of blood plasma

**Figure 29-8** (Figure Not Available) After the plastic tubing has been primed with saline, the blood flows through into the patient. *(Courtesy of Fenwal Laboratories, Deerfield, Ill.)*

can be safely administered through a filter with pores as fine as 5 mum.

It has been suggested that microaggregates of debris, which could pass through a 170-mum filter, may in part contribute to the syndrome of "shock lung" seen after transfusions of many units of blood in patients suffering from severe trauma and hemorrhage. Some practitioners therefore recommend the use of a microaggregate blood infusion filter with a mesh pore size of 40 mum (Fig. 29-9) (Figure Not Available) when multiple units of blood are administered to a trauma victim, to a patient with compromised pulmonary function, or to a neonate. Microaggregate filters tend to become blocked, impeding the rate of infusion more quickly, and are not commonly required in the emergency setting. In addition, whether the infusion of microaggregates (between 40 and 170 mum in size) is in fact harmful is still an unsettled issue. [24] Standard filters should be replaced after 2 to 3 units of blood product have been administered; most microaggregate filters should be changed after each unit. It is generally agreed that a significant number of platelets is removed by microaggregate filters, and some advise against using these filters when platelet packs are infused. Others believe that although platelets are removed with the microaggregate filters, the trapped platelets can be removed with saline flush without any significant loss. [25] Table 29-6 lists some available in-line filters.

**Rate of Infusion**

One unit of whole blood can safely be administered to a hypotensive patient at a rate of 20 mL/kg/hour. In the setting

**Figure 29-9** (Figure Not Available) Administration of blood through a microaggregate filter attached between the unit of blood and the hard plastic spike of the administration set. *(Courtesy of Fenwal Laboratories, Deerfield, Ill.)*

of hypovolemic shock and continued hemorrhage, there is no limit to the transfusion rate, and multiple units may be transfused simultaneously, even under pressure. In the stable patient, 1 unit of *whole blood* (500 mL) should be administered over approximately a 2-hour period (3 to 4 mL/kg/hour). After this time, RBCs begin to lose metabolic activity. In addition, the unit of blood, which is an excellent culture medium, is likely to become contaminated if bacteria and fungi are allowed to grow at room temperature. Packed cells should be given at approximately the same rate; plasma

products may be given more rapidly. In a patient with a healthy cardiovascular system, one should administer FFP more rapidly (about 15 to 20 minutes/unit) to correct coagulation deficits, because the coagulant activity begins to deteriorate rapidly after 20 to 30 minutes of thawing. In patients with severe anemia and congestive heart failure, a rapidly acting diuretic, such as furosemide, can be given (0.5 mg/kg IV) at the onset of transfusion to obviate circulatory overload.

If a transfusion of blood must be interrupted or delayed for some reason, the remainder of the blood unit should be returned to the blood bank. More convenient refrigerators in the emergency department or on the hospital unit *should not* be used to store blood products unless they are temperature-controlled or continuously monitored and alarmed.

Patients who are in hemorrhagic shock can receive blood through 2 large-bore catheters at different sites. Usually gravity provides a sufficient pressure gradient if the unit is raised higher above the patient to increase the rate of infusion when the clamps are wide open. If a pressure pump is used (Figs. 29-10 (Figure Not Available) and 29-11) (Figure Not Available) , the infusion can be more rapid. [26] A standard sphygmomanometer cuff should never be wrapped around a unit of blood to create increased infusion pressure, because the nonuniform application of pressure could burst the plastic bag containing the blood component.

One can dilute packed RBCs with *normal saline* (0.9% and without dextrose) before infusion simply by opening the clamps on the upper tubes of the Y infusion set and leaving the lower (recipient end) clamps closed. Although it is generally agreed (and recommended by the editors) that Ringer's lactate solution should never be mixed with blood because of possible clot formation, Cull and coworkers suggest that up to 150 mL of Ringer's lactate may be safely added as a diluent to each unit of packed RBC. [27] Therefore, blood inadvertently mixed with small amounts of Ringer's solution need not be routinely discarded. Dilution will allow for more rapid infusion by decreasing the blood viscosity, which is dependent on hematocrit, at the risk of increased volume. Alternatively, the direct addition of approximately 200 mL of normal saline to the bag of packed RBCs has been recommended to bring the hematocrit in the blood bag to approximately 45%.

## Rewarming

Blood is stored at approximately 4 °C to maintain cellular integrity and to prevent the overgrowth of microorganisms. Blood products usually passively warm to 10 °C by the time they are administered to the recipient. Adverse effects of hypothermia on cardiac conduction and flow rates are evident when rapid administration of a large volume of blood is performed without prewarming.

Various mechanisms have been used to warm blood to the ideal 35 to 37 °C. An ideal blood warmer should allow

| TABLE 29-6 -- In-Line Filters for Blood Transfusion | | |
|---|---|---|
| **Filter** | **Pore Size** | **Use and Contraindications** |
| Standard | | |
| Fenwal STD Blood Filter | 170 mum | All blood components |
| McGaw STD Blood Filter | 170 mum | All blood components |
| Special Use | | |
| Fenwal 4C2100 | 170 mum | Platelets, cryoprecipitate, antihemophilic factor concentrates, fresh whole blood |
| Microaggregate Filters | | |

| | | |
|---|---|---|
| Fenwal Microaggregate Blood Filter 4C2423 or 4C2131 | 20 mum | Removes most platelets and leukocytes from blood being transfused; do not use with fresh whole blood or concentrates of platelets or WBCs; primarily indicated for use with patients receiving *multiple transfusions* of stored blood and patients with *compromised pulmonary function* and for use in patients undergoing cardiopulmonary bypass; recommended for use in most newborns |
| Fenwal PDF-10 4C2428 | | |
| Intersept Blood Filter (Johnson & Johnson Co., New Brunswick, NJ) | 20 mum | |
| Alpha Micron-40 (Alpha Therapeutics Corp., Los Angeles) | 40 mum | |
| Bentley Disposable Blood Filter PF 127 (Bentley Labs., Inc., Irvine, Calif.) | 27 mum | |
| Hemonate (Gesco Labs., San Antonio, Tex.) | | |
| Swank In-Line Blood Filter IL-700 (Pioneer Filters, Inc., Beaverton, Ore.) | 40 mum | |
| | 20 mum | |
| Pall Ultipor SQ405 | 40 | |

| Pall Ultipor SQ405 (Pall Biomedical Products Corp., Glen Cove, NY 11542) | 40 mum | |
| --- | --- | --- |

liberal flow rates while preventing thermal hemolysis of blood cells. Commonly used devices are bath coils that allow a plastic tube to reside in a closely regulated warm water bath, as well as dry heat devices that allow blood to circulate through flat, thin bags sandwiched between aluminum

**Figure 29-10** (Figure Not Available) A controlled-pressure administration device for rapid infusion of blood products. *(Courtesy of Fenwal Laboratories, Deerfield, Ill.)*

blocks that contain electric heating elements. Both of these devices have relatively low flow rates and suboptimal thermal clearance. [28] Blood bag immersion in warm water baths is safe, but it is considered to be an imprecise and a slow method of rewarming. Although much interest surrounds

**Figure 29-11** (Figure Not Available) A rubber bladder is pumped up, and the blood unit is squeezed uniformly against a reinforced mesh. *(Courtesy of Fenwal Laboratories, Deerfield, Ill.)*

the use of microwave heating devices, the technique is not recommended by the Association of Blood Banks.

Rapid admixture warming is a promising alternative technique (see Chapter 23) . [29] The unit of whole blood is mixed with an equal amount of normal saline, which has been preheated to between 60 to 70 °C. Once mixed, the product is administered to the patient with a resultant delivery temperature of approximately 35 °C. This technique combines dilution of blood product and warming into one step. Regardless of the rewarming technique used, warming refrigerated blood to body temperature decreases its viscosity twofold to threefold and avoids venous spasm, thus facilitating transfusion.

## Monitoring

During the first 5 to 10 minutes and then every 15 minutes during a transfusion of any blood product, the patient must be carefully monitored for evidence of a transfusion reaction. Signs and symptoms that may be encountered are hives, chills, diarrhea, fever, pruritus, flushing, abdominal or back pain, tightness in the chest or the throat, and respiratory distress. A potentially life-threatening acute hemolytic transfusion reaction in a patient who has received prior transfusions may differ clinically from an allergic, nuisance reaction only by its effects on the patient's pulse and blood pressure. One can safely treat an allergic reaction to leukocytes or plasma proteins that cause hives, itching, fever, or chills by administering an antihistamine (but not into the blood infusion line) and stopping the transfusion.

When one encounters the following signs in a patient: an increase in pulse rate, a decrease in blood pressure, respiratory symptoms, chest or abdominal discomfort, or a sensation of "impending doom," one must *stop* the transfusion *immediately* and must infuse normal saline to maintain blood pressure and urine output. Samples of urine and blood should be sent to the laboratory to verify the presence of free hemoglobin. The blood bank should also receive a clotted sample of blood to reassess the presence of any immune reaction. If the conclusion of the blood bank evaluation is that the reaction is a nonhemolytic allergic response, premedication with antihistamines (diphenhydramine or hydroxyzine) and antipyretics is indicated before the next transfusion. Alternatively, washed cells could be used.

The patient in whom a hemolytic transfusion reaction is suspected should be treated vigorously and promptly. [30] Most mortality and morbidity is secondary to hypotension and shock leading to cardiovascular instability, renal insufficiency, respiratory manifestations, or hemorrhagic complications of disseminated intravascular coagulation. The initial treatment is directed at treating the hypotension by infusion of 5% dextrose in saline or lactated Ringer's solution or vasopressors, if required. The volume and rate of infusion are determined by blood pressure response. Symptomatic treatment with acetaminophen, a warming blanket, an inhaled or subcutaneous beta-agonist agent for bronchospasm or subglottic edema, or antipruritics or antihistamines is of secondary importance.

If an acute hemolytic transfusion reaction occurs, there may be some benefit from alkalinization of the urine with IV sodium bicarbonate to prevent the precipitation of free hemoglobin. Forced diuresis with mannitol to maintain the urine output at 50 to 100 mL/hour has also been advocated. The benefit from alkalinization and diuresis in the prevention of acute renal shutdown is uncertain, although the use of these techniques is commonly advocated. After shock is controlled, an assessment of hemostasis, respiratory function, renal function, and cardiac function will help direct later therapy of the complications; disseminated intravascular coagulation may call for the administration of plasma, platelets, or fibrinogen, and acute tubular necrosis may dictate careful fluid management. Hemolytic transfusion reactions have become unusual. They are rarely fatal and are usually attributable to an error in identification (such as can result from the treatment of two "John Doe" patients simultaneously).

Delayed, or "late," hemolytic transfusion reactions may occur days, or even weeks, after transfusion of RBCs. They are characterized by dropping hemoglobin levels, jaundice, hemoglobinemia, and indirect hyperbilirubinemia. [31] This complication is usually self-limited and is not life-threatening. Therapy is symptomatic, but future attempts at crossmatching for transfusions may be difficult because of the presence of RBC antibodies. Individuals so affected should wear identification tags or bracelets to alert medical personnel that prior transfusion reactions have occurred.

## CONCLUSION

On completion of a transfusion, an entry in the patient's record should be made to indicate the volume and nature of what was transfused and the presence or absence of

any reaction. The progress note, the transfusion record sheet, or the transfusion laboratory slip can be used for this purpose and should be signed and dated by the physician or nurse, in accordance with hospital policies. The bag in which the blood was stored might be discarded or returned to the blood bank, as individual policies dictate.

The practitioner should emphasize to the patient and family how critically important any blood transfusion is to the patient's care. It could then be suggested that the family consider arranging for replacement donations of units of blood to afford future patients the luxury of an ample, available supply of blood products.

# Chapter 30 - Pneumatic Antishock Garment

**Robert Norton**

Although a subject of much controversy, the pneumatic antishock garment (PASG) is widely available and commonly used, both by emergency medical technicians and paramedics in the out-of-hospital care of hypotensive patients and, to a lesser extent, by hospital emergency departments and critical care personnel. In its commercially available forms--MAST (David Clark Co., Inc., Worcester, Massachusetts) and Gladiator antishock pants (Jobst Institute, Inc., Toledo, Ohio) [1] --the PASG resembles a pair of high-waisted men's trousers. The devices are constructed from two layers of an opaque, airtight fabric sewn into three independently inflatable chambers (Figs. 30-1 (Figure Not Available) and 30-2) (Figure Not Available) .

These devices have been called by many names: MAST, military antishock trousers, medical antishock trousers, PASG, circumferential pneumatic compression device, shock pants, external counter pressure suit, air pants, MAST pants, MAST trousers, pressure pants, and G-suit. Other terms have been used to refer to the process of applying external pressure to the body, regardless of the device used. Examples are external (or circumferential) pneumatic compression and external counterpressure. [2] Because some of these names (e.g., MAST) are also registered commercial trademarks and because *pneumatic antishock garment* is the term adopted by the American College of Surgeons for its Advanced Trauma Life Support course, [3] PASG is used in this chapter to refer to the devices that are currently available.

Regardless of the name, the device has changed little in its design or application since 1903, when Crile created a "pneumatic suit" from a double layer of rubber. [4] One or both legs or the abdominal compartment could be inflated separately with a bicycle pump. Designed to manipulate blood pressure during head and neck surgery in the sitting position, the pneumatic suit was subsequently applied to the management of the trauma patient. [5] Leaks in the material inhibited widespread use of the device, and the principle of external counterpressure lay dormant until its rediscovery by the military (allegedly at Crile's suggestion) during World War II. [6] Reincarnated as the G-suit, the garment was used to provide momentary compression of up to 100 mm Hg to counteract the cerebral and retinal ischemia (with resultant loss of consciousness or vision) that occurred during certain maneuvers in high-speed aircraft. Medical interest was renewed with investigations performed by Gardner and Dohn. [7] They used a homemade G-suit in patients who were likely to experience postural hypotension (e.g., neurosurgical patients in the sitting position, patients in whom spinal anesthesia had been administered, and those with severe diabetic neuropathy). Their device subsequently became commercially available and consisted of a double-layered rectangular blanket wrapped completely around the patient from xiphoid to ankles. For the next decade or more, external counterpressure was used only in the hospital setting and usually as a last resort in cases of uncontrollable postoperative hemorrhage (Table 30-1) .

| TABLE 30-1 -- Early Uses of the PASG Device |
| --- |
| Retroperitoneal bleeding from massive pelvic trauma |
| Postoperative bleeding from hypocoagulation |
| Postural hypotension |
| Spontaneous rupture of the liver |
| Postoperative hemorrhage after: |
| Abdominal procedures |
| Nephrectomy |
| Prostatectomy |
| Renal biopsy |
| Tubal ligation |
| Hysterectomy |

| Leaking and ruptured abdominal aortic aneurysms |
| --- |
| Lower extremity fractures |
| Placenta percreta |
| Gastrointestinal bleeding |
| Ruptured ectopic pregnancy |

The military G-suit experience during the Vietnam War was the first recorded routine use of the external counterpressure principle in the preoperative stabilization of trauma patients. [8] The Army continued to develop the device until the current pants-like form was achieved. [9] The inventor of the modern-era trousers, B. H. Kaplan, became the first to adapt them to their next area of extensive use: civilian out-of-hospital application by paramedics and emergency medical technicians.

## MECHANISM OF ACTION

The mechanism of action of PASG has been the object of many experimental studies. Interpretation and comparison of results from these studies require attention to several factors: species of subjects, volume status, design of garment, inflation pressures, methods of hemodynamic measurements, and position of subjects (supine or tilted). The increase in blood pressure that results from application of PASG is due to at least three effects: enhanced venous return (autotransfusion), increased total peripheral resistance, and reduced volume loss from control of hemorrhage (Table 30-2) .

**Enhanced Venous Return**

The improvement in blood pressure seen during early PASG device studies was assumed to result from "autotransfusion" of blood from the venous system of the lower extremities and the splanchnic bed to circulation above the diaphragm. Reports stated that this mechanism could account for volumes of transfusion as large as 2 units (1000 mL) of blood, [11] yet experimental evidence to support such a large volume change has

not been found.

Increased central blood volume in humans after PASG application has been demonstrated by changes in thoracic radioactivity after 131 I-labeled albumin injection [12] and using a measurement of 99m Tc-labeled albumin. [13] Changes in normovolemic subjects are most marked when venous pooling is increased by tilting. [14] Attempts at quantification of "autotransfusion" in humans using nuclear scanning after radioactive red blood cell injection [15] have estimated that only 150 to 300 mL is actually autotransfused.

**Increased Total Peripheral Resistance**

Another explanation for the increase in blood pressure is an increase in total peripheral resistance after PASG application. This phenomenon has been well characterized in normovolemic and hypovolemic animal models. [16] [17] Using a hemorrhagic canine model, Niemann and colleagues demonstrated

| **TABLE 30-2** -- PSAG: Proposed Mechanisms of Action |
| --- |
| Blood pressure elevation |
| Increased total peripheral resistance |
| "Autotransfusion" |
| Control of bleeding |
| Direct pressure |
| Fracture stabilization |
| Effects on bleeding vessels |

|  |
|---|
| Decreased transmural pressure |

progressive increase in total peripheral resistance with inflation pressures of 40, 60, and 100 mm Hg. [16] Greater increases in total peripheral resistance occurred in the normovolemic group. Cardiac output increased only in the hemorrhagic hypovolemic group.

Hemodynamic responses to PASG in volunteer subjects are variable and sometimes contradictory. In normovolemic subjects, increases in central venous pressure and intrathoracic blood volume have been demonstrated following inflation of the PASG. [12] However, Burchard and colleagues reported no change in the cardiac index in 10 subjects after coronary artery bypass surgery, despite an increase in central venous pressure and left atrial pressure. [18] Using thermal dilution catheters in 10 subjects undergoing diagnostic cardiac catheterization, Rubal and associates found no increase in cardiac output or stroke volume, despite an increased total peripheral resistance and central venous pressure. [19] Using Doppler measurements, Hauswald and Greene reported no change in cardiac output in 10 healthy volunteers, despite a 75% reduction in aortic flow distal to the superior mesenteric artery and a 10% increase in blood pressure. [20] In a similar study using a head-up tilt to model hypovolemia in healthy volunteers, Mannering and associates found that PASG increased cardiac output and forearm blood flow in tilted patients. [21] Gaffney and colleagues found an elevated blood pressure and peripheral resistance with a decreased cardiac output and stroke volume during supine PASG application, but when the subjects were tilted 60° (head up), PASG produced a small elevation in stroke volume. [14] Savino and colleagues studied PASG use in elderly normovolemic patients and demonstrated a progressive decrease in the cardiac index and impairment in left ventricular function with increasing inflation pressures for half their patients. [22] Payen and coworkers demonstrated a significantly increased cardiac index in ventilated patients on positive end-expiratory pressure (PEEP) after inflation of PASG using pulmonary artery thermal dilution catheters to measure cardiac output. [13]

The conflicting results seen in animal and human studies can be explained in part by the volume status of subjects before inflation of the PASG, the overall effect of the subjects' homeostatic responses to increased afterload and preload, and the timing of the hemodynamic measurements. [23] Young normovolemic volunteers rapidly react to increased afterload, as manifested by an increased total peripheral resistance, with a change in heart rate or stroke volume--hence, their cardiac output may not change. Also, with normovolemia, changes in preload will have little effect on cardiac output. [17] Although no controlled hemodynamic studies have been done on hypotensive, hypovolemic volunteers, the effect is expected to be similar to the response in tilted subjects or hypovolemic animals. In that setting, the predominant response is an increase not in afterload but in preload, which results in increased stroke volume and, consequently, improved cardiac output for a variable duration. [17] [21] The complete

explanation for the mechanisms of action of PASG awaits further clinical research.

**Control of Hemorrhage**

The PASG can serve as a pressure dressing over external bleeding sites. Acting as a pneumatic splint, the device prevents continued bleeding provoked by motion at fracture sites. This is particularly efficacious with long-bone fractures and with retroperitoneal bleeding from pelvic fractures. In the management of hemorrhage associated with major pelvic fractures, the use of the PASG has been recommended as part of the initial management, followed by external fixation, surgery, or selective angiographic embolization. [24] One review of pelvic fractures has demonstrated radiographic realignment of an open-book, diastasis-type pelvic fracture after inflation of the PASG. [25] The PASG, combined with other nonsurgical therapy, also has been reported to be effective in controlling nontraumatic pelvic hemorrhage in obstetric and gynecologic patients. [26]

Principles of physics have been used to explain how external counterpressure can reduce flow from a sizable vascular defect while still allowing blood to circulate. [20]

*Laplace's law* ($T = \text{DeltaP} \times R$) determines the wall tension (T) for a cylinder (Fig. 30-3) (Figure Not Available) . T is the force tangential to the circumference of the vessel that tends to pull apart the edges of a longitudinal laceration (L in Fig. 30-3) (Figure Not Available) . Transmural pressure (DeltaP) is the difference between the intraluminal ($P_I$ ) and extraluminal ($P_E$ ) pressures ($\text{DeltaP} = P_I - P_E$ ). Increases in $P_E$ are produced by the PASG, and the tendency to bleed (DeltaP, T) declines. In addition, circumferential pressure causes the radius of the vessel (R) to decrease, limiting both of these contributions to the wall tension.

*The Bernoulli principle* takes more factors into account (Fig. 30-4) . It relates the rate of flow from the injured vessel (Q) to the surface area of the laceration site (A), transmural pressure (DeltaP), density of the fluid medium (rho), and the velocity (V) of the fluid flowing through the vessel. In this model the major action of the external counterpressure suit

is to limit transmural pressure along with the surface area of the laceration (Fig. 30-5) (Figure Not Available) . Many animal studies have tested these relationships, and nearly all models document decreased flow through lacerated vessels with external counterpressure and support one or the other of the equations listed.

# INDICATIONS

The list of clinical applications for PASG is modified frequently as new suggestions appear in the literature (Table 30-3) . Many indications that were controversial in the past are no longer so as experience with the PASG has increased. There are limited experimental and clinical data to support some suggested indications. Obviously, specific therapy directed at the underlying cause of the patient's illness or injury is the mainstay of successful treatment of any condition that may respond initially to the

supportive use of counterpressure.

**Hypovolemic Shock**

Several reports from Mattox and colleagues have changed the indication regarding the use of PASG for hypovolemic shock resulting from penetrating trauma. In a randomized clinical trial evaluating out-of-hospital trauma scores, fluid administration, emergency department and operative

| **TABLE 30-3** -- Proposed Indications for PASG Application |
| --- |
| Hypovolemic shock |
| Relative hypovolemia and hypotension |
| Spinal shock |
| Drug overdose |
| Septic shock |
| Anaphylaxis |
| Other uses |
| "Prophylactic" use (gastrointestinal bleeding, aortic aneurysm) |
| Stabilization of fractures |

| |
|---|
| Compression of external bleeding |
| Postoperative hemorrhage |
| Coagulation defects |
| Investigational uses |
| Cardiopulmonary resuscitation |
| Paroxysmal supraventricular tachycardia |

management, and survival, the investigators reported no advantage for use of the PASG in shock caused by penetrating abdominal injuries. [31] A continuation of the same study concluded that the PASG may be harmful when used with hypotensive patients who have penetrating thoracic wounds. [32] The study was conducted in an urban emergency medical services system with mean transport times <15 minutes and permitted concurrent out-of-hospital administration of large volumes of crystalloid fluid. Although no comparable controlled trials have been done for rural systems or for blunt abdominal trauma only, Chang and colleagues [33] used a prospective study design similar to that used by Mattox and colleagues [30] [31] [32] in a "medium-sized" urban community in which 50% of eligible patients had sustained blunt trauma. Although their series was small and a large proportion of cases assigned to the PASG group were excluded because of nonuse of the device, no differences in clinical outcomes were noted.

A retrospective study of patients who were severely hypotensive (systolic blood pressure, 50 mm Hg) as a result of both blunt and penetrating trauma found an improvement in survival associated with PASG use. [34] The authors concluded that the use of PASG in trauma patients with this degree of hypotension should be considered acceptable.

Recent animal models support the use of PASG in hemorrhagic shock and abdominal trauma. Using a controlled hemorrhagic shock model in sheep, Landau and coworkers found significant increases in mean arterial pressure, cardiac output, and tissue perfusion when PASG application was combined with hypertonic saline infusion. [35] Ali

and Duke studied a canine blunt splenic injury model and demonstrated improved survival and maintenance of carotid artery pressure along with decreased splenic and aortic blood flow after PASG inflation. [36] In an otherwise lethal abdominal aortic laceration model in pigs, Ali and associates found improved survival and decreased hemorrhage after PASG inflation. [37] However, in a prolonged (4-hour) hypotensive hemorrhage porcine model without concurrent volume resuscitation, PASG use was associated with greater fluid deficit, lactic acidosis, tissue edema, and hyperkalemia. [38]

In a retrospective study comparing air and ground transport of patients with primarily blunt multi-organ trauma, Moylan and associates identified use of PASG as one of several interventions in the air-transported group that were associated with improved survival. [39]

There are a number of advantages of the PASG over massive fluid replacement alone for patients with hypovolemic shock. The device is quickly applied and offers some immediate pressor response. Collapsed peripheral veins may become easier to cannulate after inflation. PASGs can be applied by persons without IV therapy skills, and the compressive effect may reduce hemorrhage from pelvic and intra-abdominal vessels.

Hence, until further studies clarify the exact role of PASGs in the management of trauma patients with hypovolemic shock, it seems prudent to continue to use them for selected patients with blunt trauma and severe hypotension (systolic blood pressure, <80 mm Hg); for patients with penetrating abdominal trauma and hypotension, *combined with* long transport times when out-of-hospital IV fluid therapy is not available; and for patients with signs of shock from other causes of hypovolemia.

## Other Uses During Hemorrhage

"Prophylactic" application (with or without inflation) may be helpful in potentially hypovolemic or hypotensive patients. Examples are patients with gastrointestinal bleeding or those with leaking abdominal aortic aneurysms. In patients with leaking abdominal aortic aneurysms, one should maintain the systolic blood pressure at approximately 100 mm Hg to avoid contributing to further hemorrhage. [42] [43] Pelvic or lower extremity fractures are well stabilized by these devices, adding significantly to the patient's comfort. [24] [25] The PASG may serve as a compression dressing over external bleeding. Controlling postoperative intra-abdominal hemorrhage has been a classic indication. [2] Bleeding aggravated by coagulation defects has also been responsive to external counterpressure. [44]

## States of Relative Hypovolemia and Hypotension

Other causes of shock in which the pneumatic suit might be helpful but for which data are limited include neurogenic (spinal) shock, shock secondary to anaphylactic shock, [45] drug overdose [46] and septic shock.

### Cardiopulmonary Resuscitation

Interest in the use of external counterpressure devices during cardiopulmonary resuscitation (CPR) is a result of recent changes in the way that blood flow is believed to occur during external chest compression (see Chapter 16) . During adult CPR, the "thoracic pump" mechanism of blood flow often predominates, and chest compression serves to raise intrathoracic pressure. Blood flows because of pressure gradients developed between intrathoracic and extrathoracic vessels. External counterpressure from the PASG is believed to augment CPR in two ways: (1) by reducing diaphragmatic excursion and therefore increasing intrathoracic pressures during compression, and (2) by compressing the infradiaphragmatic vascular bed, producing selective perfusion of vital structures above and increased peripheral resistance below the diaphragm.

Lilja and coworkers noted that PASG increased systolic blood pressure during CPR in 7 of 8 patients. [47] However, Mahoney and Murick reported no difference in initial resuscitation or survival between standard CPR and PASG-augmented CPR in a clinical, out-of-hospital study. [48] A review by Niemann and colleagues concluded that the use of PASG as an adjunct to CPR has not been proven to be beneficial and should remain experimental. [49]

### Paroxysmal Supraventricular Tachycardia

Case reports have documented cardioversion of paroxysmal supraventricular tachycardia to a sinus rhythm after the application of PASG in adults [50] and in a child. [51] The mechanism for the response has been attributed to reflex vagal excitation from increased aortic and carotid sinus pressure.

## CONTRAINDICATIONS

Pulmonary edema, congestive heart failure, and penetrating thoracic trauma are the current absolute contraindications to the use of PASGs (Table 30-4) . The increased venous return, decreased vital capacity, and elevation in pulmonary wedge pressure produced by these devices may aggravate preexisting pulmonary congestion.

### Penetrating Thoracic Injuries

External pressure applied below the diaphragm can increase the rate of blood loss from thoracic injuries. In a porcine model of thoracic aorta laceration, PASG inflation increased hemorrhage and mortality. [53] A retrospective review of penetrating cardiac wounds demonstrated an association between PASG inflation and a seven-fold lower survival rate. [54] In addition, Mattox and colleagues, in a large clinical trial, found that PASG use coupled with aggressive fluid resuscitation in hypotensive patients with penetrating thoracic injury was deleterious. [32] Finally, diaphragmatic injury may result in herniation of abdominal contents into the thorax. [55] Hence, use of PASG in the setting of penetrating thoracic injury is contraindicated.

## Cardiogenic Shock

Wayne has advocated the use of the PASG as a quickly reversible "fluid challenge" in patients with cardiogenic shock. [56] The intended use is to identify those patients in cardiogenic shock who might benefit from volume resuscitation and thus guide crystalloid infusion therapy in those individuals. Pressor agents are reserved for those patients who fail to improve or worsen with inflation. The complete

| TABLE 30-4 -- Proposed Contraindications to PASG Application |
|---|
| Absolute contraindications |
| Congestive heart failure |
| Pulmonary edema |
| Penetrating thoracic injuries |
| Relative contraindications |
| Pregnancy |
| Evisceration |
| Impaled foreign body |
| Lower extremity compartmental injury |

| |
|---|
| Lumbar spine instability |

"reversibility" of the effects of the device has not been well documented, particularly in the setting of a poorly functioning cardiovascular system. Five minutes into PASG inflation, central venous pressure, cardiac index, pulse, and central blood volume return to preinflation values. With the release of PASG pressure, the cardiac index increases to compensate for a fall in blood pressure and central venous pressure. [57] The changes after deflation are attributed to unmasking of the compensatory mechanisms (probably vasodilation) that had taken place during external counterpressure to normalize the patient's hemodynamics. Thus, mere removal of these devices after application does not necessarily restore the patient's cardiovascular system to a "baseline" state. In light of the short-lived effect seen with PASG inflation and the potentially deleterious effects following PASG deflation, use in the setting of cardiogenic shock is contraindicated.

## Relative Contraindications and Controversial Applications

Other relative contraindications that have been proposed include pregnancy, evisceration of abdominal contents, a foreign body impaled in the abdomen, and lumbar spine injury. For these situations, the leg chambers may be inflated without additional risk, and the relative risks and benefits of inflation of the abdominal binder can then be assessed. Circumferential burns and other injuries that suggest lower extremity compartmental injury also represent significant relative contraindications. Application of external counterpressure elevates the limb compartment pressure and increases muscle ischemia.

The use of the PASG in the patient with a penetrating abdominal injury in the setting of a rapid transport time and concurrent IV therapy does not appear to be advantageous. [30] [31] [32] Finally, because the hemodynamic response to PASG inflation is quite variable and at times deleterious in the elderly, [22] advanced age represents a relative contraindication to PASG use.

There are a number of situations in which PASG use has been discouraged by early writers. Although few data address these circumstances, some general review articles have discussed the judicious use of external counterpressure in these controversial settings. [58] [59] [60]

### Head Injuries

The only purely intracranial injury that can produce shock is transtentorial herniation. In the absence of this often fatal injury with its unique signs and symptoms, shock is usually the result of associated visceral injuries or spinal cord injury. However, fear that the PASG would increase cerebral edema and intracranial pressure has caused manufacturers to interdict use of the device in head injury. Several animal experiments

have found no significant effects of PASG inflation in hypovolemic or normovolemic dogs with or without an experimental "intracranial mass." The studies did document improvement in cerebral perfusion pressure with PASG use. [61] [62] [63] Gardner and colleagues evaluated the use of the PASG in 12 patients with severe head injury and an intracranial pressure <20 mm Hg. [64] They found increases in mean arterial pressure and cerebral perfusion pressure without adverse effect on intracranial pressure as long as the inflation pressure was <60 mm Hg. The use of PASG in patients with significant underlying elevations in intracranial pressure has not been studied.

**Cardiac Tamponade and Tension Pneumothorax**

Fluid infusion is a standard temporizing measure in cardiac tamponade. In a dog model of experimental cardiac tamponade, PASG inflation produced a doubling of mean arterial pressure and temporarily improved cardiac output. [65] Hence, the use of the PASG has been suggested in this setting when persons skilled in IV therapy and subsequent definitive treatment are unavailable. Tension pneumothorax is a similar state of compromised venous return that also might respond temporarily to the redistributed fluid volume resulting from PASG application. Palafox and associates, however, produced cardiac tamponade or tension pneumothorax in hypovolemic dogs and found a *decline* in arterial pressure and a rise in central venous pressure when the abdominal binder was inflated above 80 mm Hg. [63] This deterioration was believed to be caused by PASG-induced diaphragmatic elevation, which increased intrathoracic pressures and further compromised venous return. The clinical investigation of Mattox and coworkers suggests that IV therapy without PASG is safer in the patient with a penetrating thoracic injury during rapid transport for definitive therapy. [32] Hence, PASG use in hypovolemic patients with suspected cardiac tamponade or tension pneumothorax is relatively contraindicated.

# EQUIPMENT

Two of the principal manufacturers of PASGs are Jobst Institute, Inc., in Toledo, Ohio, which produces the Gladiator Antishock Pants, and David Clark Co. in Worcester, Massachusetts, which produces the MAST. Both brands come in two different sizes: adult and pediatric. Some features include X-ray transparency, perineal opening, and the ability to be applied over and under most traction splints. The trousers come with or without a gauge for monitoring air pressure. Advantages of using models with pressure gauges include the ability to (1) assess PASG inflation pressure during deflation for removal; (2) monitor garment pressure during air transport of the patient (in unpressurized aircraft); (3) evaluate PASG pressure in settings of environmental extreme temperatures; and (4) detect damaged air chambers, tubing, etc. The cost for a PASG, pump, and other equipment ranges from $535 to $640 for the adult size without gauges and $597 to $853 for the adult size with gauges. The cost for the pediatric size ranges from $465 to $806 for a unit without gauges and from $540 to $980 for a unit with gauges.

# PROCEDURE

**Application of PASG**

Before PASG application, it is useful to inspect the device and to establish the proper orientation (Fig. 30-6) (Figure Not Available) . Many find it helpful to mark the "up" side (inside) with paint or tape to aid in rapid correct application. The patient can be "logrolled" onto the opened trousers. Alternatively, with one person standing on either side and elevating the patient's

**Figure 30-6** (Figure Not Available) Pneumatic antishock garment application. (Courtesy of the American College of Surgeons, Committee on Trauma.)

legs, the trousers can be slid beneath to the buttocks. Then the patient's hips are elevated slightly, and the upper border of the trousers is placed at the costal margin. The cervical spine must be stabilized in those at risk for cervical injury. The medial portion on each leg binder is brought between the legs, and the Velcro fasteners are closed over each leg and over the abdomen.

Some emergency departments prefer to keep the deflated PASG on the resuscitation table at all times. When patients arrive, they are placed on the garment so that it is immediately accessible should its use be required later during treatment. An alternative is to assemble the trousers loosely in advance and slide them over the patient's legs. [66] During application, the operator places his or her own arms through the pants legs from the bottom, sliding the pants legs completely onto the arms. The operator then stands at the patient's feet, grabs the patient's toes, and lifts. An assistant pulls the trousers from the operator's arms and slides them over the patient, and the Velcro straps are retightened.

After device application, the foot pump hoses are attached to the stopcocks, and the foot pump is used to inflate the two leg compartments and then the abdomen. The operator can accomplish inflation faster if the compartments are initially filled by blowing into them. The lungs can provide volume more quickly than the pump during initial low-resistance inflation. After resistance to filling is met, the pump becomes more efficient at increasing trouser pressures. [66] Hanke and associates found no difference in redistribution of blood volume between simultaneous and sequential inflation of the leg and abdominal compartments. [67] Jennings and coworkers similarly found no hemodynamic benefit to simultaneous device inflation. [68] Therefore, it seems prudent when using the device for hypotension to inflate the leg compartments first and then check the blood

pressure. If an adequate blood pressure is obtained, the abdominal compartment does not need inflation. If the blood pressure is inadequate, the abdominal compartment is inflated. Blood pressure and pulse should be closely monitored during the procedure, and inflation should be stopped if the systolic blood pressure exceeds 100 mm Hg.

Pediatric devices are available, although they are used infrequently. In the absence of these smaller garments, one can efficiently manage small children by wrapping them in one leg of the adult-sized trousers. A folded sheet or blanket can be placed between the

bony prominences of the lower extremities. The inflation pressure should be titrated to a satisfactory pulse and blood pressure response.

The PASG alone can passively splint lower extremity fractures. Of the commonly available traction devices, only the Sager traction splint can be applied after the trousers are in place and inflated. The Sager splint can be used either inside or outside the device to splint one or both legs at the same time. The Hare traction splint and the Thomas splint are significantly more awkward to use with the PASG inflated. The Hare and Thomas splints present some risk of damage to the trousers themselves and result in uneven application of circumferential pressure (see Chapter 49) . As discussed later, the application of traction in combination with external counterpressure exacerbates compartmental pressures in the lower extremities. [69]

## Inflation Pressure

Of the two major PASG devices that are available, only one, the Jobst Gladiator, has a pressure gauge (or gauges) for monitoring pants pressure. As with any such piece of equipment, gauges should be periodically checked for accuracy. The other device uses pressure-relief ("pop-off") valves that limit inflation pressures to 104 mm Hg. It is reasonable to assume that most side effects and complications of these devices are proportional to the magnitude of PASG pressure and duration of pressure application. Most animal experiments and some clinical studies have suggested that hemorrhage control (presumably control of venous bleeding) in otherwise stable (often postoperative) patients is often accomplished with <40 mm Hg of inflation pressure. [2] "Autotransfusion" and blood pressure elevations may require a higher inflation pressure, although Hanke and colleagues reported a significant increase in central blood volume at 40 mm Hg and only minor progressive increases at 100 mm Hg. [67] Wayne and MacDonald suggested that most out-of-hospital patients in shock will not demonstrate blood pressure elevation at a 30-mm Hg inflation pressure. [70] The goal is to achieve a systolic blood pressure of approximately 100 mm Hg at the lowest inflation pressure possible. Obviously, time constraints may not permit careful titration of inflation pressure to blood pressure response; the patient in extremis may need simultaneous inflation of both leg and abdominal compartments to 100 mm Hg immediately on presentation.

## Deflation

### When?

In the usual case, deflation may be considered when the combination of PASG and other resuscitative measures (e.g., fluids, hemorrhage control) has resulted in restoration of satisfactory vital signs. The gradual deflation procedure to be outlined should be followed. The presence of any contraindications to continued use (e.g., congestive heart failure, renal disease, pulmonary disease) should also be considered. The presence of a coagulopathy may be a relative contraindication to removing the device.

**Where?**

If emergency surgery is indicated, the operating table may be the best place to deflate the PASG. After all monitoring and venous lines have been started, the anesthesiologist is prepared to monitor the patient, and the surgeons are in attendance, the abdominal portion can be deflated (preferably slowly--see next section). The leg compartments can be left inflated for an abdominal procedure until internal hemorrhage is better controlled. In most situations, after adequate volume resuscitation, PASG deflation can be safely performed in the emergency department.

**How?**

The PASG should be deflated gradually. Rapid deflation of the PASG in the hypovolemic patient can result in a catastrophic drop in blood pressure. The hemodynamic changes seen during deflation result from a rapid reduction in left ventricular afterload followed by a decrease in preload. [71] This process may be exacerbated by the sudden release of lactic acid and other vasoactive chemicals pooled in the abdomen and the lower extremities. [8] [72] Hence, deflation, particularly in the setting of prolonged PASG use, should be approached with caution.

Deflation should begin with the abdominal compartment. Small quantities of air are released, and the patient's blood pressure is rechecked. If no blood pressure drop occurs, more air should be released with repeated blood pressure measurements. If the systolic blood pressure falls >5 mm Hg, deflation should be halted and more fluids given until blood pressure is restored. After the abdominal compartment is deflated, the legs should be deflated in a similar manner.

## COMPLICATIONS AND DISADVANTAGES

A number of specific adverse effects have been noted in association with PASG use (Table 30-5) . In addition, patients with penetrating abdominal and thoracic trauma whose management included PASG use in conjunction with vigorous out-of-hospital fluid resuscitation have been reported to have an increased mortality rate when compared with patients treated with fluid resuscitation alone in one urban out-of-hospital setting. [31] [32] As noted in the Indications and Contraindications sections, the use of PASG in patients with penetrating truncal injuries in the setting of rapid transport times or IV fluid therapy does not appear to be warranted. Further discussion of other specific complications follows.

**Hypotension**

Clinical experience has shown that the major life-threatening complication resulting from the use of the PASG in hypovolemic patients is sudden and severe hypotension resulting from precipitous removal of the device in the absence of adequate fluid resuscitation. [8] [41] Although emergency department

| TABLE 30-5 -- Complications and Disadvantages of PASG Application |
| --- |
| Hypotension after removal |
| Metabolic acidosis |
| Respiratory compromise |
| Decreased renal perfusion |
| Other (infrequent) complications |
|   Pulmonary edema, congestive heart failure |
|   Compartment syndromes |
|   Increased wound bleeding |
|   Urination, defecation, vomiting |
|   Skin breakdown |
|   Lumbar spine movement |

| |
|---|
| Mechanical problems and disadvantages |
|   Limitation of diagnostic and therapeutic procedures |
|     Physical examination |
|     Urinary catheterization |
|     Peritoneal lavage |
|     Vascular access |
|   Environmental influences |
|     Barometric pressure |
|     Temperature |

physicians and emergency medical technicians are well aware of the problem, many consultants are not. When faced with a patient encased in vinyl from xiphoid to ankles, some consultants have rapidly removed these devices, often with disastrous consequences.

**Metabolic Acidosis**

Metabolic acidosis has been the most reproducible abnormality after application of external counterpressure. Wangensteen and associates first reported this "detrimental" effect of external counterpressure. [73] They noted acidosis (to a pH of 7.01) and an increased lactate-to-pyruvate ratio in hypovolemic dogs after 4 hours of external counterpressure. Hypovolemic dogs without external pressure had less acidosis and

lived longer. Although treatment of hypovolemia with a PASG alone and without concomitant fluid replacement is an unusual situation, these animal studies led to the recommendation to limit PASG application time. Human studies have shown mild metabolic acidosis (pH 7.33 to 7.36), and other reports suggest that the acidosis is not a problem clinically. [74] [75] Although metabolic acidosis may occur, close monitoring of arterial blood gases and correction with bicarbonate as needed should adequately address this potential problem.

## Respiratory Compromise

Abdominal binding invariably produces subjective effects on respiration; alert patients frequently complain of shortness of breath when the abdominal compartment is inflated. However, McCabe and colleagues found no changes in inspiratory and expiratory reserve volumes, maximum breathing capacity, or tidal volume in their volunteers. [76] Similarly, Batalden and coworkers found no pulmonary complications in 10 patients with external counterpressure garments who underwent positive-pressure ventilation for 24 to 48 hours. [77] However, Burdick and colleagues found atelectasis, pulmonary edema, or pneumonia in 14 of 28 similar patients. [75] Cogbill and coworkers demonstrated mild impairment of pulmonary function in healthy individuals and those with airflow obstruction when the PASG was inflated. [78] The impairment was restrictive rather than obstructive and was not clinically significant with inflation pressures <50 mm Hg. Some studies suggest that impaired pulmonary function results from mechanical binding of the thorax and decreased excursion of the diaphragm. [76] [79] Newer designs of the device incorporate smaller abdominal compartments, which should not be applied at or above the costal margin and have resulted in less restriction of vital capacity. [76] In general, investigators concerned about respiratory compromise have recommended careful attention to arterial blood gases combined with controlled positive-pressure ventilation in those patients requiring prolonged external counterpressure. [11] [75]

## Decreased Renal Perfusion

In one study, 10 subjects in PASGs for 4 to 24 hours had no significant alterations in renal blood flow. [80] However, reversible abnormalities in renal hemodynamics have been reported with external counterpressure. Normal human subjects experiencing increased abdominal pressure had a 24 to 48% decline in effective renal plasma flow and glomerular filtration rate, decreased urine output, elevated renal vein pressures, and increased urine specific gravity. [81] These changes reversed after decompression.

## Infrequent Complications

A number of case reports document some infrequent but serious complications of the PASG.

*Pulmonary edema and congestive heart failure* have been attributed in isolated cases to increased pulmonary venous return and elevated pulmonary capillary wedge pressures. [2] [75]

*Lumbar spine instability* is another potential complication of the PASG. Older PASG designs included an abdominal bladder that inflated in the back as well as over the abdomen. Rockwell and coworkers reported a case in which a lumbar spine injury was believed to have been aggravated by inflation of a circumferential abdominal bladder. [82] They also demonstrated graphically on a volunteer the marked exaggeration of lumber lordosis that is possible with the older style garment. Newer designs of commercially available suits limit inflation to the anterior aspect of the abdominal compartment. The abdominal portion of all designs of the PASG should be used cautiously in patients at high risk for an unstable lumbar spine injury.

*Lower extremity compartment syndromes* following use of PASG have been reviewed elsewhere. [83] Increased pressure within the limits of a fascial compartment results in impaired microcirculation. The compartment pressure under the PASG is dependent on the PASG inflation pressure, the mean arterial pressure, and the application of passive traction. [69] [84] During hypotension, with full PASG inflation, the compartment pressure may exceed the mean arterial pressure. [84] Many factors contribute to the development of a compartment syndrome, including prolonged shock, inflation pressure (and duration of inflation), reperfusion edema, and local tissue injury. [83] It is not completely understood which of the multiple factors are most significant in the compartment syndrome associated with PASG use. Some cases are related to a lower extremity fracture with prolonged application of

PASG, [85] but other cases have occurred in the absence of lower extremity injury. [86] Improper deflation technique (i.e., leaving the abdominal compartment inflated after deflation of the extremity compartments) may facilitate the syndrome. [87] The overall frequency of this complication seems quite low. In their review of 1120 patients, Wayne and MacDonald reported no cases of compartment syndrome. [70] Mattox and associates found an incidence of anterior compartment syndrome in approximately 1% of their PASG patients and none of the patients without PASG. [30]

*Increased bleeding from lower extremity wounds* after suit inflation was attributed in 2 cases to improvements in blood pressure. [8] The bleeding responded to rebandaging but required temporary deflation of the suit. Such bleeding may be hidden in bandages under the devices and should be considered in the differentiation of persistent hypotension from other conditions.

*Stimulation of urination, defecation, and vomiting* by suit inflation was noted as a complication in early papers. [40] Spontaneous defecation has been a "complication" in patients being treated for gastrointestinal bleeding with these devices. [78]

*Skin breakdown at pressure points* with prolonged use has prompted the suggestion to pad bony prominences. [77]

*Diaphragmatic herniation* has been reported following PASG inflation. [55] A sudden deterioration in blood pressure or respiratory status following PASG inflation should alert the clinician to this possibility. Using a porcine model of diaphragmatic injury, Ali and Qi demonstrated a significant increase in mortality rate after PASG as a result of

cardiorespiratory deterioration caused by the increase in intra-abdominal pressure. [88] If significant herniation of abdominal contents into the thoracic space has occurred, deflation of the device may not fully resolve the clinical deterioration.

## Mechanical Problems and Disadvantages

### Limitation of Diagnostic Evaluation and Therapeutic Procedures

Physical examination of the lower extremities and the abdomen is limited by the PASG. A transparent suit that allows visualization of the lower extremities has been marketed but has other disadvantages and is not commonly used. For the more common nontransparent device, the examiner must not remove the suit precipitously. In a stable patient, one compartment at a time should be deflated slowly, with careful monitoring of vital signs. If necessary for treatment of hypotension, the compartment that has just been deflated may be reinflated. Similar problems exist with the performance of diagnostic peritoneal lavage. Urinary catheterization is challenging, particularly in female patients, even with the opening provided in the garments.

Vascular access also can be a problem. Lower extremity cutdowns are difficult to perform, and a pressure bag is required to produce flow of IV solutions. However, pressurized IV fluid infusion beneath inflated PASGs reaches the central circulation promptly and in significant amounts. [89] Access to the femoral artery and vein with the PASG inflated also is severely limited, although newer designs incorporate openings at the inguinal area for better access.

### Environmental Influences on Device Pressures

With the current popularity and availability of helicopter ambulance transport, the role of Boyle's law (i.e., the volume of gas is inversely proportional to its pressure) must be reemphasized. [90] [91] As the helicopter rises, the air in the suit expands, increasing the trouser pressure. Decreasing altitude has the opposite effect. Similarly, as ambient temperature increases, trouser pressure rises, and vice versa. Most helicopter transports occur at sufficiently low altitudes that the effects of altitude are not of concern, but when the "optimal" inflation pressure is determined at the scene of the injury, changes produced by movement into the controlled climate of the ambulance or hospital must be predicted and recognized.

# CONCLUSION

The PASG is a useful adjunctive device in the care of the hypotensive patient. When the garment is used, improvements in blood pressure and pulse are often dramatic. In certain situations the device may slow hemorrhage, and external counterpressure can "buy time" until definitive therapy is instituted. A major hazard is the profound hypotension that can result from precipitous removal of the device by those who are unfamiliar with this possibility. Although the device is not helpful in the urban out-of-hospital setting for management of penetrating trauma when crystalloid therapy is

rapidly available, it appears to have a role in other settings.

# Chapter 31 - Local and Topical Anesthesia

**Michael Orlinsky, Edwin Dean**

The use of local anesthetic agents is an important aspect of the everyday practice of emergency medicine. This chapter provides an understanding of the mechanism of action; the nuances of clinical use; and the prevention, recognition, and treatment of adverse reactions to commonly used anesthetics. Detailed technical guidance for the performance of topical and infiltrative local anesthesia is provided.

## BACKGROUND

The first local anesthetic was cocaine, an alkaloid contained in the leaves of the *Erythroxylon coca* shrub from the Andes Mountains. Early Incan society used cocaine for invasive procedures, including cranial trephination.

In 1884, Koller, a colleague of Freud, used cocaine topically in the eye and is credited with the introduction of local anesthesia into clinical practice. [1] In the same year, Zenfel used a topical solution of alcohol and cocaine to anesthetize the eardrum, and Hall introduced the drug into dentistry. [2] In 1885, Halsted demonstrated that cocaine could block nerve transmission and laid the foundation for nerve block anesthesia. [3] The search for alternatives to cocaine led to the synthesis of the benzoic acid ester derivatives and the amide anesthetics.

Although local anesthetics had been used for more than half a century, it was not until the 1960s that a specific understanding of the physiochemical properties, mechanism of action, pharmacokinetics, and toxicity of these agents emerged.

## PHARMACOLOGY AND PHYSIOLOGY

### Chemical Structure and Physiochemical Properties

Most useful local anesthetic agents share a basic chemical structure:

Within this basic structure, each agent's specific chemical composition determines its main physiochemical properties: negative log of dissociation constant (p$K_a$ ), partition coefficient (a measurement of lipid solubility), and protein binding. Each property, in turn, is the principal determinant of an agent's pharmacologic activity: onset of action, potency, and duration of action, respectively. However, in vivo the activity of a given agent may be altered by other factors unrelated to physiochemical properties, and therefore the relationship between physiochemical properties and clinical activity is not exact.

The linkage (intermediate chain) between the aromatic and hydrophilic segments is either an amino-ester or an amino-amide; these two chemical structures form the basis for the two main classifications of local anesthetics. *Ester*-type agents include procaine,

chloroprocaine, cocaine, and tetracaine. The *amide*-type agents include lidocaine, mepivacaine, prilocaine, bupivacaine (Marcaine), and etidocaine. The main difference between esters and amides is their metabolic pathways. Esters are hydrolyzed by plasma pseudocholinesterase, whereas amides are metabolized in the liver through enzymatic degradation. Within the ester or amide group, alterations in chemical structure to either the aromatic or the hydrophilic portion may affect the rate of metabolism and create a different activity profile for each agent within a given group.

Chemically, local anesthetics are poorly soluble weak bases. To be commercially available in solution, an agent is combined with hydrogen chloride to produce the salt of a weak acid. In the resulting acidic solution, salts exist both as uncharged molecules (nonionized) and as positively charged cations (ionized). The uncharged form is lipid soluble, enabling it to diffuse through tissues and across nerve membranes, which the charged form cannot do. The ratio of uncharged to charged forms depends on the pH of the medium (vial solution or tissue milieu) and on the p$K$a of the specific agent. The p$K$a is the pH at which 50% of the solution is in the uncharged form, and 50% is in the charged form. Because the p$K$a is constant for a specific agent, the relative proportion of these forms is dependent on the pH of the solution in accordance with the Henderson-Hasselbalch equation:

$$pH = pK_a + \log [\text{uncharged}]/[\text{charged}]$$

When the pH of the solution or tissue decreases, a given agent exists more in its ionized, charged form; conversely, when a pH increases, the agent exists more in its nonionized, uncharged form. Because the nonionized portion is the form of drug that can diffuse through tissues and nerves, manipulation

| TABLE 31-1 -- pH and Additives of Amide Local Anesthetics | | | |
|---|---|---|---|
| **Solution Content** | **pH (Range)** | **Preservative (Methylparaben)** | **Antioxidant** |
| Plain, single dose | 4.5-6.5 | - | - |
| Plain, multidose | 4.5-6.5 | + | - |
| Commercial epinephrine single dose | 3.5-4.0 | - | + |

| | | | |
|---|---|---|---|
| Commercial epinephrine multidose | 3.5-4.0 | + | + |
| Prepared epinephrine single dose | 4.5-6.5 | - | - |

of the pH of the solution is a useful tool for the physician (see Factors Affecting Neural Blockade).

Local anesthetic agents are available in plain solution (i.e., without epinephrine), both in single-dose vials or ampules and in multidose vials. For most agents the pH is >5. Multidose vials contain methylparaben as the antibacterial preservative. Local anesthetics also are available premixed with epinephrine and are marketed in single-dose or multidose vials. They contain an antioxidant (sodium bisulfite or sodium metabisulfite) to prevent deactivation of the vasoconstrictor. These solutions must be adjusted to a more acid pH--approximately 3.5 to 4.0--to maintain the stability of epinephrine and its antioxidant. As with plain solutions, multidose anesthetic vials containing epinephrine also contain methylparaben (Table 31-1) .

How an anesthetic is supplied has several implications. First, the lower pH of epinephrine-containing solutions may both make the agent more painful on infiltration and may increase its ionized concentration, thereby delaying its onset. Adding epinephrine to plain solutions just before use does not change the pH of the solution and is reasonable for the physician to do if pain or onset time becomes an issue in a given patient. Second, the additive methylparaben found in multidose vials has been implicated in many allergic reactions. Understanding this fact helps when dealing with patients who claim to be "allergic" to local anesthetics.

## Nerve Structure and Impulse Transmission

### Functional and Structural Components of a Peripheral Nerve

The functional unit is the nerve fiber, which in its practical definition includes the nerve axon and its surrounding Schwann cell sheath. Two distinct arrangements of this nerve sheath are recognized (Fig. 31-1) (Figure Not Available) : (1) unmyelinated nerve fibers have a single Schwann cell surrounding several axons, and (2) myelinated nerve fibers have a Schwann cell wrapped around a single axon, forming a myelin sheath. Junctions between sheaths along the axon are called *nodes of Ranvier*; they contain sodium channels necessary for depolarization. As myelin sheath thickness increases from autonomic to sensory to motor fibers, the nodes of Ranvier with their sodium channels are spaced farther apart.

The most important structure affecting nerve impulse transmission is the axon membrane (Fig. 31-2) (Figure Not Available) . The membrane is composed of a double layer of phospholipid into which are embedded protein molecules that serve as channels

containing pores for the movement of ions into and out of the cell. Most pores have a filter that allows for ion-specific movement, a gate that regulates the passage of ions, and a sensor mechanism that opens or closes the gate.

Bundles of nerve fibers (Fig. 31-3) (Figure Not Available) are embedded in collagen fibrils called *endoneurium* and are surrounded by a cellular layer, the *perineurium*. The perineurium functions as a diffusion barrier and maintains the composition of extracellular fluid around the nerve fibers. Surrounding the entire structure is the outer layer of a peripheral nerve, the *epineurium*, which is composed of areolar connective tissue.

**The Nerve Impulse and Its Transmission**

At rest, the inside of a nerve fiber, or axoplasm, is negative (-70 mV) compared with the outside. This resting potential is the net result of the marked differences in ionic concentrations on each side of the axonal membrane and the forces that tend to maintain that difference. Specifically, there is a surplus of sodium extracellularly and of potassium intracellularly. The sodium channel is closed, preventing these ions from moving along their concentration gradient (out in). Although potassium can leave the cell to follow its concentration gradient (in out), it is prevented from doing so completely because of the need to maintain electrical

**Figure 31-3** (Figure Not Available) Cross section of peripheral nerve. *(From Wildsmith JAW: Peripheral nerve and local anesthetic drugs. Br J Anaesth 58:692, 1986. Reproduced by permission.)*

neutrality inside the cell. Potassium reaches an equilibrium between its concentration gradient and the electrochemical gradient, thus creating the negative resting potential.

When a nerve is excited, the sodium channel opens. At first, a slow influx of sodium ions occurs, but after a critical threshold is reached, sodium enters the cell rapidly, following the electrochemical gradients and its own concentration gradient (depolarization). The influx of sodium is halted when the membrane potential reaches +20 mV, because the inward concentration gradient for sodium is balanced by the outward electrochemical gradient. The sodium channels then close, but potassium continues to move out until the resting potential is again achieved (repolarization). When the excitation process has been completed and the nerve cell is electrically quiescent, the relative excess of sodium inside the cell and potassium outside the cell is then readjusted by the adenosine triphosphate (ATP)-dependent sodium-potassium pump.

During this process, known as the *action potential*, depolarization of a portion of the nerve causes a current to flow along the adjacent nerve fiber. This current makes the membrane potential less negative and actuates the sensor to open the next sodium channel. The action potential cycle is repeated, thereby propagating the impulse. Nerve conduction is essentially unidirectional because in the segment just depolarized (1) the sodium channel is not only closed but inactivated as well, and (2) delayed closure of specific potassium channels prevents the critical threshold from being reached. In unmyelinated nerve fibers, an impulse spreads continuously down the axon. In myelinated nerve fibers, sodium channels are situated at the nodes of Ranvier, and

current flows from node to node, causing intervening segments to depolarize at once. This saltatory conduction accounts for the faster rate of impulse transmission in myelinated fibers.

## Mechanism of Action

The way local anesthetic agents produce nerve conduction blockade can be explored practically by discussing 3 related concepts: (1) the active form of the agent responsible, (2) the physiologic basis for blockade, and (3) the method by which this process is accomplished.

### The Active Form

An anesthetic solution exists in uncharged and charged forms, with the concentration of the uncharged form increasing in more alkaline milieus. It is only this uncharged (lipid-soluble) form that can cross tissue and membrane barriers. Once the agent is through a barrier, the uncharged form re-equilibrates into uncharged and charged forms, the proportion of which again depends on the prevailing pH.

Because local anesthetics were found to be more effective in alkaline solutions, it was originally thought that the uncharged form was responsible for conduction blockade. However, the increased effectiveness of alkaline solutions is currently believed to be due to increased agent penetration through tissue barriers, while the cationic charged form is responsible for actual blockade. Although both the charged and the uncharged forms contribute to nerve blockade, the charged form predominates for most of the common anesthetic agents.

### The Physiologic Basis for Blockade

Prevention of sodium influx across the nerve membrane is the physiologic basis for conduction blockade. Local anesthetics decrease sodium influx, which decreases the rate of rise and amplitude of depolarization. When sufficient anesthetic is present, the firing threshold will not be reached, thereby preventing the action potential from forming. With no action potential, no impulse can be transmitted, and conduction blockade is achieved. The end result is local anesthesia.

### The Cellular Mechanism of Blockade

The means by which anesthetic agents prevent sodium influx is still not completely understood. It is believed that the cationic charged form blocks the action potential from inside the membrane; the agent enters the sodium channel from the axoplasmic side and binds to a receptor about halfway down the electrical gradient. [4] [5] This "specific receptor" theory is well accepted and is considered the predominant mechanism in preventing sodium influx. However, this theory cannot account for the action of benzocaine or other neutral compounds or the uncharged base form of the common local anesthetics.

In summary (Fig. 31-4) (Figure Not Available) , when a commonly used local anesthetic (other than benzocaine) is applied perineurally, it equilibrates into its uncharged and charged forms based on the tissue pH and agent p$K_a$ . The uncharged lipid-soluble form penetrates tissue, nerve sheath, and nerve membrane to gain access to the axoplasm, wherein it re-equilibrates into both forms. The charged form enters the sodium channel to effect blockade by decreasing sodium influx. The uncharged base is also involved with sodium channel blockade, but the exact nature of this mechanism is unknown.

## Factors Affecting Neuronal Blockade

A local anesthetic's activity profile (onset, potency, duration) and its ability to produce a differential blockade in mixed

<table>
<tr><td colspan="4"><strong>TABLE 31-2</strong> -- Activity Profile with Primary Physiochemical Determinant</td></tr>
<tr><th>Agent</th><th>Onset:<br>p<em>K</em>a</th><th>Potency:<br>Lipid<br>Solubility</th><th>Duration:<br>Protein<br>Binding</th></tr>
<tr><td>Tetracaine</td><td>Slow</td><td>8</td><td>Long</td></tr>
<tr><td>Procaine</td><td>Slow</td><td>1</td><td>Short</td></tr>
<tr><td>Chloroprocaine</td><td>Fast</td><td>1</td><td>Short</td></tr>
<tr><td>Lidocaine</td><td>Fast</td><td>2</td><td>Moderate</td></tr>
<tr><td>Mepivacaine</td><td>Fast</td><td>2</td><td>Moderate</td></tr>
<tr><td>Prilocaine</td><td>Fast</td><td>2</td><td>Moderate</td></tr>
</table>

| Bupivacaine | Moderate | 8 | Long |
|---|---|---|---|
| Etidocaine | Fast | 4-6 | Long |

nerves are a function of physiochemical properties, the physiologic environment, and, to some extent, manipulation by the physician.

**Onset of Action**

The p$K_a$ of an anesthetic is the primary physiochemical factor that determines onset of action. For a lower p$K_a$ , more of the lipid-soluble uncharged form is present, which thereby increases the penetrating ability and shortens the onset of action (Tables 31-2 and 31-3) . Although in isolated nerve fibers onset of action directly parallels p$K_a$ , additional in vivo physiochemical factors exert an influence. For example, prilocaine and lidocaine have the same p$K_a$ , but lidocaine's onset is faster because of its enhanced ability to penetrate through nonnervous tissue.

The site of administration has significant clinical impact. As the amount of interspersed tissue or the size of the nerve sheath increases, the onset times are prolonged for all agents because of the greater distance that the agent must travel to reach its receptor. The role of p$K_a$ becomes more important as the tissue barrier increases. For example, both lidocaine (p$K_a$ 7.9) and bupivacaine (p$K_a$ 8.1) have comparably rapid onsets (2 to 5 minutes) with subcutaneous (SQ) infiltration. However, with brachial plexus block, the onset times are prolonged, but less so for lidocaine (15 minutes) than for bupivacaine (25 minutes).

The pattern of onset for large nerves is determined by the structural arrangement of fibers. Peripheral (mantle) fibers will be blocked before core fibers. Because mantle fibers innervate more proximal regions, nerve blockade proceeds in a proximal-to-distal progression.

A faster onset time may be created by the addition of sodium bicarbonate to raise the anesthetic solution's pH, which yields a higher concentration of the uncharged lipid-soluble form more rapidly. Increasing the total dose also shortens onset time. This can be accomplished by using a higher concentration of the same volume or a greater volume with the same concentration.

For most procedures performed in the emergency department, it is unnecessary to choose a local anesthetic (or iatrogenically alter its activity) based on onset time, but one must be cognizant of the normal delays to avoid premature additional doses that might prove toxic.

**Potency**

The lipid solubility of an anesthetic is the primary physiochemical factor that determines potency. Lipid solubility refers to an agent's partition coefficient, not the concentration of lipid-soluble form that is present as determined by p$K_a$ or pH. Because the nerve membrane is basically lipid, the more lipophilic an anesthetic, the easier it can pass into the cell and the fewer the molecules that are needed for conduction blockade (see Tables 31-2 and 31-3) .

The relative degree of vasodilation produced by local anesthetics also affects potency. Lidocaine is more lipid soluble than prilocaine or mepivacaine, but it produces more vasodilation, which leads to more rapid vascular absorption. This more rapid absorption decreases the number of molecules available to penetrate the nerve. So although lidocaine is twice as potent as prilocaine or mepivacaine in vitro, it is equipotent in vivo. Another important factor is uptake by adipose tissue. Etidocaine, which in vitro is more potent than bupivacaine, is less so in vivo, because its greater absorption by fat results in fewer molecules being available for conduction blockade.

Although this is not a primary reason for its use, epinephrine, by producing vasoconstriction and making more molecules available to the nerve, increases the depth of anesthesia. Agent concentration also is important. By increasing the concentration of the anesthetic agent, potency is enhanced.

Choosing an anesthetic for its inherent potency is usually not necessary for any given site, because the concentration of an agent may be manipulated to make most drugs equianesthetic. For example, lidocaine, being one fourth as potent as bupivacaine, is usually used at 4 times the concentration (1% to 2% vs 0.25% to 0.5%, respectively). One must be cognizant, however, that for different sites and techniques, different concentrations and volumes of a given agent are needed to produce adequate blockade.

| **TABLE 31-3** -- Physiochemical Properties of Selected Local Anesthetics | | | | | |
|---|---|---|---|---|---|
| **Agent** | **Type** | **Site of Metabolism** | **p$K_a$** | **Lipid Solubility (Partition Coefficient)** | **Protein Binding (%)** |
| Tetracaine | Ester | Plasma | 8.5 | High (4.1) | 76 |

| | | | | | |
|---|---|---|---|---|---|
| Procaine | Ester | Plasma | 8.9 | Low (0.02) | 6 |
| Chloroprocaine | Ester | Plasma | 8.7 | Low (0.14) | -- |
| Lidocaine | Amide | Liver | 7.9 | Medium (2.9) | 64 |
| Mepivacaine | Amide | Liver | 7.6 | Medium (0.8) | 78 |
| Prilocaine | Amide | Liver | 7.9 | Medium (0.9) | 55 |
| Bupivacaine | Amide | Liver | 8.1 | High (27.5) | 95 |
| Etidocaine | Amide | Liver | 7.7 | High (141.0) | 94 |

**Duration**

The protein binding of an anesthetic primarily determines the duration of action. This is because agents that bind more tightly to the protein receptor remain in the sodium channel longer (see Tables 31-2 and 31-3) . Similar to potency, and for the same reason, the duration of action is inversely influenced by the vasodilation produced by local anesthetics. Prilocaine, which is less protein bound than lidocaine, in vivo produces a longer duration of action because of its lesser degree of vasoactivity. The duration of action also varies with the mode of administration, being shorter when an agent is applied topically than when it is injected into the tissues.

Physicians may *prolong the duration of action by several methods.* Increasing the dose, usually by increasing the concentration, prolongs duration but is limited by the possibility of a toxic reaction and the maximum effective concentration of a drug. Although controversial, raising the pH of the anesthetic solution also has been shown to prolong duration. [6] [7]

The most practical way to increase duration is to use solutions that contain epinephrine. [8] Epinephrine causes vasoconstriction, thereby decreasing systemic absorption, which allows more molecules to reach the nerve. The effect of epinephrine varies according to

the agent. Anesthetics that intrinsically produce more vasodilation (e.g., procaine, lidocaine, mepivacaine) benefit more from epinephrine's vasoconstrictive action. The long-acting, highly lipid-soluble agents (e.g., bupivacaine, etidocaine) are less affected because they are substantially taken up by extradural fat and released slowly. In fact, lidocaine with epinephrine may produce almost as long a duration of action as bupivacaine with epinephrine.

The selection of an agent with a long duration of action should be considered if the procedure is lengthy or if postoperative analgesia is desired. Conversely, a shorter acting agent combined with epinephrine or an alkalinized plain solution could be used. Making a specific choice of anesthetic is discussed later.

# TOPICAL ANESTHESIA

Local anesthetic agents may be applied topically to mucous membranes, skin, and lacerations. There are sufficient differences among these sites to merit a separate discussion of each. Topical anesthesia of the eye is discussed in Chapter 67 .

## Mucous Membranes

### Agents and Properties

Effective anesthesia of the intact mucous membranes of the nose, mouth, throat, tracheobronchial tree, esophagus, and genitourinary tract may be provided by several anesthetics, although tetracaine, lidocaine, and cocaine are the most effective and commonly used agents (Table 31-4) . For intraoral or pharyngeal anesthesia, benzocaine (14% to 20%) is commonly used. The anesthesia produced is superficial and does not relieve pain that originates from submucosal structures.

*Tetracaine* solution is an effective and potent topical agent with a relatively long duration of action. It may be used in concentrations from 0.25% to 1% with a recommended maximum adult dose of 50 mg. However, it has the disadvantage of high toxicity, especially the likelihood of severe cardiovascular toxicity without any preceding central nervous system stimulatory phase.

*Lidocaine* also is an effective topical agent that is marketed in a variety of forms (solutions, jelly, and ointments) and concentrations (2% to 10%). It is perhaps most familiar to emergency physicians as the 2% viscous solution prescribed for inflamed or irritated mucous membranes of the mouth and pharynx. The advantage of familiarity should not obscure the fact that misuse can lead to serious toxicity. Topical lidocaine provides an adequate duration for most procedures, with a maximum safe dose of 250 to 300 mg.

*Cocaine* is an effective, albeit potentially toxic, topical agent. Its use is limited to mucous membrane anesthesia of the upper respiratory tract. Although it is an ester, hepatic metabolism occurs, as does hydrolysis by plasma pseudocholinesterase. Cocaine is the only anesthetic that produces vasoconstriction at clinically useful concentrations. This

major advantage is offset by its susceptibility to abuse and its toxic potential. Absorption is enhanced in the presence of inflammation. Toxic effects occur as a result of both stimulating the central nervous system directly and blocking norepinephrine reuptake in the periphery. It should not be administered to patients who are sensitive to exogenous catecholamines. Clinical manifestations include central nervous system excitement, which culminates in seizures and hyperthermia plus central and peripheral effects that result in hypertension, tachycardia, and ventricular arrhythmias. Acute myocardial infarction has been reported secondary to topical application. [9] Cocaine is commonly used as a 4% solution with a maximum (although variable and controversial) safe dose of 200 mg (2 to 3 mg/kg). Coronary vasoconstriction may occur with a 2-mg/kg dose applied to the nasal

**TABLE 31-4** -- Practical Agents for Emergency Department Use--Mucosal Application

| | | Maximum Dosage * | | | |
| | | | | | |
| Agent | Usual Concentration (%) | Adult (mg) | Pediatric (mg/kg) | Onset (min) | Duration (min) |
| Tetracaine | 0.5 | 50 | 0.75 | 3-8 | 30-60 |
| Lidocaine | 2-10 | 250-300 | 3-4 | 2-5 | 15-45 |
| Cocaine | 4 | 200 | 2-3 | 2-5 | 30-45 |

* These are conservative figures; see text for explanations.
The lower dosage should be used for a maximum safe dose when feasible.

mucosa. Topical cocaine should be used cautiously, if at all, in patients with coronary artery disease.

Among other topical anesthetic agents, dyclonine and benzocaine offer distinct advantages. Dyclonine is a ketone derivative without an ester or amide linkage and

therefore may be useful in patients who are allergic to the common anesthetics. Extensive experience with the topical preparation has shown it to be effective and safe. [10] Dyclonine is marketed in 0.5% and 1.0% solutions, with a maximum adult recommended dose of 300 mg.

*Benzocaine* is an ester that is marketed only in its neutral form and is available in 14% to 20% preparations (Cetacaine, Americaine, Hurricane). Its low water solubility prevents significant penetration of the mucous membranes, making it essentially nontoxic systemically if it is applied to the *intact* mucosa. However, it is not a potent anesthetic; it has a brief duration; and compared with other topicals, it is more allergenic. Benzocaine is usually dispensed in an admixture with other therapeutic ingredients and is clinically effective only at relatively high (>14%) concentrations. Benzocaine is available as a nonprescription gel and liquid (Anbesol, 6.3% to 20%) and is commonly used topically by dentists to produce mucosal anesthesia before intraoral nerve blocks (see Chapter 33) . Adriani and Zepernick recommend this agent for lubricating catheters, airways, endotracheal tubes, and laryngoscopes. [10] In their experience with approximately 150,000 patients, only one adverse reaction (methemoglobinemia) occurred.

Similar to reactions to infiltration anesthesia, toxic reactions to topically applied anesthetics correlate with the peak blood levels that were achieved and not necessarily the dose that was administered. Systemic absorption of a topical agent is more rapid and therefore achieves a higher level than the same dose given by infiltration (Fig. 31-5) (Figure Not Available) ; therefore, the *total dose for a topical anesthetic should be considerably less than that used for infiltration at a given site*. Also, fractionating the total dose into three portions over several minutes effectively reduces peak blood levels.

An important potential adverse reaction with topical anesthesia of the nose, mouth, and pharynx is that inadvertent suppression of the gag reflex, combined with difficulty swallowing, may lead to aspiration. Infection from use of solutions from multidose vials for topical anesthesia of the larynx and trachea has not been substantiated.

**Technique and Precautions**

Emergency physicians often prescribe 2% viscous lidocaine for patients with pharyngitis, stomatitis, dental pain, or other inflammatory or irritative lesions in the oropharynx. The common misconception that topical anesthesia is innocuous may result in poor patient instruction with serious consequences. Several reports of seizures and death from topical lidocaine exist in the literature. [11] [12] Toxic blood levels may occur for several reasons: (1) the anesthetic effect of viscous lidocaine lasts only for 30 to 60 minutes, and patients with recurrent pain may either ignore or be ignorant of the safe dosing interval of 3 hours and medicate themselves more frequently; (2) patients tend to increase each dose to obtain greater relief; and (3) inflammation may increase systemic absorption. In addition, painful oral lesions may last for several days.

Continued medication use allows not only the lidocaine but also its major metabolites, monoethylglycinexylidide (MEGX) and glycinexylidide (GX), to accumulate. Both MEGX and GX are produced from hepatic metabolism of lidocaine and are excreted in the

urine. They possess anesthetic and antiarrhythmic activity and have central nervous system toxicity potential. Although these metabolites are less potent than lidocaine, their elimination half-lives, especially that of GX, are considerably longer. Several investigators regard MEGX and GX to be the causes of central nervous system toxicity with prolonged topical use. [12]

The length of time viscous lidocaine is retained in the mouth and whether the excess is expectorated or swallowed also affect the blood level produced. Expectorating the medication after swishing it in the mouth produces much lower blood levels than either swallowing it after swishing or swallowing it straight. It seems logical that the most hazardous mode of administration would be to retain the solution in the mouth "until absorbed."

Physicians must explain clearly the proper way to use viscous lidocaine and inform patients *not to dose themselves ad libitum*. The recommended maximum adult dose is 300 mg (15 mL of a 2% solution) no more frequently than every 3 hours. When possible, the patient should decrease the dose by using direct cotton swab applications. When gargled or swished in the mouth, application time should be limited to 1 to 2 minutes, and the excess solution should be expectorated. It is reasonable to limit use to 2 or 3 days, especially if swallowing the solution is necessary to obtain relief. Lower doses should be prescribed for patients who have risk factors for decreased clearance (see Systemic Toxic Reactions). Doses for children should be calculated by weight, based on 3 mg/kg. Because infants cannot expectorate well, *viscous lidocaine should not be given for minor oral irritation and teething*. Because anesthesia of the oropharynx can interfere with swallowing and cause aspiration, it is recommended that no food be eaten for 1 hour after application. Special note should be made of the over-the-counter availability of benzocaine, commonly used for toothaches and teething. A gel or liquid (Anbesol) is available in 6.3% to 20% formulations. When used repeatedly in the oral cavity on irritated tissue, significant

absorption may occur, and systemic toxicity, often in the form of methemoglobinemia, may result.

## Intact Skin

### Agents and Uses

Although stratum corneum provides a cutaneous barrier that prevents the commonly marketed aqueous solutions (acid salts) from producing anesthesia, saturated solutions of the bases of local anesthetics are effective on intact skin. Interestingly, when applied topically to *abraded* skin, most anesthesia agents result in peak blood levels similar to those resulting from infiltration (and therefore less than mucosal) in 6 to 10 minutes.

**Lidocaine cream.**

Lubens and coworkers have used 30% lidocaine cream, saturated on a gauze pad adherent to an elastic patch, for a myriad of procedures. [13] Despite its effectiveness,

safety, and painless application, the practicality of its use in an emergency setting is limited. However, Lubens and colleagues report an impressive list of uses, including minor operative procedures (e.g., excision of lesions, incision and drainage of abscesses), lumbar puncture, venipuncture, and allergy testing. [13] In many of these procedures, duration of effect need not be long, so that a shorter application time may be used.

**Tetracaine base patch and Emla cream.**

These agents have been extensively studied and appear to be effective and safe. Their activity profiles make them more applicable to emergency medicine than 30% lidocaine cream. Tetracaine base has been available for several years as a solution or gel, but only recently as a patch preparation. It is effective in crossing the lipid-rich barrier of the stratum corneum because it is highly lipophilic. Emla, which stands for *eutectic mixture of local anesthetics*, was approved in the United States in 1992. It contains 2.5% lidocaine and 2.5% prilocaine in a unique oil-and-water emulsion yielding 5% Emla. The mild lipophilic and hydrophilic properties of the component drugs are greatly increased when mixed together, allowing absorption through intact skin.

Although Emla has been more extensively studied, tetracaine base seems to offer emergency physicians the advantage of being able to obtain effective anesthesia with a shorter application time and a longer duration. [14] For both preparations, shorter onset, increased depth of anesthesia, longer duration, and higher blood levels will vary directly with more application time, use on thinner or inflamed skin, or larger dose. [15] Both preparations also exhibit a reservoir effect [16] whereby, during application, the drug is deposited in the stratum corneum and will continue to diffuse along its concentration gradient, even after it is removed from the skin.

Tetracaine base and Emla are useful in the emergency department for providing anesthesia for the following procedures: venous cannulation, venipuncture, or any needle insertion, including preinfiltration anesthesia and lumbar puncture, a variety of minor surgical procedures, and anesthetization of the tympanic membrane and external auditory canal. In addition, Emla has been used effectively for ulcer debridement. [17]

**Ethyl chloride and Fluori-Methane sprays.**

These topical agents are often used for limited skin incisions (e.g., small abscess drainage). When sprayed onto the skin, these agents evaporate with cooling to the point of freezing. Anesthesia is effective and immediate, but drawbacks include its short duration (only up to 1 minute), potential pain on thawing, and possible lowered resistance to infection and delayed healing. Ethyl chloride spray is highly volatile and should not be used around flames or sparks (e.g., electrical cautery).

## Technique

**Lidocaine cream.**

This 30% cream is saturated on a gauze pad adherent to an elastic patch and placed over the area to be injected or incised during the procedure. [13] Both the high concentration of anesthetic and an occlusive patch are necessary to achieve effective skin penetration. The duration of action varies with the application time. For most procedures, a 45-minute application time is minimum; to achieve a topical anesthetic duration of ½ hour, a 2-hour application is necessary.

**Tetracaine base patch and Emla cream.**

Both agents demonstrate a reservoir effect. Depending on the application time, the physician may see the anesthetic effect increase, or even first begin, many minutes after drug removal. Because of the factors just mentioned, it is not easy to be exact concerning application times and duration. Nevertheless, tetracaine base requires a minimum of 20 to 30 minutes' application time and will produce a several hour duration. Emla requires an application time of 1 to 2 hours for a reported duration of 30 minutes to several hours. Occlusive dressings seem to increase Emla penetration whenever the cream is used. For either drug, using patches is more convenient and causes no loss of effectiveness.

Emla dosage is based on the amount of cream applied, not on the amount of anesthetic. Each gram of Emla cream contains 25 mg of lidocaine and 25 mg of prilocaine. Dosages are given in *grams of cream*, not milligrams of anesthetic. In general, Emla is applied as a thick layer to intact skin under an occlusive dressing for about 1 hour before a procedure. A thick layer approximates 1 to 2 g applied per 10 cm2 . For minor procedures such as needle insertions, apply 2.5 g of Emla over 20 to 25 cm2 for at least 1 hour. When starting IV lines, it may be helpful to prepare 2 sites in case of technical difficulties at the first site. For more painful procedures, apply about 2 g of cream per 10 cm2 for at least 2 hours. The appropriate total dose applied also is determined by the maximum application area (MAA). The MAA is based on the patient's weight as follows: up to 10 kg, MAA = 100 cm2 ; 10 to 20 kg, MAA = 600 cm2 ; over 20 kg, MAA = 2000 cm2 .

**Ethyl chloride and Fluori-Methane sprays.**

The technique of application is that the inverted bottle is held 25 cm from the skin, and a stream of spray is directed along the proposed incision until the area turns white and hard. An incision must be made immediately, because the effect may be fleeting. Some clinicians use these vapocoolant sprays to decrease the pain of an injection of a more traditional local anesthetic such as lidocaine.

**Adverse Events**

General adverse reactions to anesthetic agents are discussed in the Complications section of this chapter.

Tetracaine base is quite safe. Blood concentration of tetracaine is very low after proper

use. In 1 study, [16] approximately 25% of the patients developed cutaneous erythema

at the site of application. This vasodilatory effect may actually be an advantage when starting IV lines or performing venipuncture.

Emla is also quite safe. Although there is a high rate of local skin reactions, these are mild and transient, disappearing 1 to 2 hours after cream removal. Despite the reported successful use of Emla cream on skin ulcers, [17] Powell and colleagues demonstrated an increase in bacterial growth and infection and inflammation rates when used in experimental wounds. [18] The main concern with Emla is methemoglobinemia resulting from the metabolites of prilocaine. Despite occasional reports of this problem, which appears to be due to overdose or other risk factors, [15] the risk of clinically significant methemoglobinemia seems remote when Emla is used properly. It is contraindicated in any infant <3 months of age and in those infants between 3 and 12 months of age who are currently taking methemoglobinemia-inducing drugs (nitrites, sulfonamides, antimalarials, phenobarbital, and acetaminophen). The risk of inducing adverse effects also is increased in patients with anemia, respiratory or cardiovascular disease, glucose-6-phosphate dehydrogenase (G-6-PD) or methemoglobin reductase deficiencies.

Prolonged inhalation of ethyl chloride spray may produce general anesthesia, coma, or cardiorespiratory arrest. Ethyl chloride also is flammable, which precludes its use with electrocautery.

## Lacerations

In 1980, Pryor and colleagues reported their experience with a topical anesthetic solution (tetracaine-Adrenalin-cocaine [TAC]) prior to wound repair. [19] Thus began ongoing controversy concerning the efficacy and safety of this compound. The original formula, used in most subsequent studies, consists of a solution of 0.5% tetracaine, 1:2000 epinephrine (Adrenalin), and 11.8% cocaine. Traditionally, anesthesia is produced by firmly applying a solution-saturated gauze pad or cotton ball directly to the laceration for 10 minutes; the resulting loss of cutaneous sensation is centered about the area of application. The recent advent of a gel formulation for TAC and alternative mixtures of agents promise to improve the ease and safety of topical anesthetic solutions for wound repair in the emergency department.

### Indications and Contraindications

The use of TAC (and related topical wound anesthetics) is generally restricted to young children with wounds <5 cm in length in whom the delay for anesthetic application is acceptable and proper application can be assured. Because of the vasoconstrictor agents (epinephrine and cocaine) in TAC, TAC is generally not used in structures without collateral blood supply (e.g., the digits, the tip of the nose, the pinna of the ear, the penis).

Compared with infiltrative anesthesia, the advantages of TAC include painless

application, no distortion of wound margins, good hemostasis, and good patient and parental acceptance in the pediatric age group. As compelling as the case for TAC may appear, some disadvantages have made its acceptance less than unanimous. TAC appears to be less effective on the trunk and extremities than on the face and scalp and less effective than lidocaine infiltration in these areas. There also is a 10- to 20-minute onset time, [20] which may be a drawback in a busy emergency department. The cocaine component presents many drawbacks, including cost and federal regulations that require storage in a locked cabinet and maintenance of separate written records for 2 years after the drug is dispensed.

Another concern with TAC is that some emergency departments use wound care protocols that advocate wound preparation be done before anesthetic application. Hence, wound preparation would likely be painful and therefore incomplete. Proper cleansing of traumatic injuries and surgical debridement cannot be accomplished without some form of local anesthesia. One approach is to remove large debris and clotted blood to allow for the appropriate application of TAC and then finish wound prep once the wound is properly anesthetized.

Two other oft-mentioned drawbacks may be more theoretical than real. Vasoconstrictor-induced higher infection rates (see Complications) have not been borne out clinically. Also, the argument that the necessary 10-minute application period is time consuming and takes valuable nursing time is partially offset by using the child's caretaker or adhesive paper tape alone (in the older, reliable patient) to hold the solution in place. With the use of gel, it is not necessary for anyone to "hold" the medicine in place, although the application time is still required.

**Agents and Effectiveness**

Three clinical trials directly compared TAC with infiltrative lidocaine. Without specifying wound location, Pryor's group found equal anesthetic effect. [19] Complete anesthesia produced by TAC ranged from 82% to 86%, compared with 83% to 92% for SQ lidocaine. The remaining patients obtained partial anesthesia. Hegenbarth and coworkers and Anderson and associates demonstrated results similar to those of Pryor and colleagues. [21] [22] Furthermore, Hegenbarth and colleagues found TAC to be equal to lidocaine only on the face and scalp, and inferior to it at other locations. Other investigators using different study designs found TAC effectiveness to be equal to or slightly less than seen in earlier studies. More recent studies confirm excellent rates of effectiveness, especially on the face and scalp. [26] [27]

Concerned about potential toxic effects and theoretic vasoconstrictor-induced higher infection rates, several investigators compared TAC with its various components. On the face and scalp, TAC was found to be superior to tetracaine alone, although on nonfacial areas, both produced equally poor results. [23] TAC was found more effective than cocaine alone [25] and more effective than a tetracaine-epinephrine solution in the same dosage ratio. [28]

In 1990, Bonadio and Wagner showed that an epinephrine-cocaine solution (epinephrine 1:2000, cocaine 11.8%) was equal to TAC in effectiveness. [26] Bonadio

and Wagner also found that half-strength TAC (tetracaine 0.25%, epinephrine 1:4000, cocaine 5.9%) achieved excellent results for dermal lacerations of the face, lip, and scalp. [24] Smith and Barry, when comparing three strengths of TAC, found equal effectiveness among them and recommended the lowest strength cocaine formulation (tetracaine 1.0%, epinephrine 1:4000, cocaine 4.0%). [29] Most recently, Schilling and colleagues found that a lidocaine-epinephrine-tetracaine solution (lidocaine 4.0%, epinephrine 1:1000, tetracaine 0.5%) was as effective as TAC. [30] Ernst and colleagues found similar effectiveness to TAC using a slightly different lidocaine-epinephrine-tetracaine solution (lidocaine 4.0%, epinephrine 1:2000,

tetracaine 1.0%). [31] The non-cocaine-containing formulations are generally considered less toxic and have advantages in terms of reduced cost and avoidance of controlled substance precautions during storage.

**Application and Dosage**

Because the topical mixtures noted above (especially TAC) are not innocuous anesthetics, attention must be paid to the technique of application and recommended maximum dose. Unfortunately, there is no uniformly accepted application technique, component composition, or component concentrations.

Generally, topical solutions such as TAC are applied as follows: The wound is placed in a gravity-dependent position, and TAC is carefully instilled to fill the wound cavity; after 3 minutes, a single 2-cm × 2-cm gauze pad or cotton ball saturated with TAC is applied to the wound; the pad should be taped, and the caretaker or nursing personnel should hold it firmly for 15 to 20 minutes. The person holding the gauze should wear latex examination gloves to minimize the risk of cutaneous absorption of the solution. The average dose of TAC solution needed is 2 mL.

Based on the known maximal safe dose of infiltrative tetracaine and mucosal application of cocaine and an estimate of solution absorption onto the applicator, Hegenbarth and colleagues estimated the maximum safe dose of full-strength TAC to be 0.09 mL/kg. [21] Other useful estimates for maximum topical doses are tetracaine, 50 mg, and cocaine, 150 to 300 mg. The key to safety is to *avoid TAC on mucosal surfaces or in areas in which sniffing or swallowing may accidentally occur*. Topical mucosal anesthesia is discussed elsewhere in this chapter.

One can prepare an epinephrine-cocaine gel by adding 0.15 g of methylcellulose to 1.5 mL of epinephrine-cocaine solution. The mixture is stirred thoroughly for a minute or two until a gel consistency is obtained. The gel is applied as follows: After sterile preparation, the wound is placed in a gravity-dependent position, and the gel is applied by a cotton-tipped swab to coat the entire wound cavity and margins. The wound is allowed to stand for 15 to 20 minutes and then is thoroughly washed to remove the gel from the wound cavity. In Bonadio and Wagner's study, the average dose used was 0.35 mL of gel containing only 40 mg of cocaine. [27]

**Adverse Events**

Mucosal application may lead to significant systemic toxicity, and fatalities have been reported following this application mode. Even after nonmucosal TAC use, cocaine levels appear in the blood (tetracaine does not) and cocaine metabolites appear in the urine in the majority of patients, [32] [33] although these levels are generally not clinically significant.

Gel formulations of TAC tend to stay in the wound and reduce the risk of solution runoff onto mucosal surfaces. There is no need to use a gauze pad to apply the medication in gel formulation or to hold it in place. Since the entire applied dose will stay in the wound, with little absorption, when an overlying gauze is incorporated, a lower dose generally can be used. Gel also provides a more uniform application to tissues improving anesthetic effectiveness.

Ischemic complications from applying vasoconstrictors to tissues containing end arteries may occur. Therefore, TAC should be avoided on the digits, the tip of the nose, the penis, and the pinna. TAC should be used with caution, if at all, in patients with coronary artery disease, uncontrolled hypertension, seizures, and peripheral vascular disease. Any patient with a decreased plasma cholinesterase level is at increased risk for systemic toxic effects (see Complications).

# INFILTRATION ANESTHESIA

Infiltration anesthesia involves injection of an anesthetic agent directly into the tissue prior to surgical manipulation. Field block anesthesia also may be considered a form of infiltration anesthesia, particularly because the useful agents, concentrations, and recommended maximum dosages are the same. A field block involves an SQ injection of an agent in such a manner as to create a field of anesthesia around the operative site. The injection is made proximal to or surrounding the area to be manipulated.

**Indications and Contraindications**

Infiltration anesthesia is indicated whenever good operative conditions can be obtained by using this technique. It may be used for the majority of minor surgical procedures such as excision of skin lesions, incision of abscesses, and suturing of wounds. An advantage of infiltration over nerve block and general anesthesia is that it is considered both quicker and safer. Local infiltration also can provide hemostasis, both by direct distention of tissue and by the concurrent use of epinephrine.

The major disadvantage of local infiltration, when compared with nerve blocks, is that a relatively large dose of drug is needed to anesthetize a relatively small area. For extensive wounds, the amount of anesthetic required may risk systemic toxicity. However, the maximum allowable volume can be increased by adding epinephrine, by using a lower concentration of anesthetic agent, or by doing both (Table 31-5) . When large volumes are anticipated and a nerve block is anatomically feasible, the nerve

block may be preferred. Infiltration should be avoided for large procedures in small children and in apprehensive patients, especially those with prior adverse reactions (whether vasovagal or otherwise). Local infiltration also distorts the tissues that will be incised or repaired and may not be desirable in areas requiring precise alignment (e.g., some lip repairs).

## Choice of Agent

Local anesthetic agents most frequently used for infiltration are 0.5% to 1.0% lidocaine, 0.5 to 1.0% procaine, and

| TABLE 31-5 -- Maximum Allowable Volume (Adults) | | | |
|---|---|---|---|
| **Agent** | **Concentration (%)** | **Maximum * Safe Dose (mg)** | **Maximum Volume (mL)** |
| Lidocaine | 0.5 | 300 | 60 |
| | 1.0 | 300 | 30 |
| Bupivacaine | 0.25 | 175 | 70 |
| Lidocaine-epinephrine | 0.5 | 500 | 100 |
| | 1.0 | 500 | 50 |
| Bupivacaine-epinephrine | 0.25 | 225 | 90 |

* Some physicians recommend 400 mg as the maximum safe dose for bupivacaine.

**TABLE 31-6** -- Practical Agents for Emergency Department Use--Local Infiltration

| Agent | Concentration (%) | Maximum Dose * | | Onset (min) | Duration |
| --- | --- | --- | --- | --- | --- |
| | | Adult (mg) | Pediatric (mg/kg) | | |
| Procaine | 0.5-1.0 | 500 § (600) | 7.0 (9) | 2-5 | 15-45 min |
| Lidocaine | 0.5-1.0 | 300 (500) | 4.5 (7) ‖ | 2-5 | 1-2 hr |
| Bupivacaine | 0.25 | 175 (225) | 2.0 (3) ¶ | 2-5 | 4-8 hr |

\* These are conservative figures; see text for explanation.
Higher dose for solutions containing epinephrine is in parentheses.
These values are for the agent alone; they can be extended considerably with the addition of epinephrine.
§ Some authorities recommend up to 1000 mg or 14 mg/kg for procaine.
‖ Some authorities recommend up to 7 mg/kg for plain lidocaine in children older than 1 year.
¶ Because of lack of clinical trial experience, drug companies do not recommend the use of bupivacaine in children under the age of 12 years.

0.25% bupivacaine (Table 31-6) . Lidocaine has been the agent most commonly used because of its excellent activity profile, low allergenicity and toxicity, user familiarity, and ready availability. Procaine is useful for patients who are allergic to amide anesthetics. Because of its prolonged duration, bupivacaine is considered by some physicians to be the preferred anesthetic, especially when postoperative analgesia is desired, for prolonged procedures, or even for short procedures that may be interrupted in a busy emergency department.

A comparison of equianesthetic doses of lidocaine and bupivacaine for infiltration anesthesia (Table 31-7) reveals that the duration of action is the major difference between the 2 agents. For the majority of emergency department procedures, it is not necessary to extend the duration of anesthesia beyond 1 hour. Plain lidocaine would therefore seem to be a logical anesthesia choice. However, after laceration repair, patients experience a moderate amount of pain when the lidocaine wears off in about 1 hour. [34] Bupivacaine was demonstrated to reduce this pain following laceration repair for at least 6 hours. This benefit of a prolonged duration of anesthesia must be weighed against the hazards of injury to an unprotected limb or the annoyance of prolonged

numbness to patients who have had simple surgical procedures.

A prolonged duration of anesthesia can be achieved by adding epinephrine, sodium bicarbonate, or both to lidocaine (see Duration) or by using bupivacaine. Advantages of epinephrine are that it provides excellent wound hemostasis and slows systemic absorption. This latter property decreases the peak blood level, thereby decreasing the potential for a toxic reaction, or, conversely, it allows a greater volume of agent to be used for extensive lacerations. The major disadvantage of epinephrine is the theoretical damage to host defenses (Table 31-8) . Adding bicarbonate decreases the pain of administration, but such an addition usually entails bedside preparation (see subsequent discussion). Bupivacaine, if used with due caution, is safe and easy to use. The deciding factors are many, but some logical choices are as follows:

> For a wound with excessive bleeding: lidocaine with epinephrine
>
> For an apprehensive patient: lidocaine with sodium bicarbonate
>
> For anticipated prolonged postprocedure pain: bupivacaine.

## Equipment

The pain of injection can be reduced by use of small-gauge needles. Ideally, a 30-ga needle should be used if injection is made through the skin. If the injection is made through the cut edges of the wound, a 25- to 27-ga needle should suffice. A small-gauge needle also helps slow the rate of injection and reduce the rate of tissue distention. A 10-mL syringe is recommended both for its ease of handling and for the relatively slow rate of injection it allows.

## Technique

Once an agent has been chosen, proper technique of administration should be used to minimize pain, prevent bacterial spread, and avoid intravascular injection.

| TABLE 31-7 -- Comparison of 1% Lidocaine (L) and 0.25% Bupivacaine (B)--Infiltration Anesthesia | | | |
|---|---|---|---|
| | **Lidocaine** | **Bupivacaine** | **Advantage** |

| | | | |
|---|---|---|---|
| Onset | 2-5 min | 2-5 min | Equal |
| Effectiveness (equianesthetic dose) | Excellent | Excellent | Equal |
| Duration | 1-2 hr | 4-6 hr | B |
| Infection potential | No | No | Equal |
| Administration pain | Less | More | L |
| Maximum volume * --plain lidocaine | Less | More | B |
| Maximum volume--epinephrine | Less | More | B |
| Toxic potential | Less cardiotoxic; equal CNS | More cardiotoxic; equal CNS | L |

CNS, central nervous system.

* See Table 31-5 for volume and concentration comparison.

| TABLE 31-8 -- Epinephrine Use | |
|---|---|
| **Advantages** | **Disadvantages** |

| | |
|---|---|
| 1. Prolongs duration | 1. Impairs host defenses--increases infection [*] |
| 2. Provides hemostasis | 2. Delays wound healing [*] |
| 3. Slows absorption:<br>  Decreases agent toxi potential<br>  Allows increased dose | 3. Do not use for:<br>  Areas supplied by end arteries<br>  Patients"sensitive" to catecholamines |
| 4. Increases level of blockade | 4. Toxicity--catecholamine reaction |

* Based on laboratory studies.
For example, in patients taking MAO inhibitors.

**Buffering**

For a given anesthetic agent, lowering the pH by adding epinephrine increases pain, whereas raising the pH by adding sodium bicarbonate decreases pain dramatically. However, it is probable that pH per se is not the sole factor. It has been shown that the pain produced by various agents does not correlate strictly with the pH. For instance, although bupivacaine (pH 5.5) is more painful than lidocaine (pH 6.5), chloroprocaine (pH 3.4) is less painful than bupivacaine, and procaine (pH 4.3) is less painful than lidocaine. Sodium bicarbonate probably works by increasing the ratio of nonionized to ionized molecules, which either renders the pain receptors less sensitive or causes a more rapid diffusion of solution into the nerve and hence a shorter time to anesthetic onset.

To alkalinize lidocaine, 1 mL of sodium bicarbonate (8.4%, 1 mmol/mL) is added to every 10 mL of anesthetic solution. Unfortunately, as the pH of the solution is raised, the anesthetic becomes unstable and has a decreased shelf life. Therefore, it was initially recommended that buffered lidocaine be prepared just prior to use to avoid precipitation and degradation. However, buffered lidocaine retains its effectiveness for 1 week, and refrigeration may further increase its shelf life. [35] [36] Bicarbonate may be combined with plain lidocaine for both infiltrative anesthesia [37] [38] and digital nerve blocks. [39] In one volunteer study, sodium bicarbonate also was combined with lidocaine with epinephrine, with good effect. [36]

Sodium bicarbonate also can be added to bupivacaine, but the tendency for the solution to precipitate is great as the pH rises. Precipitation also directly varies with the concentration of bupivacaine and the time since mixture. Cheney and colleagues showed that 0.05 mL of 8.4% sodium bicarbonate (measured in a tuberculin syringe)

could be mixed with 10 mL of 0.5% bupivacaine without precipitation. [40] However, extrapolating data from another study, [41] this solution would need to be used within 5 minutes to avoid precipitation. Since the advantage of using bupivacaine is to prolong the duration of anesthesia, and this effect also can be accomplished somewhat by using buffered lidocaine (plain or with epinephrine), it seems impractical at this time to risk precipitation by "buffering" bupivacaine.

### Warming

Warming an anesthetic to body temperature (37 to 42 °C) reduces the pain of infiltration. [42] [43] However, Bartfield and colleagues, using lidocaine warmed to 38.9 °C, found that the warmed lidocaine was somewhat more painful than room temperature buffered lidocaine during intradermal injection. [44] Interestingly, Brogan and colleagues, using lidocaine warmed to 37 °C, found the warmed lidocaine and room-temperature buffered lidocaine to be equivalent during wound infiltration. [45] Neither study found a synergistic effect with combined warming and buffering. Martin and associates also found warmed (37 °C) lidocaine to be no less painful than buffered lidocaine. [46] Anesthetic solutions can be warmed in a baby-food warmer with thermostatic temperature control or in an IV solution warmer. Warming is not believed to adversely affect shelf life of the local anesthetic.

### Injection

Placement of the injection should be subdermal to minimize needle puncture pain and the tissue distention that occurs with intradermal placement. Placing the needle "up to the hub" and injecting while withdrawing along the just-created subdermal tunnel also minimizes distention. After an initial injection, rather than totally withdrawing the needle from the tissue, the needle may be redirected along another path to lessen the number of punctures. Slowly injecting the smallest volume necessary also helps to reduce pain.

Bierman described a technique of patient distraction by applying light pressure to alternate sides of the wound with one's fingers and repeated ambiguous questioning about feeling the light pressure rather than the ongoing wound injection. [47] A school-age child can be asked to count backward or say the "ABCs" as a form of distraction during injection.

Because the patient barely feels a needle placed subcutaneously and skin puncture is often quite painful, ideally, all *wound injections should be made through the wound edge and not via a skin puncture* (Fig. 31-6) . [48] Concern about spreading infection by passing a needle through a fresh wound edge has not been substantiated clinically. However, in the case of a grossly contaminated wound, some clinicians may choose to inject the anesthetic through intact skin.

Preventing a systemic toxic reaction is best accomplished by avoiding an intravascular injection. However, for infiltration anesthesia with small-gauge needles, aspiration is usually unnecessary unless the injection is deeper than the SQ area or the area to be

injected contains many large vessels.

## SPECIAL CONSIDERATIONS

There are many applications of infiltration anesthesia in the emergency department, but the following special cases deserve separate mention.

### Hematoma Block

Hematoma block has been used for many years to provide anesthesia for reduction of fractures, particularly of the distal forearm and hand. Its popularity has waned somewhat because of the fear of introducing infection at the fracture site and its limited efficacy. Although several studies show the hematoma block to be safe, the anesthesia it provides is not as good as that provided by the Bier block. [49] [50] However, there are several reasons to retain this technique. The procedure is simple and quick to perform and does not require additional personnel. There is no need to wait for an anesthesiologist or for the patient to digest a meal, as when general anesthesia is used. A lower dose of anesthetic agent is required compared with that required for the Bier block (see Chapter 34) . Lastly, it is useful when the Bier block and general anesthesia are contraindicated.

This simple technique involves preparing the skin over the fracture site, inserting the needle into the hematoma (confirmed by aspirating blood), and slowly injecting from 5 to 15 mL of plain 1% lidocaine or 5 to 10 mL of plain 2% lidocaine, depending on the fracture site, into the fracture cavity and around the adjacent periosteum. Onset of adequate anesthesia occurs in about 5 minutes and may last for several hours. This procedure should not be performed through dirty skin, into open fractures, or in small children.

### Intra-articular Anesthesia

The history and physical examination of an acutely traumatized knee may underestimate the severity of an injury. Instillation of 5 mL of 1% lidocaine after joint aspiration may help to relieve pain and facilitate examination, but its use is not routinely recommended. [51] Spasm and apprehension are often not relieved by local anesthesia, and the occasional knowledge gained usually does not influence the treatment plan. Intra-articular anesthesia of the knee has no effect on gait pattern or joint proprioception. [52] Therefore, if otherwise indicated, postprocedure weight bearing may be allowed without fear of producing or increasing injury. Intra-articular anesthesia may enhance elbow use following aspiration of a hemarthrosis associated with a radial head fracture. [53] The technique of administration is analogous to arthrocentesis (see Chapter 57) .

### Intrapleural Anesthesia

**Indications**

Intrapleural anesthesia is a relatively new procedure with applicability to emergency medicine. This technique introduces local anesthetic into the pleural space (i.e., between the parietal and visceral pleura) through an epidural catheter. The anesthetic also can be introduced through a previously placed chest tube. The technique has been used to provide relief for several conditions, primarily postthoracotomy pain; postcholecystectomy pain; and, most importantly for emergency

physicians, posttraumatic chest pain (i.e., rib fractures, pneumothorax, hemothorax). This procedure is useful not only for pain relief, but also for allowing the patient to turn, cough, and deep breathe, thereby helping to prevent atelectasis and pneumonia.

Although not unanimous, most studies show that intrapleural anesthesia is effective in providing analgesia. [54] [55] Possibly correctable reasons thought responsible for less than satisfactory results in some studies include dilution of anesthetic by blood in the pleural cavity, inadequate dosing, malposition of the catheter tip, malposition of the patient after injection, and loss of anesthetic through a previously placed chest tube.

Concern has been raised that intrapleural anesthesia may create a level of anesthesia below the umbilicus and make posttraumatic abdominal examinations unreliable. Until this issue is clarified, it seems prudent to rule out intra-abdominal injury before intrapleural anesthesia is used. [56]

**Technique**

If a chest tube is in place, it is preferable to inject anesthesia into the pleural space via the chest tube. Theoretically, the tube should be clamped for 10 to 15 minutes to allow the anesthetic to diffuse. When the tube cannot be taken off suction, or if no tube is present, the local anesthetic is injected percutaneously. The percutaneous method as described by Stromskag and colleagues is presented. [57] With the patient in the lateral position (with the affected side up), a 16-ga Tuohy needle is placed 8 to 10 cm from the posterior midline in the 8th intercostal space. The needle is angled at 30° to 40° to the skin, aimed medially, bevelled upward, and directed just above the rib. After perforating the posterior intercostal membrane (felt as a distinct resistance), the stylet is removed, and a well-wetted, air-filled glass syringe is attached to the Tuohy needle. The needle is advanced until it enters the pleural space, which is denoted by the plunger being drawn down the syringe due to the negative pressure created during inspiration. The syringe is then removed, and an epidural catheter is introduced 5 to 6 cm into the pleural space. The Tuohy needle is removed, a chest radiograph is taken to confirm proper position, and the catheter is secured.

The most commonly used anesthetic and dose is 20 mL of bupivacaine 0.5%. A repeat dose every 8 hours has been shown safe. [58] What position the patient should be in during or after the injection of anesthetic or whether the chest tube, if one is in place, should be clamped, taken off suction, elevated, or left alone are questions not yet fully answered.

The solution presumably diffuses from the pleural space "back" through the parietal pleura and the intercostal muscle to reach the intercostal spaces, where it blocks the intercostal nerves. The level of anesthesia can extend from T2 to T12 and involve skin, chest and abdominal wall, and, potentially, the viscera if the visceral afferent fibers are blocked at the sympathetic chain in the paravertebral gutter.

Although not yet a consistently proven or a completely standardized technique, intrapleural anesthesia offers promise for patients and is a potentially valuable procedure for the emergency physician.

### Xiphodynia

Patients may complain of epigastric or lower thoracic discomfort which is completely or nearly completely reproduced with light xiphoid pressure. This painful condition may be preceded by prolonged coughing, retching, or muscular exertion. Local infiltration of the tender area with 5 to 7 mL of local anesthetic solution (1% lidocaine or 0.5% bupivacaine) using a 25- or 27-ga needle will generally produce considerable improvement in the condition. [59] Some clinicians incorporate a steroid agent with the anesthetic (e.g., 80 mg triamcinolone) for a protracted anti-inflammatory effect. The xiphoid should be easily palpable and generally lies less than 2 cm below the skin surface. Should deeper penetration be required or the patient complain of new pain or respiratory distress following the procedure, an upright chest radiograph is recommended to rule out an inadvertent pneumothorax. Diagnostic injection of the xiphoid process should follow standard clinical evaluation for chest and abdominal discomfort.

## COMPLICATIONS

### Local Anesthetic Effect on Wounds

In addition to producing neural blockade, local anesthetics also have potential effects on wound healing and wound infection.

#### Wound Healing

Local anesthetics produce cytotoxic effects on cell structure and function in a dose- and time-related manner. These effects, demonstrated at doses well below those used clinically, involve fibroblasts more than nervous tissue. Collagen synthesis is inhibited by lidocaine and bupivacaine. [60] Morris and Tracey found that lidocaine in increasing concentrations progressively reduced the tensile strength of wounds. [61] Epinephrine added to 1% and 2% concentrations of lidocaine further reduced tensile strength, but when epinephrine was added to distilled water or to 0.5% lidocaine, it had little effect. Several conclusions appear to be clinically relevant:

Although it may have a delayed anesthetic onset, lidocaine 0.5% solution, without

epinephrine if possible, may be best for wound strength.

When possible, local anesthetics should be avoided in poorly healing wounds, e.g., leg ulcers, or where fast healing is essential.

Local anesthetics in high concentrations might help prevent excessive scarring in patients prone to keloids or hypertrophic scars.

Eriksson and colleagues found that lidocaine reduces the inflammatory response in wounds by decreasing the number of white cells and their metabolic activity. [62] While an inflammatory response may be beneficial in a contaminated wound, it can be detrimental in a sterile wound because of the tissue toxicity that is created by the release of superoxide anions, lysosomal enzymes, thromboxanes, leukotrienes, and interleukins.

**Wound Infection**

Although not generally appreciated, it has long been known that local anesthetics possess antimicrobial activity in vitro. Lidocaine and procaine at 2% concentrations--and, to a lesser extent, at lower concentrations--have been shown to inhibit the culture growth of most gram-negative organisms, with only *Pseudomonas aeruginosa* being particularly resistant.

[63] Gram-positive isolates also were significantly affected by lidocaine and, to a lesser extent, by procaine. A more recent study [64] supports this contention by showing excellent inhibition of bacterial growth by lidocaine 2% and bupivacaine 0.5%, but much less inhibition as anesthetic concentration is lowered to lidocaine 1% and bupivacaine 0.125%. Administering anesthetics before obtaining culture material, including injecting a joint prior to arthrocentesis, may give false-negative culture results and should be avoided if possible. This effect also may be significant in obtaining wound cultures when anesthetic ointments have been previously applied. Furthermore, it has been shown [65] that adding sodium bicarbonate to lidocaine greatly enhances its inhibitory effect on bacteria. Although local anesthetics can interfere with culture testing, several studies show that, by themselves, local anesthetics do not appear to alter the incidence of wound infection.

Epinephrine appears to exert a deleterious effect on host defenses, at least in animal models. Studies with infiltrative and topically applied epinephrine solutions in iatrogenically contaminated animal wounds show an increased potential for infection. [67] [68] Possibly, epinephrine-induced vasoconstriction contributes to tissue hypoxia, which has been shown to retard the killing of *Staphylococcus aureus* by leukocytes, and to a reduction in leukocyte migration into the tissue. [69] Although it has been recommended that solutions containing epinephrine not be used because of the potential enhancement of wound infection, most *clinical* studies using topical anesthesia with vasoconstrictor properties (e.g., tetracaine, epinephrine, and cocaine mixtures) have not demonstrated significantly increased infection rates.

## Local Injuries

Injuries may rarely result from direct application of an anesthetic agent to a nerve or from passage of a needle through soft tissue structures. However, this theoretical concern should not deter the physician from the routine use of local anesthetics for accepted conditions. Factors implicated in transient or persistent neuropathy include acidic solutions, additives, the agent itself, needle trauma, compression from hematomas, and inadvertent injection of neurolytic agents. For example, Born described a series of 49 wrist and metacarpal blocks using bupivacaine in which 8 patients developed a significant neuropathy. [70] He postulated that damage occurred from the trapping of the drug in a confined space and recommended that whenever bupivacaine is used in this situation, it be used in low concentration and volume. Infection, hematomas, and broken needles are other local problems that can be averted by using proper technique. Erroneous needle placement also can produce complications such as pneumothorax during brachial plexus or intercostal block.

It is commonly stated that epinephrine-containing solutions, when injected into tissues containing end arteries, can lead to profound ischemia and gangrene. Areas of special concern include the digits, penis, tip of the nose, or earlobe. This concern is real but is probably overrated when dilute concentrations of epinephrine-containing solutions are used (1:200,000). Some authors state that small quantities of such solutions are usually tolerated on the nose and pinna but advise against their use in the fingertips. [71] Because bupivacaine can be used when prolonged anesthesia is required, and because tourniquets can be used in the digits, there is little need for vasoconstrictors in the digits, although some clinicians use a dilute epinephrine solution there as well (see Chapter 32) . The use of phentolamine (Regitine), which produces postsynaptic alpha-adrenergic blockade, is recommended for vasoconstrictor-induced tissue ischemia. This medication is usually given by local infiltration in a dose of 0.5 to 5.0 mg diluted 1:1 with saline. If local infiltration is ineffective or limited by tension within a tissue compartment, or if the area of vasoconstriction is large, phentolamine may be given by the intra-arterial route. [72]

## Systemic Toxic Reactions

Although they occur in only 0.1% to 0.4% of local anesthetic administrations, systemic toxic reactions are the most frequent serious adverse reactions encountered (Table 31-9) . [73] On administration of a local anesthetic, some of the drug reaches its intended target, and some is absorbed quickly into the systemic circulation. Peak blood levels are generally produced within 30 minutes, with levels then falling because of distribution and elimination.

### High Blood Levels

Systemic toxic reactions generally result from high blood levels of local anesthetic agents. Several factors are important in producing high blood levels, including site and mode of administration, rate, dose and concentration, addition of epinephrine, specific

drug, clearance, maximum safe dosage, and inadvertent intravascular injection.

**Site and mode of administration.**

Comparing the modes of administration for a given dose, the intravascular route produces the highest levels, followed by topical mucosal application, then infiltration (see Fig. 31-5) (Figure Not Available) . Within each mode, the more vascular the site, the more systemic absorption that occurs and the higher the level obtained. The following blocks are arranged in decreasing order of systemic absorption: intercostal, caudal, epidural, brachial plexus, SQ. Therefore, it follows that the site of administration is an important variable in determining the safe dose of an anesthetic. For example, lidocaine 400 mg may produce a nontoxic blood level with abdominal wall SQ infiltration, but when used for an intercostal nerve block, a toxic level would likely result from this dose.

**Rate.**

A more rapid IV injection will produce a higher blood level than a slower injection. Similarly, a single topical application leads to a higher level than a dose that is fractionated over time.

**Dose and concentration.**

The larger the total dose, the higher the peak blood level. It is uncertain whether increasing the concentration while maintaining the total dose by decreasing the volume affects the serum level.

**Addition of epinephrine.**

When infiltrated, epinephrine produces vasoconstriction and reduces systemic absorption, thereby resulting in lower peak blood levels.

**Specific drug.**

When comparing local anesthetics on a milligram-to-milligram basis, the more potent agents are more toxic. Because anesthetics are used in equipotent doses (e.g., 1 mg bupivacaine versus 4 mg lidocaine), they are approximately equitoxic. Blood levels achieved by a particular agent depend on the agent's absorption into, and its distribution and clearance from, the circulation. Agents with high lipid solubility and lower protein binding (etidocaine > bupivacaine > lidocaine > mepivacaine) tend to become

| | | | | **TABLE 31-9** -- Differentiating Systemic Adverse Reactions | | | |
|---|---|---|---|---|

| Findings | Toxic Reactions | Allergy | Vasovagal | Excess Catecholamines, Anxiety *1 (Endogenous), Vasoconstrictor (Exogenous) |
|---|---|---|---|---|
| *Relatively specific signs and symptoms* | Metallic taste Tongue numbness Drowsiness Nystagmus Slurred speech Seizures * Coma Respiratory arrest * | Acute rhinitis Pruritus * Dermatitis Urticaria * Facial swelling Laryngospasm Bronchospasm * | Syncope *2 | Headache Hypertension * Palpitations Apprehension *3 |
| *Overlapping signs and symptoms* | Paresthesia Lightheadedness Tinnitus Tremor Tachypnea Tachycardia (early) Bradycardia * Hypotension * Cardiac arrest | Lightheadedness Tachycardia * Hypotension * Cardiac arrest Nausea and vomiting Dyspnea | Lightheadedness Tinnitus Tachypnea Tachycardia (early) Bradycardia * Hypotension * Diaphoresis | Paresthesia * Lightheadedness * Tremor Tachypnea * Tachycardia * Nausea and vomiting Dyspnea Diaphoresis |

* Denotes common and significant reactions:
1 . Anxiety reaction, including hyperventilation syndrome.
2 . Vasovagal syncope occurs with patient upright; any loss of consciousness in the recumbent position implies a severe toxic or anaphylactic reaction.
3 . Although apprehension is classically associated with anxiety and vasoconstrictor reactions, milder toxic and allergic reactions may cause patient apprehension.


sequestered in tissue and have a slower absorption and hence lower blood levels. Agents with a greater volume of distribution or a faster clearance (etidocaine > lidocaine

> mepivacaine > bupivacaine) also produce lower blood levels. Together these effects produce margins of safety for each anesthetic, with etidocaine having the greatest safety margin, followed by bupivacaine, which is equal to or better than lidocaine.

Esters are difficult to measure in the blood because of their rapid hydrolysis by pseudocholinesterase. As a group, toxicity is inversely proportional to the rate of hydrolysis, such that tetracaine is slowly hydrolyzed and hence most toxic, chloroprocaine is quickly hydrolyzed and least toxic, and procaine falls between the two.

**Clearance.**

Amides are metabolized by the liver, in which the clearance rate is a function of hepatic blood flow and extraction capacity of the liver. Decreased hepatic flow, produced by norepinephrine, propranolol, or general anesthesia, slows clearance and potentially raises drug blood levels. Decreased extraction, associated with congestive heart failure, cirrhosis, or hypothermia, likewise may produce a higher blood level. Hypovolemia, which decreases hepatic flow and hence clearance, does not raise blood levels because it causes an offsetting decrease in absorption.

Decreased clearance of esters, hence an increased risk for toxicity, occurs in patients with either low levels of pseudocholinesterase or an atypical form of pseudocholinesterase. Low levels occur in various disease states, including severe liver disease and renal failure, and in pregnancy. Atypical pseudocholinesterase is an inherited trait, and its presence reduces the hydrolysis rate of procaine to a greater extent than low levels do.

There are significant differences between the pediatric and adult population concerning drug distribution and metabolism. Neonates exhibit both reduced levels of pseudocholinesterase and reduced hepatic metabolism, thus increasing the risk of toxicity. In older children, the effects of increased hepatic metabolism and a relatively larger volume of distribution increase their tolerance for higher doses.

**Maximum safe dosage.**

Before one can appreciate this concept, the principles of dosage calculation must be understood (Table 31-10) . The *maximum safe dose* for a drug may be defined as the dose that produces a blood level of the drug just below the toxic level. Based on the previous discussion, it is obvious that *one* maximum dose for an anesthetic agent appropriate for all patients and all conditions cannot be stated. A maximum safe dose cannot be based solely on the weight of a patient. In fact, in an adult, peak blood levels do not correlate well with weight, because the volume of the drug distribution is relatively constant. [74] [75] However, as an approximation, Arthur and McNicol have recommended basing maximum dosages for children on weight. [76] Plain lidocaine may be used in doses of up to 4.5 mg/kg, whereas the addition of epinephrine allows for a maximum dose of 7 mg/kg. Bupivacaine is not recommended for children under the age of 12. Furthermore, the dose should be modified according to the site and mode of

administration.

Maximum safe doses as stated in package inserts should be used only as guidelines because (1) most of them are derived from animal experiments and are based on absorption data only; (2) levels vary with administration site, use of a vasoconstrictor, and, to some extent, with the health of the patient; (3) they can often be exceeded safely when accurately administered (e.g., bupivacaine with epinephrine has been shown to be safe in peripheral nerve blocks and local infiltration in doses of up to 400 mg); and (4) they may be toxic even within the "safe range" when inadvertently injected IV.

---

**TABLE 31-10** -- Calculation of Anesthetic Doses

Anesthetic solutions are marketed with drug concentration expressed as percentages (e.g., bupivacaine 0.25%, lidocaine 1%). To ascertain the strength of a solution in milligrams per milliliter, consider the following:

A 1% solution is prepared by dissolving 1 g of drug in 100 mL of solution.

Therefore, 1 g/100 mL = 1000 mg/100 mL = 10 mg/mL.

To calculate the strength from the percentage quickly, simply move the decimal point 1 place to the right. Examples:

| | | |
|---|---|---|
| 0.25% = 2.5 mg/mL | (e.g., bupivacaine) | |
| 0.5% = 5 mg/mL | (e.g., tetracaine) | |
| 1% = 10 mg/mL | (e.g., lidocaine) | |

| | | |
|---|---|---|
| 2% = 20 mg/mL | (e.g., viscous lidocaine) | |
| 4% = 40 mg/mL | (e.g., cocaine) | |
| 5% = 50 mg/mL | (e.g., lidocaine ointment) | |
| 20% = 200 mg/mL | (e.g., benzocaine) | |

When combined in an anesthetic solution, epinephrine is usually in a 1:100,000 or 1:200,000 dilution.

0.1 mL of 1:1000 epinephrine in 10 mL anesthetic solution = 1:100,000 dilution = 0.010 mg/mL.

0.1 mL of 1:1000 epinephrine in 20 mL anesthetic solution = 1:200,000 dilution = 0.005 mg/mL.

some examples of epinephrine content:

| | **1:100,000** | **1:200,000** |
|---|---|---|
| 5 mL | 0.050 mg | 0.025 mg |
| 10 mL | 0.100 mg | 0.050 mg |
| 20 mL | 0.200 mg | 0.100 mg |

> Therefore, 50 mL of 1% lidocaine with epinephrine 1:200,000 contains 500 mg lidocaine and 0.25 mg epinephrine.

**Inadvertent intravascular injection.**

Most toxic reactions are caused by inadvertent IV injections of anesthetics whose doses were calculated for their intended extravascular sites. For example, lidocaine 300 mg is a safe infiltrative dose that would likely cause toxicity if directly injected into the bloodstream.

Anesthetics that are injected IV must pass through the lungs before they reach other organs. Lung tissue sequesters a significant amount of drug, which lowers the arterial blood concentration. Therefore, anesthetics that bypass the lungs-- either in cases of inadvertent injection into the carotid or vertebral arteries or in patients with intracardiac right-to-left shunts--can produce central nervous system toxicity at low doses. Similarly, intra-arterial injections about the head or neck are capable of retrograde flow into the cerebral circulation if the injection pressure exceeds the arterial pressure. Because the blood volume in the brain is only about 30 mL at any given moment, even 1 mg of lidocaine injected into the carotid artery can produce toxic concentrations. Also, in a patient with low cardiac output and hypovolemia, preferential cerebral blood flow can enhance central nervous system toxicity.

**Host Factors**

Four factors--hypoxia, acid-base status, protein binding, and concomitant drugs--tend to lower the body's systemic tolerance to local anesthetic agents.

**Hypoxia.**

It was initially thought that local anesthetic overdose produced central nervous system stimulation and subsequent intracellular hypoxia, which then became the key precipitant to all toxic manifestations of the drug. It is now known that hypoxia may enhance anesthetic toxicity, but it is not the primary factor.

**Acid-base status.**

Although studies of metabolic alkalosis have produced conflicting results, acidosis, particularly respiratory acidosis, can increase toxicity. The elevated $CO_2$ produced by respiratory acidosis crosses the blood-brain barrier, where it may act directly on the receptor and indirectly by lowering intracellular pH. This latter property causes more drug to ionize, furthering the block in the sodium channel and increasing the potential for toxicity.

**Protein binding.**

Unbound drug concentration relates more closely to toxic effects than does total drug concentration (bound plus unbound) as measured in the blood. The amount of alpha-acid glycoprotein (AAG), the major plasma protein responsible for binding local anesthetics, is considerably decreased in neonates compared with adults, thereby allowing for a greater percentage of free drug. Arthur and McNicol imply that low AAG levels in neonates are responsible for an increased toxic potential. [76] Tucker lists several disease states that alter AAG levels and protein binding but questions whether they lead to changes in free drug concentration in vivo. [77]

**Concomitant drugs.**

For years, barbiturates were used prophylactically to prevent, and therapeutically to treat, local anesthesia-induced seizures. Although at least the latter purpose proved valid, barbiturates were found to worsen anesthetic-induced apnea and cardiovascular depression. Central nervous system depressants should be used with caution when concern exists for local anesthetic toxicity. Central nervous system stimulants have been shown to increase anesthetic-induced excitability and should be avoided. Mixtures of local anesthetics essentially have an additive effect on toxicity, so if 2 drugs are used at half strength, they produce the same degree of toxicity as if each was used alone at normal strength.

### Recognition of Central Nervous System Toxicity

The earliest manifestation of systemic toxicity is central nervous system stimulation resulting from blockade of inhibitory synapses. Central nervous system depression follows and is produced by direct depression of the medulla, although hypoxia may play a role. Signs and symptoms are dose related. Potential signs and symptoms of central nervous system toxicity, in increasing order with representative plasma lidocaine concentrations in parentheses, are numbness of the tongue, light-headedness, tinnitus (4 mug/mL), visual disturbances (6 mug/mL), muscle twitching (8 mug/mL), convulsions (10 mug/mL), coma (15 mug/mL), and apnea (20 mug/mL). Drowsiness, commonly seen at lower doses with lidocaine, is not associated with bupivacaine or etidocaine. Tetracaine may produce apnea or cardiovascular toxicity without central nervous system manifestations.

### Recognition of Cardiovascular Toxicity

Moderate blood concentrations (e.g., lidocaine, 3 mug/mL) of local anesthetics produce slight increases in cardiac output, heart rate, and arterial pressure because of the effects of direct peripheral vasodilation and central nervous system

stimulation. At concentrations generally well above central nervous system toxicity levels, local anesthetics cause direct myocardial depression, manifesting as hypotension and bradycardia, which can lead to cardiovascular collapse. These agents

also slow electrical conduction leading to reentry phenomenon and various supraventricular and potentially lethal ventricular dysrhythmias (especially with bupivacaine and etidocaine).

**Prevention**

Knowledge of factors contributing to toxicity should guide preventive measures. Esters should be avoided in patients with an atypical form or a quantitative deficiency of pseudocholinesterase. Amides should be used with caution in patients with severe liver disease or congestive heart failure. Maximum safe dosages, based on site, technique, epinephrine use, and patient status, should be observed. Epinephrine should be added whenever possible to decrease the drug absorption rate at vascular sites. Drug concentration can be reduced with saline dilution to increase the volume for administration when a large area must be infiltrated. Frequent aspiration should be performed in areas of high vascularity. However, even a negative aspiration may not prevent IV administration. [78] Therefore, slow infiltration is advised for safety and is also associated with less pain.

**Treatment of Systemic Toxicity**

No physician should perform local anesthesia without the ability to recognize and treat a toxic reaction; this includes having all necessary equipment and drugs readily available and being knowledgeable in their use. Despite taking all possible precautions, toxic reactions still occur, and maintaining a rapport with the patient allows early detection and treatment.

Providing proper oxygenation and ventilation at the earliest sign of a reaction is the cornerstone of treatment. Patients who are alert should be encouraged to moderately hyperventilate to lower the $P_{CO_2}$ and raise the seizure threshold. Likewise, for patients who cannot adequately ventilate, intubation with high-flow oxygen and hyperventilation should be performed. An IV line should be started, and vital signs and cardiac rhythm should be monitored closely.

Seizures are generally self-limited but should be treated if they persist or prevent adequate ventilation. Because respiratory depression secondary to toxicity may follow, low-dose diazepam, 2 to 5 mg, or an ultrashort-acting barbiturate (thiopental or sodium methohexital) is preferred. For seizures that persist, intubation and use of a paralyzing agent are recommended to ensure an effective airway and prevent further lactic acidosis. Beware: if toxicity is caused by an ester, especially if there is an associated pseudocholinesterase problem, succinylcholine will compete with the anesthetic for the pseudocholinesterase and may increase the toxicity of both compounds.

Cardiovascular toxicity manifesting as hypotension and bradycardia should be treated with fluids, leg elevation, alpha- and beta-agonists (epinephrine, ephedrine, or dopamine), or atropine as the need dictates.

Although lidocaine (with diazepam pretreatment) has been shown to be effective for

bupivacaine-induced ventricular dysrhythmias, there is strong theoretical and experimental evidence that bretylium is the antidysrhythmic drug of choice. [79] [80] High doses of atropine and epinephrine can be successful in correcting pulseless idioventricular rhythm. Cardiopulmonary resuscitation, of course, should be instituted when necessary.

## Allergic Reactions

### Allergenic Agents

True allergic reactions are rare, accounting for only 1% to 2% of all adverse reactions, but they are important to recognize because of their serious potential. Ester solutions (procaine, tetracaine), which produce the metabolite para-aminobenzoic acid (PABA), account for the great majority of these reactions. Amide solutions (lidocaine, bupivacaine) are rarely involved, and it is usually the preservative methylparaben (MPB), which is structurally similar to PABA, that is responsible. Hence, although pure esters and pure amides do not cross-react, amides may appear to do so if multidose vials containing MPB are used. Also, patients may manifest an allergic response on first contact to a local anesthetic because of previous sensitization to these agents. MPB is found in creams, ointments, and various cosmetics, and PABA is an ingredient in many sunscreen preparations.

Although cell-mediated delayed reactions manifesting as dermatitis may occur, it is immediate hypersensitivity that most concerns the emergency physician. A spectrum of signs and symptoms may occur, from rhinitis and mild urticaria to bronchospasm, upper airway edema, or full-blown anaphylactic shock. Onset may be immediate, at times even occurring during administration of the agent. Diphenhydramine and SQ epinephrine are useful for mild urticaria and bronchospasm, respectively. IV epinephrine, hydrocortisone, and diphenhydramine may be required for more serious reactions.

The more frequent problem facing emergency physicians is the management of the patient who claims to have a past history of local anesthetic allergy. Indeed, most patients, as well as their physicians, tend to assume that any adverse reaction to a local anesthetic procedure is an allergy. Because allergy is rarely the cause, a careful history and a review of prior records, if available, become crucial in evaluating these patients. The history should attempt to uncover the actual cause of the past reaction and the specific agent involved. Inquiry should be made concerning the exact signs and symptoms, technique of administration, amount of drug used, and how the patient was treated. If an allergic reaction cannot be ruled out and the drug previously used is known, it is often recommended to use an agent from the other class (whether amide or ester). Lidocaine from a dental cartridge does not contain MPB, and if this were the allergenic source, then an ester agent could be used. However, if lidocaine from a multidose vial is implicated, one should not use an ester, because MPB may cross-react with PABA. In this case, it may be safer to use an amide without MPB or to choose an alternative (see below). In most cases the allergen is an ester, and the patient can safely be given an amide without MPB. Single-dose ampules of 1% lidocaine without

MPB, readily obtainable from a resuscitation cart, can be used for this purpose.

However, uncertainty often exists regarding the specific agent involved, and the physician must choose an alternative approach to local anesthesia. If the wounds are extensive and the risk is acceptable, general anesthesia may be used;

conversely, if minimal pain is expected and the procedure is short (e.g., 1 or 2 sutures or staples in the scalp), no anesthesia may be required. Other alternatives include parenteral narcotics, benzodiazepines, nitrous oxide inhalation, or a combination of these. These methods may be useful, but the degree of anesthesia produced is often not sufficient. Antihistamines injected into a wound have been successfully used for many years and represent a good alternative. Local anesthetic efficacy is found in varying degrees in all antihistamines.

**Diphenhydramine**

Several studies demonstrated that 1% diphenhydramine (Benadryl) is as effective as 1% lidocaine for infiltrative anesthesia. [ As long as diphenhydramine is not used at concentrations >1%, potential problems of skin necrosis or significant sedation seem to be rare. *The standard 5% parenteral form should be diluted to 1% for SQ injection* (1 mL drug to 4 mL saline). The duration of action for diphenhydramine is shorter than that for lidocaine but appears to be adequate for most procedures. The injection pain of diphenhydramine exceeds that of lidocaine but can be diminished by reducing the concentration to 0.5%. At this concentration, the effectiveness of this agent on facial wounds is lost. [84] The addition of epinephrine to 0.5% diphenhydramine results in a more painful solution with a shorter duration of action than a standard buffered lidocaine with epinephrine solution. [85]

**Skin Testing**

Skin testing and progressive SQ challenge doses deserve special mention because they appear to be logical and well-studied approaches. Intradermal skin testing with local anesthetics is, at best, controversial. False-positive results are frequently produced by local histamine release secondary to needle trauma, tissue distention, or preservatives in the solution. [86] In addition, a high incidence of false-negative results can occur, and it is questionable whether these low-molecular-weight drugs or their allergenic metabolites are ever capable of eliciting positive responses. [87] Other disadvantages of skin testing include its time-consuming nature and its potential hazard when even minute traces of an allergen may precipitate a serious reaction. SQ challenge testing in graduating doses has been advocated and may well eliminate many false responses, but it does not eliminate the problems of time and hazard. Swanson, recognizing that allergy to pure lidocaine is extremely rare, recommends 0.1 mL as a one-shot intradermal skin test. [88] Although his approach eliminates the time disadvantage, the intradermal placement can still produce false responses. It would seem more reasonable to give this test dose subcutaneously while exercising due caution in the unlikely event that a patient exhibits a serious reaction.

**Summary**

Generally speaking, the optimal approach to the patient with a presumed anesthetic allergy is to determine the specific anesthetic agent associated with a presumed allergic reaction and then use a preservative-free agent from the other class (see above discussion). If the agent is unknown, then one should either use an antihistamine or give 0.1 mL of preservative-free lidocaine as an SQ test dose, proceeding with the full dose if no reaction occurs within 30 minutes. Given the studies mentioned earlier, the prudent choice would seem to be diphenhydramine (Benadryl).

## Catecholamine Reactions

Anxiety and vasoconstrictor (epinephrine) reactions are discussed together, because each produces similar manifestations that are related to elevated catecholamine levels. These reactions, which are difficult to distinguish from each other, are relatively common, although generally not serious.

Excess catecholamine levels produce tachycardia; palpitations; hypertension; apprehension; tremulousness; diaphoresis; tachypnea; pallor; and, on occasion, anginal chest pain. Thus, catecholamine excess may resemble the central nervous system stimulation phase of local anesthetic toxicity.

Catecholamine reactions are usually not caused solely by exogenous epinephrine, because if it is used in its optimal concentration (1:200,000), the maximum safe dose (0.25 mg) is rarely exceeded. However, many patients produce significant endogenous catecholamines secondary to anxiety about the anesthetic approach or upcoming procedure. In this case, even the addition of small amounts of epinephrine could trigger a catecholamine reaction. Therefore, patient preparation should include proper explanation and reassurance to decrease anxiety. Caution also should be exercised in patients who have hyperthyroidism, hypertension, or atherosclerotic cardiovascular disease, but those conditions do not contraindicate the judicious use of epinephrine-containing anesthetics. Epinephrine-containing anesthetics should not be given to patients on monoamine oxidase inhibitors (MAOIs).

Treatment includes stopping further drug administration; observing the patient closely; and administering alpha- and beta-antagonists or benzodiazepine agents, if necessary, to combat severe reactions.

## Vasovagal Reactions

Vasovagal reactions are not uncommon, especially in dental procedures (reported incidence, 2% to 3%), during which the patient is generally in an upright position. The patient initially experiences anxiety when a triggering event, commonly the sight or sensation of needle insertion, causes a loss of sympathetic tone and an increase in vagal tone. The resultant hypotension and bradycardia may lead to syncope. Preparation to decrease patient anxiety and *administration of injections with the patient*

*recumbent* are useful preventive measures. Concomitant cardiac monitoring also may help identify the onset of vagally induced bradycardia when suggested by past history. Treatment consists of laying the patient flat and elevating the legs. Rarely, atropine may be required. Should a patient lose consciousness while in a recumbent position, diagnoses other than vasovagal syncope should be considered, although significant bradycardia, and even complete heart block, may accompany a vagal reaction in the supine patient.

## Miscellaneous Reactions

### Malignant Hyperthermia

There is some concern that amides may produce malignant hyperthermia in susceptible individuals. It is recommended

that esters be used in lieu of amides in patients from families with this genetic abnormality.

### Methemoglobinemia

Despite an excellent safety margin and the fact that many consider it to be the agent of choice for IV regional anesthesia, prilocaine has declined in popularity recently because of its ability to cause methemoglobinemia. [89] The effect is seen when cumulative or single doses exceed 600 mg. In large doses lidocaine also may produce methemoglobinemia.

Benzocaine, a common ingredient in several over-the-counter and prescription topical anesthetics, is well known to produce methemoglobinemia. [90] Although this reaction is infrequent and may occur secondary to a heterozygote enzyme deficiency, it also is known to occur with excessive topical or SQ application in normal individuals, especially children. Methemoglobinemia may rarely occur following the use of benzocaine-containing sprays used to anesthetize the throat (e.g., during endoscopy).

# Chapter 32 - Nerve Blocks of the Thorax and Extremities

**Edwin Dean, Michael Orlinsky**

The use of nerve blocks is an integral part of the practice of emergency medicine. This chapter provides technical guidance for the performance of commonly used nerve blocks.

While virtually every peripheral nerve can be blocked at some point along its course from the spine to the periphery, digital nerve blocks of the fingers and toes are more commonly used than proximal blocks. In a survey of practicing emergency physicians, 99% used digital blocks, 43% used blocks at the wrist, 4% at the elbow, and 4% at the axilla. [1]

Nerve blocks may be underused in the emergency department. Potential applications include femoral blocks for femur fractures, ankle blocks for foot lacerations, intercostal blocks for rib fractures, supraorbital blocks for foreheads traumatized by contact with a windshield or other blunt object, and ear blocks for ear lacerations.

The preparation, technique, choice of anesthetic, precautions, and complications are similar for all nerve blocks and are described in general in the following sections. The clinician is encouraged to use the same basic techniques and precautions for all nerve blocks. Specific precautions unique to a particular nerve block are included with the description of that block; obvious precautions, such as aspiration before

injection when the needle is in close proximity to a vascular structure, are not restated to avoid redundancy.

## GENERAL CONCEPTS

**Indications**

For most of the lacerations and injuries seen in the emergency department, local infiltrative anesthesia is adequate and more efficient than using a nerve block. Local infiltration is quick, reliable, and effective compared with many of the nerve blocks, which tend to require a more extensive setup and have a less reliable and longer onset. Furthermore, those patients who require extensive repair and anesthesia of the entire extremity are often referred to a specialist, who may prefer to examine an unanesthetized limb.

In general, a nerve block is indicated when it will provide advantages over other techniques. Scenarios in which this requirement is met include the following:

When distortion from local infiltration hampers closure (e.g., facial wounds) or

may compromise blood flow (e.g., fingertip)

When anesthesia is required over a large area and multiple injections would be painful, or the large amount of anesthetic needed for local infiltration exceeds the recommended dose

When a nerve block is the most efficacious form of treatment, as in an intercostal block for treating a rib fracture in a patient with chronic obstructive pulmonary disease

When extensive limb surgery or manipulation is required and other options are not available

## Preparation

A brief history, including drug allergies, medications, and systemic illnesses should be taken from the patient. Peripheral vascular, heart, and liver disease increase the risk of severe complications. Monoamine oxidase inhibitors preclude the use of anesthetic agents with epinephrine, as the vasoconstrictor may cause exaggerated cardiac responses in this setting.

### Instructions

The procedure should be explained to the patient, including the pain of the needle insertion, paresthesias that may be felt, and possible complications that may occur. *The possible need for additional anesthetic or alternate procedures if the initial nerve block fails should also be discussed beforehand*. The patient should understand that the additional administration of anesthetic is part of the normal procedure rather than an attempt to correct an incomplete nerve block.

### Equipment

The degree of equipment preparation depends on the extent of the procedure. For a simple digital block, a 10-mL syringe, an 18-ga needle for drawing the solution from the vial, and a 3.75-cm, 25- or 27-ga needle for the nerve block will suffice. Note that the needle sizes given in the text are general recommendations. For most blocks, a needle 2 gauges larger or smaller or 1 cm longer or shorter is adequate.

For more elaborate blocks, the necessary equipment is listed in Table 32-1 . Use of an extension tubing set between the needle and a stopcock syringe assembly facilitates independent needle placement and syringe manipulation. In addition, standard resuscitation equipment for advanced cardiac life support should be readily available any time local anesthetic agents are given.

### Choice of Anesthetic

The factors influencing the choice of anesthetic agent for nerve block are similar to those for local infiltration. In general, most nerve blocks are done for the repair of painful traumatic injuries that are likely to cause pain long after the repair is complete. In such cases, anesthetics with the longest duration of action should be selected to maximize the patient's analgesia. For most of the blocks described in this chapter, 0.25% bupivacaine is suggested as the anesthetic of choice, but equal volumes of 1% lidocaine with epinephrine can be substituted. Care must always be taken to *avoid exceeding* the recommended dosages of anesthetic.

Buffering the anesthetic may lessen the pain of infiltration. Buffered lidocaine is less painful and equally as efficacious as plain lidocaine in digital blocks. [2] Similarly, heating the anesthetic to 37 to 41 °C also reduces the pain of digital blocks (also see Chapter 31) . [3]

### Positioning of the Patient

Ideally, nerve blocks should be performed with the patient in the supine position. This enhances patient comfort and prevents the unexpected vasovagal syncope that may occur when the patient is in an upright position. Vasovagal syncope may be induced from the pain of the injection or "needle phobia." Although this reaction is more common with fair-skinned patients, every patient is at risk for fainting at the site of a needle or during a painful injection.

### Preparation of the Area to Be Blocked

Although the incidence of infection following nerve blocks is minuscule, the field should be prepared in an aseptic fashion before needle puncture. This can be accomplished by swabbing the area with povidone-iodine solution or alcohol and then using standard aseptic injection technique. Sterile drapes and gloves are recommended in addition to aseptic skin preparation for the initiation of blocks that (1) are close to large joints, vessels, and nerves; (2) are located in inherently contaminated areas of the body; or (3) require

| **TABLE 32-1** -- Equipment Needed for Proximal Nerve Block Trays |
| --- |
| 6 gauze sponges |
| 4 towels |

| |
|---|
| 1 antiseptic-solution receptacle |
| 1 receptacle for saline flush solution |
| 1 anesthetic-solution receptacle (30-mL capacity) |
| 1 10-mL syringe for local anesthetic injection |
| 1 30-mL syringe for nerve block injection |
| 1 three-way stopcock |
| 1 IV extension tubing set |
| 1 18-ga needle for withdrawing anesthetic from the vial |
| 1 each 3.75-cm 23-, 25-, and 27-ga needles for nerve blocks |

simultaneous palpation of the underlying structures while injecting.

**Choosing the Nerves to Block**

Successful anesthesia often requires blocking 2 or more nerves. When deciding which nerves are involved, 2 points must be considered. First, the cutaneous distribution of the various peripheral nerves differs slightly from patient to patient, and second, the sensory innervation of adjacent nerves overlaps to some degree. A liberal margin of error should be used when determining which nerves supply the desired area of anesthesia.

**Locating the Nerve**

Inserting the needle in close proximity to the nerve--that is, "next to" small nerves and "into" the nerve sheath of large nerves--is the essential step in a successful block. When searching for the nerve, its anatomic relationship at the block site can be generalized into 1 of 3 types. First are those sites with good structural landmarks (e.g., prominent bones or tendons) immediately next to the nerve. For example, the digital nerves are reliably found at the 2, 4, 8, and 10 o'clock positions around and just superficial to the proximal phalanx, and the median nerve lies between the palpable palmaris longus and flexor carpi radialis tendons at the proximal crease of the wrist. Second are the sites with easily palpable arteries adjacent to the nerves such as in the axilla and groin. Third are the sites with poor landmarks.

Blocking nerves with good structural or vascular landmarks is straightforward: the landmarks are palpated, the course of the nerve in relation to those landmarks is visualized in the mind's eye, and the needle is inserted in close proximity to the nerve. Paresthesias, as discussed later, may ensure close proximity.

Blocking those nerves with poor landmarks, such as the radial nerve at the elbow, requires skill through practice, some degree of luck, or a nerve stimulator if they are to be blocked consistently.

**Nerve Stimulator**

Despite its sophisticated name, a nerve stimulator is inexpensive and simple to use. With minimal practice, the needle can be placed easily and reliably within a few millimeters of the nerve. The device is simple, consisting of a battery-operated nerve stimulator that delivers current in the range of 0.1 to 0.5 mA, a disposable Teflon-coated needle, and an electrocardiogram pad. One wire electrode from the nerve stimulator is attached to the electrocardiogram pad, which is placed on the limb approximately 10 to 15 cm from the injection site. The other wire electrode is attached to the Teflon-coated needle at its hub. Once inserted into the tissue, the needle tip is electrically active. When in close proximity to the nerve, electric current stimulates the nerve, producing twitching of the muscles supplied by that nerve. For example, when the ulnar nerve is stimulated, the small and ring fingers flex. Anesthetic solution can then be injected into the area at the stimulation site.

**Paresthesia**

When a nerve stimulator is not available, another useful technique to ensure that the needle tip is in close proximity to the nerve is to elicit a paresthesia. By touching and mechanically stimulating the nerve with movement of the needle tip, a tingling sensation or jolt known as a *paresthesia* is felt along the distribution of the nerve.

In practice, the jolt of a true paresthesia is often difficult to distinguish from the "ouch" of a pain-sensitive structure. When blocking proximal nerves of the elbow or axilla, the paresthesia travels far enough away from the injection site that it can be distinguished from locally induced pain. Paresthesias at the level of the hand and wrist are much less

reliably distinguished from pain. In both cases, the paresthesia is a subjective feeling that requires intelligent and cooperative patients who understand what they are expected to feel and who remain relaxed and attentive so that they are able to distinguish an ouch from a jolt. All too often, the patient in pain is willing to tell the physician what the physician wants to hear: the sensation that just made the patient jump was a "paresthesia." Before the procedure, a simple explanation of what the patient should or may feel will facilitate cooperation.

## Injecting the Anesthetic

One strives to ensure that the anesthetic agent is not inadvertently injected into the vessels or nerve bundle. Before injection, the syringe is aspirated to check for blood. If no blood is aspirated, the anesthetic is injected while the extremity is observed for blanching, which suggests intravascular injection. If blanching occurs, the needle should be repositioned before further injection. The technique of syringe aspiration is not necessary when using 25- or 27-ga needles in the proximity of small vessels (e.g., during digital blocks).

The onset and duration of anesthesia are greatly influenced by the proximity of the injected anesthetic to the nerve. Onset is within a few minutes if the anesthetic is in immediate proximity to the nerve. Onset takes longer or may not occur if the anesthetic must diffuse more than 2 to 3 mm, which underscores the importance of locating the nerve before injection.

More anesthetic is required if it must diffuse a large distance to the nerve. A range of suggested volumes of anesthetic is given with each nerve block description. For blocks in which a definite paresthesia is elicited or a nerve stimulator is used, the minimal recommended amount of anesthetic suffices. For many of the blocks of the smaller nerves, paresthesias are not easily elicited, and the anesthetic is placed in the general vicinity of the nerve. For these blocks, or when doubt exists about proximity of the needle to the nerve, larger amounts of anesthetic are recommended. This point cannot be emphasized strongly enough. The difference between a successful and an unsuccessful block may be merely an additional 2 mL of anesthetic. When in doubt, err on the high side of the recommended dosage.

Onset of the anesthetic occurs in 2 to 15 minutes, depending on the distance the anesthetic must diffuse to the nerve. One should wait for 30 minutes before deciding that the block was unsuccessful.

## Complications and Precautions

Complications may result from poor peripheral nerve block technique. General precautions include measures to minimize nerve injury, intravascular injection, and systemic toxicity.

No statistics exist on the complication rate from nerve blocks performed by emergency physicians. Anesthesiologists report rates of less than 1% for blocks of the nerves of the

hand and wrist and up to 10% for axillary blocks. [4] In this study there was no breakdown of serious versus minor complications, although local complications (hematoma and neuropathy) are most common. Generally, infrequently performed blocks, blocks that require high doses of anesthetic, and blocks close to major vascular structures have the higher rates of problems. The technical ability of the practitioner largely determines the outcome.

**Nerve Injury**

Neuritis, an inflammation of the nerve, is the most common nerve injury but overall is a rare complication. [5] [6] The patient may complain of pain and various degrees of nerve dysfunction, including paresthesia or motor or sensory deficit. Most cases are transient and resolve completely. Supportive care and close follow-up are the mainstays of treatment.

Nerve injury can occur secondary to (1) direct trauma from the needle, (2) ischemia due to intraneuronal injection, or (3) chemical irritation from the anesthetic. Proper needle style, positioning, and manipulation minimize direct nerve damage. A short beveled needle should be used and maneuvered so that the bevel is parallel to the longitudinal fibers. Sharp pain or paresthesia indicates that the needle is close to or in the nerve. Excessive needle movement should be avoided when the needle tip is contacting a nerve. Attaching the needle to an IV extension tube reduces needle movement and subsequent nerve damage during syringe plunger manipulation.

Intraneuronal injection may cause nerve ischemia and injury. Elicitation of a paresthesia or severe pain suggests that the needle has made contact with the nerve. *When a paresthesia is elicited, the needle must be withdrawn 1 mm before the anesthetic is injected.* If the paresthesia occurs during injection, the needle must be repositioned. Most neurons are surrounded by a strong perineural sheath through which the nutrient arteries run lengthwise. Injection directly into a nerve sheath may increase the pressure within the nerve and compress the nutrient artery. Impaired blood flow results in nerve ischemia and subsequent paralysis. Intraneuronal injection is often heralded by severe pain, which worsens with further injection and may radiate along the course of innervation. The operator may notice difficulty depressing the syringe plunger. If the needle tip is in proper position, slow injection of the anesthetic should be minimally painful, and the anesthetic should go in without resistance.

Concentrated anesthetics can produce a chemical irritation of the nerve. Therefore, emergency clinicians should use only recommended doses and concentrations of anesthetic (Table 32-2) .

**Intravascular Injection**

Intravascular injection results in both systemic and limb toxicity. Inadvertent intravascular injection produces high blood levels of the anesthetic, with resultant toxicity, as discussed earlier. Particular care must be taken when administering large

amounts of anesthetic in close proximity to large blood vessels.

Intra-arterial injection of anesthetic with epinephrine may cause peripheral vasospasm that further compromises injured tissue. Intravascular anesthetic is not toxic to the

| Site | Anesthetic: 1.0% Lidocaine or 0.25% Bupivacaine (Both with Epinephrine) | Volume (mL) |
|---|---|---|
| **TABLE 32-2** -- Recommended Volumes of Anesthetic for Various Nerve Blocks | | |
| **Axillary** | | 40-50 |
| **Elbow** | | |
| Ulnar | | 5-10 |
| Radial | | 5-15 |
| Median | | 5-15 |
| **Wrist** | | |
| Ulnar | | 5-15 |
| Radial | | 5-15 |
| Median | | 3-5 |

| | | |
|---|---|---|
| **Hip** | | |
| Femoral | | 10-30 |
| 3-in-1 | | 30-50 |
| **Knee** | | |
| Tibial | | 5-15 |
| Peroneal | | 5-10 |
| Saphenous | | 5-10 |
| **Ankle** | | |
| Posterior tibial | | 5-10 |
| Deep peroneal | | 3-5 |
| Saphenous, sural, and superficial peroneal | | 4-10 each |
| **Intercostal** | | 5-15 each |

| | 1.0% Lidocaine or 0.25% Bupivacaine (Both *Without* Epinephrine) | |
|---|---|---|
| **Hand** | | |
| Metacarpal and web space | | 2-4 |
| Finger | | 1-2 |
| **Foot** | | |
| Metatarsal | | 10 |
| Web space | | 3-5 |
| Toe | | 2 |

limb itself, although it may produce transient blanching of the skin by displacing blood from the vascular tree. Epinephrine, however, can cause a prolonged vasospasm and subsequent ischemia if it is injected into an artery. Severe epinephrine-induced tissue blanching or vasospasm may be reversed with local or intravascular injection of phentolamine.

Although vasospasm associated with epinephrine in anesthetic solutions has not been observed for nerve blocks, experience in related clinical situations should guide therapy. Roberts and Krisanda used a total of 5 mg of phentolamine infused intra-arterially to reverse arm ischemia following 3 mg of epinephrine inadvertently administered into the brachial artery during cardiac resuscitation. [7] Digital ischemia from inadvertent epinephrine autoinjection has been treated by both proximal "digital block" with 2 mg of phentolamine [8] and by local infiltration at the ischemic site with 1.5 mg of phentolamine. [9]

The route of phentolamine administration may be guided by the clinical situation.

Phentolamine must reach the site of vasospasm. Arterial injection has the advantage of

delivering the medication directly to the spasmed arteries. Local infiltration may be effective for ischemia of a single toe or finger. For larger areas of involvement or in instances where local infiltration is ineffective, intra-arterial injection should be used. A dose of 1.5 to 5 mg appears to be effective in most cases, [7] [8] [9] though a total of 10 mg may be used for local infiltration. Phentolamine 5 mg can be mixed with 5 to 10 mL of either normal saline or lidocaine. The small volume of the distal pulp space may limit the infiltration dose volume to 0.5 to 1.5 mL in the fingertip. Larger volumes and dosages can be used in proximal infiltrations. For intra-arterial infusion at the radial artery in the wrist or the dorsalis pedis on the dorsum of the foot, dosages of 1.5 to 5 mg of phentolamine are suitable. Slow infusion or graded dosages of 1 mg may provide enough phentolamine to reverse ischemia without excessive systemic effects, primarily hypotension.

### Hematoma

Hematoma formation may result from arterial puncture, particularly during axillary and femoral blocks. Direct pressure for 5 to 10 minutes usually controls further bleeding. Use of the recommended 23- to 27-ga needle minimizes bleeding from the punctured artery.

### Infection

Infection is rare and can be minimized by following aseptic technique and using the lowest possible concentration of epinephrine. Injection should be made through noninfected skin that has been antiseptically prepared. Injection through a site of infection may spread the infection to adjacent tissues, fascial planes, and joints. This is particularly a concern in the hand and foot.

### Limb Injury

Injury to the anesthetized limb can result if the patient is permitted to use the limb or is advised to use heat or cold application or to perform wound care before the anesthesia has worn off. With major nerve blocks, the patient should not be released home until sensation and function have returned. With minor blocks, the patient may be sent home but should be properly cautioned. Care must be taken to avoid ischemia-producing compression dressings (e.g., elastic bandages), because the anesthetized area may not sense impending problems.

## SPECIFIC NERVE BLOCKS

### Intercostal Nerve Block

The blocking of the intercostal nerves produces anesthesia over an area of their cutaneous distribution (Fig. 32-1) (Figure Not Available) . It is useful for analgesia of rib fractures. Rib fractures are typically quite painful, causing the patient to splint

respirations to avoid excessive movement of the injured site. The resulting hypoventilation, atelectasis, and poor expectoration may cause hypoxia or lead to pneumonia, particularly in patients with preexisting pulmonary disease and minimal respiratory reserve, in which further impairment of function causes significant respiratory compromise. Anesthetizing the injured rib eases the pain and facilitates deep breathing and coughing.

**Figure 32-1** (Figure Not Available) Area of anesthesia and cutaneous distribution of the intercostal nerve. *(From Moore DC: Regional Block: A Handbook for Use in the Clinical Practice of Medicine and Surgery. 4th ed. Springfield, Ill, Charles C Thomas, 1971, p 159. Reproduced with permission.)*

It is unclear whether intercostal nerve blocks are superior to oral analgesics in the treatment of rib fractures. Current studies suggest that intercostal block may be superior to analgesics in patients who have undergone thoracotomies. [10] [11] Those receiving intercostal nerve blocks had better results on pulmonary function tests, greater oxygenation, and earlier ambulation and discharge than those receiving opioid analgesics. There are no controlled studies comparing intercostal blocks with oral analgesics in patients with the kinds of rib fractures that are commonly managed on an outpatient basis.

There are several arguments against the routine use of intercostal nerve blocks in the emergency department. Rib fractures are often tolerated well in young patients, who usually require minimal oral analgesics. Furthermore, these blocks have a short duration of action. The typical duration of action of a long-acting anesthetic with epinephrine is 8 to 12 hours. However, the patient often receives partial analgesia for up to 3 days, a period of time that cannot be attributed to the direct action of the anesthetic on the nerve. Perhaps the anesthesia reduces muscle spasm and the associated cycle of pain.

The perceived high incidence of pneumothorax and unsuccessful blocks is another reason intercostal nerve blocks are not used in the emergency department. Yet these fears appear to be unfounded. Moore reported that in more than 10,000 individual rib blocks done by physicians in all stages of training, the incidence of pneumothorax was less than 0.1% 12 and Moore and Bridenbaugh reported a 98% success rate in a series of 5000 intercostal nerve blocks. [13] Only 31% of emergency clinicians use this block, suggesting it may be underused. [1]

The suggested approach to discussing intercostal blocks is to give patients the facts with regard to duration of analgesia and possible complications and then allow them to decide on the method for themselves. Often they prefer oral analgesics initially but may return for further relief of pain, at which time they are more amenable to the nerve block.

**Anatomy**

Each thoracic nerve exits the spine through the intervertebral foramen, which lies midway between adjacent ribs. It immediately gives off the posterior cutaneous branch, which supplies the skin and muscles of the paraspinal area. The intercostal nerve continues and gives off the lateral cutaneous branches at the midaxillary line. These

branches are the sensory supply to the anterior and posterior lateral chest wall.

The intercostal nerve runs with the vein and artery in the subcostal groove (Fig. 32-2) (Figure Not Available) . Posteriorly, the nerve is separated from the pleura and the lungs by the thin intercostal fascia. Particular care must be taken to avoid puncture of the thin fascia and underlying lung when blocking the nerve in the posterior aspect of the back. Fortunately, most rib fractures occur in the anterior or lateral portion of the ribs and can be blocked in the posterior axillary line, where the internal intercostal muscle lies between the nerve and the lung's pleura and provides a buffer for minor errors in needle placement.

**Technique**

For adequate analgesia of most rib fractures, the lateral cutaneous branch needs to be anesthetized. Blocks are usually performed between the posterior axillary and midaxillary line at a point proximal to the origin of this branch. Explanation of the procedure, its benefits, and its risks, including potential pneumothorax, systemic toxicity, and ineffective block, should be done before proceeding.

A 10-mL syringe with a 3.75-cm, 23- to 25-ga needle is used. The rib is palpated, and the area is prepared in the usual aseptic manner. The index finger of the nondominant hand is used to retract the skin at the lower edge of the rib cephalad and up over the rib (Fig. 32-3) (Figure Not Available) . With the syringe in the opposite hand, the skin is punctured at the tip of the finger that is retracting the skin. The syringe is held at an 80° angle with the needle pointing cephalad. The hand holding the syringe rests on the chest wall for stability. In this position, the depth of needle penetration is well controlled. The needle is slowly advanced until it comes to rest on the lower border of the rib.

At this point, the skin retraction is released. The skin returning to its natural position moves the needle shaft perpendicular to the chest wall and the needle tip to the inferior margin of the rib. The syringe is shifted from the dominant hand to the index and thumb of the nondominant hand. The middle finger of the same hand rests against the shaft of the needle and, by exerting gentle pressure on the shaft, walks the needle off the lower edge of the rib. Again, the palm of the hand is planted firmly on the chest wall to ensure control of the needle. With the help of the dominant hand, the needle is slowly advanced 3 mm. The needle is aspirated, and then 2 to 5 mL of anesthetic are injected while the needle is carefully moved in and out 1 mm, which ensures that the compartment containing the nerve between the internal and external intercostal muscles is penetrated. This may also serve to minimize intravascular injections. The procedure is repeated ideally on the *2 ribs above and below* to ensure that the overlapping innervation from adjacent nerves is blocked.

Although the procedure just discussed seems extensive, it takes 1 to 2 minutes to perform once the operator is familiar with the technique, and 3 to 5 intercostals can be blocked in 10 minutes total time.

**Precautions**

The needle must be initially placed at the lower edge of the rib. If it contacts the rib above this point, it cannot be walked off the lower edge of the rib at the proper angle. If it is inserted too low, over the intercostal space, it may be advanced too deep through the pleura and into the lung before the operator realizes the misplacement. Before inserting the needle, the depth of the bone should be estimated. If the bone is not encountered by this depth, needle position should be reevaluated. Even after the needle has been properly walked off the edge of the rib, care must be taken to avoid puncture of the pleura and lung. The depth of the intercostal groove in which the nerve runs is 0.6 cm posteriorly, diminishing to 0.4 cm anteriorly.

Because the incidence of pneumothorax is low, a chest radiograph is not routinely required after this procedure. The asymptomatic patient is observed for 15 to 30 minutes and instructed to return if problems arise. If the patient has

**Figure 32-3** (Figure Not Available) Method of retracting skin and the proper needle insertion site for intercostal block. See text for details. *(From Moore DC: Regional Block: A Handbook for Use in the Clinical Practice of Medicine and Surgery. 4th ed. Springfield, Ill, Charles C Thomas, 1971, p 153. Reproduced with permission.)*

symptoms of pneumothorax (e.g., cough, a change in the nature of the pleuritic pain, or shortness of breath), he or she should have a chest film taken before discharge.

If the physician inadvertently causes a pneumothorax, treatment depends on the size. Many pneumothoraces are small. Those <20% may be observed for 6 hours. [13] If the pneumothorax does not grow in size, the patient may be released home with arrangements for follow-up. Needle aspiration of larger pneumothoraces may be all that is needed. A chest tube is necessary if this method fails (see Chapters 8 and 9) .

Toxicity from the epinephrine or the anesthetic is another complication. The intercostal block is associated with the highest plasma concentrations of anesthetic for a given dose when different sites are compared. [14] The exact reason for the high plasma levels is not entirely clear. Moore and colleagues suggest this is because the anesthetic is injected close to the intercostal vessels [14] ; however, other blocks are also performed close to large vessels without a comparable rise in plasma levels. Regardless, the maximum recommended dose of anesthetic should not be exceeded. Systemic absorption of epinephrine may prove dangerous in patients taking monoamine oxidase inhibitors or those with preexisting heart disease of hypertension. Bupivacaine without epinephrine should be used in these patients.

## Nerve Blocks of the Upper Extremity

The upper extremity is supplied by the brachial plexus, whose branches--primarily the median, radial, ulnar, and musculocutaneous nerves--can be blocked at the axilla, elbow, wrist, hand, or fingers. Nerve blocks at the axilla and elbow are seldom used in the emergency department. Nerve blocks of the wrist are performed occasionally before

painful procedures or for repair of injuries to the hand. Metacarpal and digital blocks are used frequently to treat fractures, lacerations, and infections of the fingers.

**Nerve Blocks at the Elbow**

The median, ulnar, and radial nerves can be blocked at the elbow, providing anesthesia to the distal forearm and hand (Fig. 32-4) (Figure Not Available) . These nerves are blocked individually to supplement incomplete axillary blocks or blocked together to provide anesthesia to the forearm and hand. For most injuries extensive enough to require nerve block at the elbow, all 3 nerves must be blocked for successful anesthesia because of the variable and overlapping innervation of the forearm. Furthermore, injuries to the proximal and middle forearm may require an additional circumferential subcutaneous (SQ) field block of the lateral, medial, and posterior cutaneous nerves.

**Ulnar nerve: Anatomy and technique.**

The ulnar nerve can be palpated in the ulnar groove on the posteromedial aspect of the elbow between the olecranon and medial condyle of the humerus (Figs. 32-5 (Figure Not Available) and 32-6) (Figure Not Available) . This nerve supplies the innervation to the small finger and ulnar half of the ring finger and the ulnar aspect of the hand (see Fig. 32-4) (Figure Not Available) .

With the elbow flexed, the nerve is palpated in the groove. A 3.75-cm, 23- to 25-ga needle is inserted 1 to 2 cm proximal to the groove and directed toward the groove parallel to the course of the nerve. The needle tip comes to rest close to the proximal end of the groove. Care is taken to avoid blocking the nerve in the groove, where it is prone to damage. For similar reasons, paresthesia may be elicited but is not vigorously sought. Once the needle tip is properly positioned, 5 to 10 mL of anesthetic are deposited. If a nerve stimulator is used, flexion of the small and ring fingers signals proximity to the nerve.

**Radial nerve: Anatomy and technique.**

The radial nerve and sensory branch of the musculocutaneous nerve run together in the sulcus between the biceps and the brachioradialis muscle on the anterolateral aspect of the elbow (Fig. 32-7) (Figure Not Available) . The block produces anesthesia to the lateral dorsum of the hand and the lateral aspect of the forearm (see Fig. 32-4) (Figure Not Available) .

The sulcus in which the nerve runs is palpated between the sharp border of the biceps muscle and the medial border of the brachioradialis in the antecubital fossa just proximal to the skin crease of the elbow. Palpation is greatly facilitated by having the patient, with the elbow flexed at 90°, contract and relax these muscles isometrically so that their borders are better defined. The skin is punctured with a 3.75-cm, 23- to 25-ga needle halfway between the muscles, or 1 cm lateral to the biceps tendon, at a point 1 cm proximal to the antecubital crease. A paresthesia is sought at a depth of 2 cm by probing in a fan-like pattern. If one is unsuccessful after a brief search, 5 to 15 mL of

anesthetic should be injected at this depth. Because of the depth and poor landmarks of this nerve, the nerve stimulator greatly facilitates the search for the nerve, which, when stimulated, produces extension of the fingers and wrist.

**Median nerve: Anatomy and technique.**

The median nerve runs medial to the brachial artery in the anteromedial aspect of the elbow (see Fig. 32-7) (Figure Not Available) . The nerve block anesthetizes the index, middle, and radial portion of the ring fingers and the palmar aspect of the thumb and lateral palm (see Fig. 32-4) (Figure Not Available) .

The brachial artery is palpated in the flexed arm at the elbow just proximal to the antecubital crease and medial to the prominent biceps tendon. Once the anatomy is defined and marked in the flexed arm, the arm is extended to 30°. A 3.75-cm, 23- to 25-ga needle is inserted slightly medial to the artery and perpendicular to the skin to the depth of the artery, about 2 to 3 cm, and 5 to 15 mL of anesthetic are injected. Again, the nerve stimulator facilitates the process and produces flexion of the wrist and index finger.

## Nerve Blocks at the Wrist

The median, ulnar, and radial nerves may be blocked at the wrist, providing anesthesia to the hand. Although 43% of surveyed emergency physicians use this block in their practice, 89% of these stated that they use it rarely. [1]

Most extensive injuries and procedures for which a wrist nerve block could be used can also be managed using local infiltration or a digital block. Compared with direct infiltration, wrist block anesthesia can have a slow and unreliable onset and can require more time to take effect if all 3 nerves are to be blocked. There are several circumstances, however, in which wrist nerve blocks are more advantageous than other types.

Diffuse lesions that can be difficult to anesthetize with local infiltration can easily be anesthetized with a wrist block. Deep abrasions with embedded debris, commonly the result of "road burn" from bike and motorcycle crashes, can be cleaned and debrided painlessly after a nerve block at the

**Figure 32-4** (Figure Not Available) Cutaneous nerve supply of the upper limb. *(From Bridenbaugh LD: The upper extremity: Somatic blockade. In Cousins M, Bridenbaugh PO (eds): Neural Blockade in Clinical Anesthesia and Management of Pain. 2nd ed. Philadelphia, JB Lippincott, 1988, p 412. Reproduced with permission.)*

wrist. Hydrofluoric acid burns, which require treatment with numerous SQ injections of calcium gluconate, are handled mercifully after a wrist nerve block. Hence, wrist blocks are advantageous in the severely swollen and contused hand, whenever small amounts of anesthetic injected locally may increase the tissue pressure and produce further pain.

Compared with nerves in the axilla and elbow, the nerves in the wrist are more easily located anatomically and can be blocked more reliably. All 3 nerves lie in the volar aspect of the wrist near easily palpated tendons. A nerve stimulator is not necessary but may be useful in locating the nerves, particularly when one is learning how to perform these blocks.

The anatomy and technique for blocking each nerve individually follow. Note that the median nerve lies in the midline and deep to the fascia, and the ulnar and radial nerves lie on their respective sides and have branches that wrap around dorsally. Blocking all 3 nerves at the wrist requires a block that, when viewed end-on, roughly resembles a horseshoe straddling a horseshoe stake (Fig. 32-8) (Figure Not Available) .

**Median nerve: Anatomy and technique.**

In the wrist, the median nerve lies below the palmaris longus or slightly radial to it between the palmaris and the flexor carpi radialis (see Figs. 32-8 (Figure Not Available) and 32-9) (Figure Not Available) . Both tendons are easily palpated, but the palmaris may be absent in up to 20% of patients, in which case the nerve is found about 1 cm in the ulnar direction from the flexor carpi radialis. The nerve lies deep to the fascia of the flexor retinaculum at a depth of 1 cm or less.

The palmaris longus is located by having the patient oppose the thumb and small finger with the wrist flexed against resistance. The site of the nerve block is selected on the radial border of the palmaris tendon just proximal to the proximal wrist crease. A 3.75-cm, 25-ga needle is inserted perpendicularly and advanced slowly until a slight "pop" is felt as the needle penetrates the retinaculum and a paresthesia is produced. If no paresthesia ensues, it may be elicited in a more ulnar direction under the palmaris tendon. If a paresthesia is still not elicited, 3 to 5 mL of anesthetic are deposited in the proximity of the nerve at a depth of 1 cm under the tendon. It is better to err slightly on the deep side of the retinaculum and continue depositing anesthetic as the needle is withdrawn, because the retinaculum is an effective barrier to a successful nerve block from a superficially injected anesthetic.

**Radial nerve: Anatomy and technique.**

The radial nerve follows the radial artery into the wrist but gives off branches proximal to the wrist. These branches wrap around the wrist and fan out to supply the dorsal radial aspect of the hand (Fig. 32-10) .

Nerve block requires an injection in close proximity to the artery and a field block that extends around the dorsal aspect of the wrist. A 3.75-cm, 25-ga needle is inserted immediately lateral to the palpable artery at the level of the proximal palmar crease. At the depth of the artery 2 to 5 mL of anesthetic are injected. Another 5 to 10 mL are distributed in an SQ field block from the initial point of injection to the dorsal midline. The needle must be withdrawn and repositioned to complete the block. The discomfort of numerous needle sticks is decreased if the needle is repositioned to a site that has been

anesthetized previously.

**Ulnar nerve: Anatomy and technique.**

The ulnar nerve follows the ulnar artery into the wrist, where they both lie deep to the flexor carpi ulnaris (see Figs. 32-8 (Figure Not Available) and 32-9) (Figure Not Available) . The flexor carpi ulnaris tendon is easily palpated just proximal to the prominent pisiform bone by having the patient flex the wrist against resistance. At the level of the proximal palmar crease, the artery and the nerve lie just off the radial border of the flexor carpi ulnaris; however, the nerve lies between the tendon and the artery and deep to the artery, making it difficult to approach the nerve from the volar aspect of the wrist without involving the artery.

Nerve block of the ulnar nerve can be carried out by 2 different approaches: lateral and volar (see Fig. 32-9) (Figure Not Available) ; the lateral approach may be easier because of the reason stated previously. For the lateral approach, a 3.75-cm, 25-ga needle is inserted on the ulnar aspect of the tendon at the proximal palmar crease and is directed horizontally under the flexor carpi ulnaris for a distance of 1.0 to 1.5 cm. After a paresthesia is elicited, 3 to 5 mL of anesthetic are deposited. It is important to elicit a paresthesia, because the nerve lies in a thick neurovascular bundle. If no paresthesia is elicited, the needle can be directed toward the ulnar bone at a point deep to the flexor carpi ulnaris, and the anesthetic can be inserted as the needle is withdrawn.

Like the radial nerve, cutaneous nerves branch off the ulnar nerve, wrap around the wrist, and supply the dorsum of the hand. These are blocked with a 5- to 10-mL SQ band of anesthetic from the lateral border of the flexor carpi ulnaris to the dorsal midline (see Fig. 32-8) (Figure Not Available) . Another advantage of the lateral approach is that the dorsal branches can be blocked from the same injection site.

**Nerve Blocks of the Digits**

The digital nerve block is one of the most useful and most used blocks in the emergency department. Indications for choosing it include repair of finger lacerations and amputations, reduction of fractures and dislocations, drainage of infections, removal of fingernails, and relief of pain (e.g., from a fracture or burn). Most emergency physicians (99%) use this block in their practice; 82% use it frequently. [1]

The digital block is superior to local infiltration in most circumstances. Wound infiltration may be a problem in the finger that has tight skin and can accept only a limited volume of anesthetic. Administration of anesthetic into this restricted space increases the tissue pressure, impairing capillary blood flow and causing pain. Fibrous septa in the fingertip also restrict the space available for the injected substance and even limit the spread of small amounts of anesthetic.

**Anatomy.**

Each finger is supplied by 2 sets of nerves. These nerves, the dorsal and palmar digital

nerves, run alongside the phalanx at the 2 and 10 o'clock positions and the 4 and 8 o'clock positions, respectively (Fig. 32-11 (Figure Not Available) *A-D*). Many physicians mistakenly believe that all 4 nerves must always be blocked to obtain adequate anesthesia.

The principal nerves supplying the finger are the palmar digital nerves, also called the *common digital nerves*. These nerves originate from the deep volar branches of the ulnar and median nerves, where they branch in the wrist. The palmar digital nerves follow the artery along the volar lateral aspects of the bone, one on each side, and supply sensation to the volar skin and interphalangeal joints of all 5 digits. In the middle 3 fingers, these nerves also supply the dorsal distal aspect of the finger, including the fingertip and nailbed. Only the volar branches must be blocked to obtain

**Figure 32-10** Distribution, anatomy, and blockade of the radial nerve at the wrist.

adequate anesthesia of these fingers distal to the distal interphalangeal joint.

The dorsal digital nerves originate from the radial and ulnar nerves, which wrap around to the dorsum of the hand. *They supply the nailbeds of the thumb and small finger and the dorsal aspect of all 5 digits up to the distal interphalangeal joints*. Unlike the middle 3 fingers, all 4 nerves are usually blocked in the thumb and fifth finger, especially to obtain anesthesia of the fingertip and nailbed (Fig. 32-12 (Figure Not Available) *A* and *B*).

**Technique.**

The digital nerves can be blocked anywhere in their course, including sites in the finger, in the web space between the fingers, and between the metacarpals in the hand. There are a variety of approaches to the nerves, including the dorsal and palmar approaches and the web space approach. Each has its merits. The technique is similar at each level.

The dorsal approach has the advantage of thinner, less pain-sensitive skin compared with volar approaches. The hand can be held firmly and flat on the table, preventing withdrawal. The disadvantage is that 2 injections are needed from this approach to block both volar digital nerves.

The dorsal approach can be used in the dorsum of the hand at the metacarpals, just proximal to the finger webs at the proximal end of the proximal phalanx, or distal to the web. Clinical situations may dictate which site to use; however, given equal circumstances, the preferred site is just proximal to the finger web. Here the nerve's location is more consistent than in the hand, and there is more soft tissue space to accommodate the volume of injected substance than there is in the distal finger. Digital block at the web is more efficacious in onset and requires less time to anesthesia than metacarpal block done proximal to the metacarpophalangeal joint. [15]

Digital block requires only aseptic injection technique after the skin is prepared with

alcohol or povidone-iodine. Sterile gloves and drapes are not necessary, although examination gloves are recommended.

Anesthesia is deposited at the positions of the 4 digital nerves (2, 4, 8, and 10 o'clock in relationship to the bone), using a 3.75-cm, 25- or 27-ga needle (see Fig. 32-11 (Figure Not Available) *A*). The block is performed from the dorsal surface, where the skin is thinner, easier to penetrate, and less sensitive than that of the volar surface. The needle insertion site is at the web space, just distal to the knuckle at the lateral edge of the bone (Fig. 32-12 (Figure Not Available) *C*). Once the needle tip is subdermal, it usually contacts the bone. A wheal of 0.5 to 1.0 mL of anesthetic without epinephrine is injected at this level. This serves to block the dorsal digital nerve and provide anesthesia at the injection site. The needle is then passed lateral to the bone and toward the palmar surface until the palmar skin starts to tent slightly. The needle is withdrawn 1 mm and aspirated for checking for an inadvertent IV position, and 0.5 to 1.5 mL of anesthetic are injected. This procedure is repeated on the opposite side of the finger. The result is a circumferential band of anesthesia at the base of the finger. *Firm massage of the injected area* for 15 to 30 seconds enhances diffusion of the anesthetic through the tissue to the nerves.

A variation of the dorsal approach is performed as follows: after injecting 1 side of the finger, the needle is redirected (without removing it) across the top of the digit to anesthetize the skin on the opposite side (Fig. 32-12 (Figure Not Available) *D*). The needle is then withdrawn and inserted at the site that was anesthetized, and the block is continued as described earlier. The presumed advantage of this method is that it minimizes the pain of the second skin puncture. This procedure requires that the needle be placed across the dorsal aspect of the finger, allowing the possible disadvantage of extensor tendon puncture and trauma.

The *palmar* and *web space* approaches can be used most successfully for the middle 3 fingers where a single puncture is required to block both volar nerves. This technique takes advantage of the anatomic fact that only the volar digital nerves must be blocked to obtain anesthesia of the total finger (except the proximal dorsal surface). If the thumb or fifth finger must be anesthetized, the dorsal branches must also be blocked to obtain anesthesia of the fingertip and fingernail area.

The palmar approach requires an injection in the palm, which is slightly more painful than an injection in the dorsal skin. The needle is inserted directly over the center of the metacarpal head, and anesthetic is slowly injected while the needle is advanced to the bone. At this point the needle is withdrawn 3 to 4 mm and angled slightly to the left and right of center to block both digital nerves without withdrawing the needle (Figs. 32-13 and 32-14) . To be successful, a palpable soft tissue fullness should be appreciated. The technique requires 4 to 5 mL of anesthetic.

A variation of this is particularly useful in fingertip injuries in toddlers. The finger is pinched side-to-side just distal to the proximal finger crease. This tents the skin at the finger crease, which is injected subcutaneously with 0.5 to 1.0 mL of anesthetic. This single injection diffuses to the volar nerves and provides anesthesia to many fingertip injuries common in toddlers. With the web space approach, the patient's hand is held by

the physician in such a way that the physician's thumb and index finger are over the dorsal and volar metacarpal head, respectively. The physician's third finger is used to separate the patient's fingers to expose the web space, while the fourth and fifth fingers support the finger being anesthetized (Fig. 32-15) . The needle is inserted into the web space, 1 mL of anesthetic is injected, and the needle is slowly advanced until it is next to the lateral volar surface of the metacarpal head. Only 2 mL of anesthetic are injected, and the needle is advanced slowly past the midline of the metacarpal head to the opposite digital nerve (Fig. 32-16) . The operator's index finger is used to palpate a fullness as the anesthetic is injected. By redirecting the needle to the adjacent finger without withdrawing it, both fingers may be blocked with a single puncture.

The anesthetic solution should be 2 to 4 mL of anesthetic *without epinephrine*. The total amount is reduced if the block is performed in the finger itself. The injection should go in smoothly, without resistance of the syringe plunger. Although the finger is forgiving of transient pressure from excessive anesthetic, if the injection site becomes excessively tense as the volume is injected, the amount injected should be limited. If epinephrine-containing solutions are inadvertently used for a digital block of the digits in otherwise healthy individuals without peripheral vascular disease, it is unlikely that serious ischemic injury will occur. Significant vasoconstriction will not last >60 minutes, within the time interval for which an ischemic tourniquet can safely be used in the same area. [16] However, if the entire digit remains blanched for >15 minutes, it is prudent to reverse the alpha-agonism of epinephrine with phentolamine (see Complications and Precautions subsection and Chapter 31) .

Onset of anesthesia occurs in 1 to 15 minutes and lasts for 20 minutes to 6 hours, depending on the anesthetic agent used.

**Alternative techniques**

**Jet injection technique.**

Jet injection for digital nerve block can be used effectively and is less painful than standard needle techniques. [17] The technique described by Ellis uses 0.15 mL of 1% lidocaine delivered by a jet injector at 2600 psi. Three injections are given to the lateral aspect of the proximal phalanx: the first, midway between the volar and dorsal surfaces; the second, dorsal to this; the third, volar. A total of 0.45 mL is administered to each side of the phalanx at the 2, 3, and 4 o'clock positions and the 8, 9, and 10 o'clock positions in relationship to the bone.

The potential disadvantages of jet injection include lacerations that may occur with tangential injection. Holding the injector perpendicular to the skin avoids this problem. Thick skin associated with older age, manual labor, and male gender may require larger volumes of anesthetic.

The advantages of this technique are less pain of injection and avoidance of "needle phobia," particularly in children.

**Transthecal digital block technique.**

The transthecal block is performed by a single injection into the flexor tendon sheath, which produces rapid and complete finger anesthesia. It was first described by Chiu in 1990 after noting rapid finger anesthesia after injection treatment of a trigger finger. [18] Cadaver studies suggest that injected fluid diffuses out of the tendon sheath and around the phalanx and all 4 digital nerves.

The flexor tendon is palpated in the palm proximal to the metacarpal phalangeal joint. A 25-ga needle is introduced at a 45° angle and advanced to the sheath/tendon. Slight pressure is applied to the syringe plunger. If the sheath has been entered, the anesthetic should flow freely. If it does not, it is presumed that the tendon has been entered, and the syringe is withdrawn slowly while constantly applying slight pressure. A total of 2 mL of 1% lidocaine is injected. Smaller volumes are used in children. After the needle is removed, pressure is applied over the tendon proximally to facilitate distal spread. Average onset of anesthesia is 3 minutes. [19]

The advantage of this technique is the single injection. However, Hill and colleagues found the technique to be "clinically equal" to traditional digital blocks. [20] Indeed, they found that patients judged the technique to be slightly more painful and it took slightly longer than the traditional method. Theoretically, the technique may increase the risk of injury to the tendon.

**Complications and precautions.**

The small size of the digital arteries and nerves makes intravascular or intraneural injection less likely. Inadvertent intravascular injection may cause digit ischemia from vasospasm or displacement of blood out of the capillary bed by the anesthetic. Blanching of the finger as the anesthetic is injected suggests intravascular

**Figure 32-17** (Figure Not Available) Cutaneous distribution of the nerves to the lower extremity. *(From Bridenbaugh PO: The lower extremity: Somatic blockade. In Cousins M, Bridenbaugh PO (eds): Neural Blockade in Clinical Anesthesia and Management of Pain. 2nd ed. Philadelphia, JB Lippincott, 1988, p 425. Reproduced with permission.)*

injection. If this is observed, the injection should be discontinued. Usually the ischemia is transient and self-resolving, and serious complications are rare. Massage or topical application of nitroglycerin paste may be attempted if ischemia persists. Vasoconstrictors such as epinephrine are best avoided when blocking the digital nerves. Although the incidence of vasospasm and resultant ischemia is rare and primarily occurs in patients with underlying vascular disease, [21] this complication is easily avoided by the choice of anesthetic agent. As noted above, if one mistakenly chooses an epinephrine-containing solution and vasospasm develops, the ischemia should be relieved with local infiltration of phentolamine (see Chapter 31) .

Commonly the digital nerve is lacerated or damaged by the initial injury to the finger. Careful evaluation using 2-point discrimination should be performed to determine the

extent of nerve injury before nerve block. Even if nerve injury is questionable, it should be documented in the chart, and the *patient should be advised of the injury before the nerve block.* Careful evaluation and patient education should prevent misconceptions as to the cause of the nerve injury. Although most isolated digital nerve injuries are not debilitating, they heal slowly and can be annoying to the patient. Digital nerve injury proximal to the distal interphalangeal joint may be repaired surgically. Nerve repair may be immediate when specialty consultation is available or delayed following initial simple closure.

## Nerve Blocks of the Lower Extremity

The lower extremity is supplied by 5 nerves whose branches can be blocked at the hip, knee, ankle, foot, or toes. As in the arm, proximal nerve blocks in the leg are seldom used in the emergency department. Nerve blocks at the ankle are used occasionally in the treatment of foot lacerations and to perform otherwise painful procedures on the foot. Metatarsal and digital blocks in the foot are used frequently to treat ingrown toenails, fractures, and lacerations of the forefoot and toes.

### Nerve Blocks of the Hip

Nerve blocks of the femoral, obturator, lateral femoral cutaneous, posterior cutaneous, and sciatic nerves at the hip provide anesthesia and paralysis to the skin and muscles of the leg (Fig. 32-17) (Figure Not Available) . Posteriorly, the posterior cutaneous and sciatic nerves can be blocked together with a single injection. Anteriorly, the femoral, obturator, and lateral femoral cutaneous nerves can be blocked by 3 separate injections or by a single injection using the 3-in-1 technique described by Winnie and colleagues. In this technique a large amount of anesthetic is injected into the femoral nerve sheath and permitted to track into the pelvis to the point at which the anterior nerves run in a common sheath. [22]

Proximal lower extremity nerve blocks are rarely used in the emergency department. Injuries to the thigh and calf that call for extensive repair and anesthesia often require a specialist and repair in the operating room. Injuries to the ankle and foot are more easily anesthetized at that level. Only 3% of emergency physicians use blocks at the hip on occasion. [1]

Femoral nerve blocks alone may be the analgesia of choice for femoral shaft fractures. Studies supporting the femoral block in the treatment of femoral shaft fractures date back to 1940 and continue to appear. [23] [24] [25] In a study by McGlone and colleagues, patients preferred femoral block to opioid analgesia; however, this means of analgesia has yet to be adopted as routine treatment for femoral shaft fractures. [24] The procedure requires only femoral nerve blockade and does not rely on the performance of a successful 3-in-1 block.

#### Anatomy.

The leg is supplied by 5 nerves. Anteriorly, the femoral, obturator, and lateral femoral

cutaneous arise from the lumbar plexus and travel a short distance in a common nerve sheath (Fig. 32-18) (Figure Not Available) . In the pelvis, the lateral femoral cutaneous and obturator nerves exit the sheath. The lateral femoral cutaneous nerve supplies the sensory innervation to the lateral thigh. The obturator nerve supplies the sensory innervation to the anterior medial thigh and the motor supply to the adductor muscles. The femoral nerve continues as the saphenous nerve, which supplies the sensory innervation to the medial calf and ankle.

Posteriorly, the sciatic and posterior cutaneous nerves arise from the lumbar and sacral plexuses and exit the pelvis together in the sciatic notch. The posterior cutaneous nerve supplies sensory innervation to the posterior thigh and buttocks. The sciatic nerve does not supply the thigh but at the knee divides into the tibial and peroneal nerves and supplies much of the calf and foot. The tibial nerve supplies the muscles of the calf and sensation to the plantar surface of the foot. The peroneal nerve supplies sensation to the lateral aspect of the calf and dorsal and plantar aspects of the foot. Both the tibial and peroneal nerves give off branches that form the sural nerve, which supplies sensation to the lateral aspect of the foot (Fig. 32-19) (Figure Not Available) .

**Technique for femoral nerve block.**

With the patient in the supine position, the skin is prepared in an aseptic fashion. The femoral artery is palpated 1 to 2 cm below the inguinal ligament (see Fig. 32-18) (Figure Not Available) , and a wheal of anesthesia is raised immediately lateral to the artery. With the nondominant

**Figure 32-18** (Figure Not Available) Anatomy of the femoral, obturator, and lateral femoral cutaneous nerves and the location of the injection site for the femoral and 3-in-1 nerve blocks. *(Adapted from Winnie AP, Ramamurthy S, Durrani Z: The inguinal paravascular technique of lumbar plexus anesthesia: The "3 in 1 block." Anesth Analg 52:989, 1973. Reproduced with permission.)*

**Figure 32-19** (Figure Not Available) *A* and *B,* Anatomy and innervation of the calf and foot. *(From Shurman DH: Ankle-block anesthesia for foot surgery. Anesthesiology 44:348, 1976. Reproduced with permission.)*

hand placed firmly on the artery, a 1.25-cm, 22-ga needle, attached to an extension tube setup and a 20-mL syringe, is inserted adjacent to the artery at a 90° angle to the skin and underlying vessels. The needle is advanced until a paresthesia is elicited or the needle pulsates laterally, indicating a position immediately adjacent to the artery. If a paresthesia is felt or the needle is assumed to be in the immediate vicinity of the nerve, 10 to 20 mL of anesthetic are injected. If a paresthesia is not elicited, 10 to 20 mL of anesthetic can be injected in a fan-like pattern lateral to the artery in a blind attempt to anesthetize the femoral nerve. The onset of anesthesia should occur in 15 to 30 minutes and should last for 3 to 8 hours.

**Technique for the 3-in-1 block.**

The 3-in-1 technique differs from the femoral nerve block in that it requires a larger amount of anesthetic solution and compression distal to the injection site. In the thigh,

the femoral nerve is sandwiched between the quadratus lumborum and iliopsoas muscles, whose fasciae are continuous with a nerve sheath that contains all 3 nerves higher in the pelvis. A large amount of anesthetic properly placed next to the femoral nerve tracks back along the sheath and anesthetizes all 3 nerves.

The 3-in-1 block requires precise injection of the anesthesia into the nerve sheath. In the 3-in-1 block, once the needle is in the correct position, the fingers palpating the artery are removed and placed gently but firmly distal to the needle while 20 to 30 mL of anesthetic are injected. Distal pressure should be maintained for 5 minutes while the anesthetic diffuses proximally.

A nerve stimulator facilitates the search for the nerve and placement of the needle tip in the fascial sheath. The nerve is covered by a thick insulating fascia. When the needle punctures this sheath, the nerve is stimulated at currents of 0.5 mA or less.

**Precautions.**

The femoral nerve block requires that large amounts of anesthetic be injected close to large nerves and vessels. Standard precautions should be used to avoid intravascular and intraneuronal injection, and recommended anesthetic dosages should be observed.

**Nerve Blocks of the Ankle**

Nerve block of the 5 nerves of the ankle--the deep peroneal (anterior tibial), posterior tibial, saphenous, superficial peroneal (musculocutaneous), and sural nerves--provides anesthesia to the foot. Depending on the desired area of anesthesia, 1 or more of the 5 nerves are blocked. These blocks can be used in operative procedures and repair of injuries to the foot. They are particularly useful in providing anesthesia to the sole of the foot for laceration repair and foreign body removal.

A nerve block of the foot is better tolerated by the patient than local infiltration in all but the most minor procedures. The skin of the sole is thicker and more tightly bound to the underlying fascia by connective tissue septa than is skin in other parts of the body. Puncturing this skin can be difficult and is always quite painful. The fibrous septa can limit the amount and spread of anesthetic. If large amounts of anesthesia are injected, the volume of injected

substance quickly exceeds the space available, possibly leading to painful distention of the tissue and circulatory compromise of the microvasculature. Local infiltrative anesthesia is adequate for treating minor injuries in which only small amounts of anesthetic are needed. For treatment of larger injuries, including incision and drainage, extensive wound care, and foreign body removal, the ankle block is better tolerated.

**Anatomy.**

The foot is supplied by the 5 nerve branches of the principal nerve trunks (see Fig. 32-17) (Figure Not Available) . Three nerves are located anteriorly and supply the dorsal

aspect of the foot. Two nerves are located posteriorly and supply the volar aspect.

The anteriorly located nerves are the superficial peroneal, deep peroneal, and saphenous nerves. The superficial peroneal nerve (also called the *dorsal cutaneous* or *musculocutaneous nerve*) actually consists of multiple branches that supply a large portion of the dorsal aspect of the foot (see Fig. 32-19) (Figure Not Available) . These are located superficially between the lateral malleolus and extensor hallucis longus tendon, which is easily palpated by having the patient dorsiflex the big toe. The deep peroneal nerve (also called the *anterior tibial nerve*) supplies the web space between the big and second toes. In the ankle, it lies under the extensor hallucis longus tendon. The saphenous nerve runs superficially with the saphenous vein between the medial malleolus and tibialis anterior tendon, which is prominent when the patient dorsiflexes the foot. The saphenous nerve supplies the medial aspect of the foot near the arch.

The posteriorly located nerves are the posterior tibial and sural nerves. The sural nerve runs subcutaneously between the lateral malleolus and the Achilles tendon and supplies the lateral border, both volar and dorsal, of the foot (see Fig. 32-19) (Figure Not Available) . The posterior tibial nerve runs with the posterior tibial artery, which can be palpated between the medial malleolus and the Achilles tendon. It lies slightly deep and posterior to the artery.

The posterior tibial nerve is one of the major nerve branches to the foot. After passing through the ankle, it branches into the medial and lateral plantar nerves, which supply sensation to most of the volar aspect of the foot and toes and supply motor innervation to the intrinsic muscles of the foot.

**Technique.**

Complete nerve block of the foot requires blocking 3 SQ nerves and 2 deeper nerves (Fig. 32-20) (Figure Not Available) . Once familiar with the anatomy, the experienced physician can anesthetize all 5 nerves quickly by placing SQ band blocks around 75% of the ankle circumference and 1 deep injection next to the palpable posterior tibial artery and the other under the extensor tendon of the big toe.

The 5 nerves of the foot are commonly blocked in combinations of 2 or more. Small procedures clearly within the distribution of 1 nerve may require only a single nerve block; however, overlap of the nerve's sensory distribution frequently necessitates blocking a number of nerves for adequate anesthesia. Nerve block of the sural and posterior tibial nerves together anesthetizes the bottom of the foot and is the most useful combination.

**Posterior tibial.**

The posterior tibial nerve is blocked in the medial aspect of the ankle between the medial malleolus and the Achilles tendon. The injection site is determined by palpating the tibial artery just posterior to the medial malleolus. A point 0.5 to 1.0 cm superior to this is marked. If the artery is not palpable, a site 1 cm above the medial malleolus and

just anterior to the Achilles tendon is used (see Figs. 32-20 (Figure Not Available) and 32-21) (Figure Not Available) .

A 3.75-cm, 25-ga needle is directed at a 45° angle to the mediolateral plane (the needle is almost perpendicular to the skin), just posterior to the artery. At the estimated depth of the artery, approximately 0.5 to 1.0 cm deep, the needle is wiggled slightly in an effort to produce a paresthesia. If the paresthesia is elicited, 3 to 5 mL of anesthetic are injected after careful aspiration to check for inadvertent intravascular placement of the needle tip. If no paresthesia is produced, the needle is advanced inward, again at a 45° angle, until it hits the posterior aspect of the tibia. The needle is then withdrawn slightly, about 1 mm, and 5 to 7 mL of anesthetic are injected while the needle is withdrawn another 1 cm. A rise in the temperature of the foot, due to vasodilation from loss of sympathetic tone, may herald a successful block. This finding may be used as an indicator of onset and depth of anesthesia.

**Sural nerve.**

The sural nerve is blocked on the lateral aspect of the ankle between the Achilles tendon and the lateral malleolus (see Fig. 32-20) (Figure Not Available) . It lies superficially and is blocked at a level about 1 cm above the lateral malleolus. A band of anesthesia is injected subcutaneously between the Achilles tendon and the lateral malleolus using 3 to 5 mL of anesthetic.

**Superficial peroneal nerves.**

The superficial peroneal nerves are blocked on the anterior aspect of the ankle between the extensor hallucis longus tendon and the lateral malleolus. They lie superficially and are blocked using 4 to 10 mL of anesthetic placed subcutaneously in a band between these landmarks.

**Deep peroneal nerve.**

The deep peroneal nerve is blocked anteriorly beneath the extensor hallucis tendon (see Fig. 32-20) (Figure Not Available) . It is blocked at a level 1 cm above the base of the medial malleolus and between the extensor hallucis longus and anterior tibial tendons. The tendons are palpated by having the patient dorsiflex the big toe and foot, respectively. After an SQ wheal is placed, the needle is directed about 30° laterally and under the extensor hallucis tendon until it strikes the tibia (at a depth of less than 1 cm). The needle is withdrawn 1 mm, and 1 mL of anesthetic is injected.

The saphenous nerve is blocked anteriorly between the medial malleolus and the anterior tibial tendon. It lies superficially and is blocked with 3 to 5 mL of anesthetic injected subcutaneously between these landmarks.

**Nerve Blocks of the Metatarsals and Toes**

Like the nerve blocks in the hand and fingers, the nerve blocks in the foot and toes are commonly used in the emergency department. Indications for using these blocks include repair of lacerations, drainage of infections, removal of toenails, manipulation of fractures and dislocations, and otherwise painful procedures requiring anesthesia to the forefoot and toes.

Digital nerve blocks in the foot and toes are superior to local infiltration anesthesia in all but the most minor procedures. In the toes, the limited SQ space does not accommodate enough injected material for adequate infiltrative anesthesia. Furthermore, the fibrous septa, which attach the volar skin to the underlying fascia and bone, limit the spread and volume of injected substances. On the plantar surface, even small amounts of local infiltrate can cause painful distention and local ischemia of the tissues.

**Anatomy.**

Each toe is supplied by 2 dorsal and 2 volar nerves. These nerves are branches of the major nerves of the ankle. The dorsal digital nerves are the terminal branches of the deep and superficial peroneal nerves. The volar nerves are branches of the posterior tibial and sural nerves.

The location of the nerves in relation to the bones varies with the site of the foot. In the toes, the nerves lie at the 2, 4, 8, and 10 o'clock positions in close relationship to the bone. In the proximal foot, the nerves run with the tendons and are not in close relationship to the bones (Fig. 32-22) (Figure Not Available) .

**Technique.**

The digital nerves can be blocked at the metatarsals, interdigital web spaces, or toes. The bones of the foot can be palpated easily from the dorsum and are used as the landmarks for estimating the location of the nerves. Proximally, the nerves' relationship to the bones is less consistent, making definitive needle placement and successful block less reliable. In the toes, the position of the nerves is more consistent; however, there is minimal SQ tissue space available for the injected solution. At the web space, the nerves are located in close relationship to the bone, and there is ample space for injecting the anesthesia; hence, for most procedures, the web space is the preferred site

for the digital nerve block.

The technique for toe and metatarsal blocks is similar. All 4 nerves supplying each toe are usually blocked because of their sensory overlap. The blocks are performed from the dorsal surface, where the skin is thinner and less sensitive than that on the plantar aspect. A total of 5 mL of anesthetic is deposited in a fan-like pattern in the space between the metatarsal bones (see Fig. 32-22) (Figure Not Available) . A 1-mL skin wheal is placed dorsally between the metatarsal bones. The needle is then advanced until the volar skin tents slightly, and 2 mL are injected as the needle is withdrawn. Without removing it, the needle is redirected in a different volar direction, and

**Figure 32-22** (Figure Not Available) Anatomy and technique for digital nerve block at the metatarsals. *(From Bridenbaugh PO: The lower extremity: Somatic blockade. In Cousins M, Bridenbaugh PO (eds): Neural Blockade in Clinical Anesthesia and Management of Pain. 2nd ed. Philadelphia, JB Lippincott, 1988, p 437. Reproduced with permission.)*

the procedure is repeated. A total of 5 mL is used in each metatarsal space. Again, because of sensory overlap, 2 or more spaces need to be anesthetized for each toe to be blocked.

For the web space block, a site on the dorsum just proximal to the base of the toe is selected. Using a 10-mL

**Figure 32-23** Technique of digital nerve blocks in the interdigital web spaces of the foot.

syringe, a 3.75-cm, 27-ga needle is inserted at the lateral edge of the bone (Fig. 32-23) . A wheal is placed subcutaneously between the skin and bone, using 0.5 to 1.0 mL of anesthetic. This serves to block the dorsal nerve and minimize pain at the needle insertion site. The needle is then advanced just lateral to the bone and toward the sole until

**Figure 32-24** (Figure Not Available) Alternative techniques of digital nerve blocks in the toes. *(From Locke RK, Locke SE: Nerve blocks of the foot. JACEP 4:698, 1976. Reproduced with permission.)*

the needle tents the volar skin slightly. The needle is withdrawn 1 mm, and 0.5 to 1.0 mL is injected. As the needle is withdrawn, another 0.5 mL is injected to ensure a successful block. The procedure is repeated on the opposite side of the toe. In this manner, 2 columns of anesthesia are placed on each side of the toe in the area through which the 4 digital nerves run. A total of 2 to 4 mL of anesthetic is used. For blocks done in the toe itself, the procedure is the same, but smaller amounts of anesthetic are used because of the limited SQ space and fear of vascular compression. A total of less than 2 mL is used. Alternative techniques using a single injection site, as described for the finger, can be performed (Fig. 32-24) (Figure Not Available) .

**Complications and precautions.**

The precautions that apply to the hand and fingers apply to the foot and toes. Ischemic

complications can be avoided by paying attention to skin changes during the injection. Blanching heralds possible intravascular injection or vascular compression. If the skin blanches, halt the procedure and reevaluate the position of the needle and the amount and content of the injected solution. The total volume of anesthesia should not exceed the recommended amount. Although podiatrists commonly use anesthetics containing small concentrations of epinephrine for digital blocks in patients without underlying vascular disease, [16] [21] this practice is generally not recommended because of concern about vasospasm and ischemia (see Complications and Precautions subsection and Chapter 31) .

Note any neural or vascular injuries before the injection. The close proximity of these structures to the skin and bones means that they are frequently injured. Deficits, even if questionable, should be documented in the records and brought to the attention of the patient before the nerve block.

## CONCLUSIONS

Regional nerve blocks of the thorax and extremities are valuable adjuncts to the clinician's armamentarium. They may be used for wound anesthesia prior to exploration, irrigation, debridement, and repair. They may be used to reduce the pain of certain procedures (e.g., arthrocentesis, fracture reduction, or dislocation manipulation). A rib block may permit better thoracic mechanics. When these procedures are performed, the cautions outlined in Chapter 31 and the current chapter should be heeded.

# Chapter 33 - Regional Anesthesia of the Head and Neck

**James T. Amsterdam**

The use of intraoral and extraoral regional anesthesia is both simple and convenient. [1] [2] [3] Nerve blocks are used to attain anesthesia in areas of broad distribution in the face with a minimal amount of anesthetic and resultant tissue distortion. Local anesthetic blocks are effective for closing facial lacerations, especially those of the lips, the forehead, and the midface, where the swelling caused by local infiltration is undesirable. Local anesthetic blocks are also effective for the relief of pain, for anesthesia in debridement, and for diagnostic purposes.

Patients with dental pain who do not get relief with a regional dental block most likely do not have pain of dental origin. Regional blocks are also therapeutic for both surgical procedure anesthesia and pain control for dental emergencies such as toothaches and dry sockets (see Chapter 69) . In cases in which the patient is thought to be seeking drugs and one wishes to avoid narcotics, a dental anesthetic block is frequently the treatment of choice.

Topical anesthetic solutions such as tetracaine-Adrenalin-cocaine (TAC) solution are useful in small lacerations of the scalp and face because of the vascularity of these areas. TAC is not to be used on or near mucous membranes. Detailed information on the use of TAC appears in Chapter 31 . More extensive discussions of the general complications of local anesthetics and of regional anesthesia are provided in Chapters 31 and 32 , respectively. Ophthalmologic anesthesia is discussed in Chapter 67 . Blocks about the ears and nasal anesthesia are discussed in Chapter 68 .

The procedures and techniques described here generally carry a low morbidity. The supraperiosteal and mental nerve infiltrations can generally be learned through reading and experimentation; more sophisticated blocks (e.g., inferior alveolar block) are best learned under the instruction of an experienced physician, a dentist, or an oral and maxillofacial surgeon.

## ANATOMY OF THE FIFTH CRANIAL (TRIGEMINAL) NERVE

The fifth cranial nerve, the trigeminal nerve, is the sensory nerve to the face (Fig. 33-1 (Figure Not Available) *A*) and is the largest of the cranial nerves. It takes its origin from the midbrain and enlarges into the gasserian, or semilunar, ganglion. One gasserian ganglion supplies each side of the face. The gasserian ganglion is a flat, crescent-shaped structure approximately 10 mm long and 20 mm wide that divides into 3 branches: the ophthalmic, maxillary, and mandibular nerves (Fig. 33-1 (Figure Not Available) *B*).

**Ophthalmic Nerve**

The first division, the *ophthalmic nerve* (V1 ), is the smallest branch in the gasserian ganglion. It leaves the cranium through the superior orbital fissure and has 5 cutaneous branches. These branches are as follows:

1. The medial and lateral branches of the supraorbital nerve, which emerge on the face through the supraorbital notch. These two sensory nerves pierce the frontalis muscle and extend to the lambdoid suture on the back of the skull.
2. The supratrochlear nerve, which is sensory to the medial aspect of the forehead just above the glabella.
3. The infratrochlear nerve.
4. The lacrimal nerve.
5. The external nasal nerve.

In addition to being sensory to the forehead, branches of the ophthalmic nerve are sensory to the cornea, the upper eyelid, structures in the orbit, and the frontal sinuses.

**Maxillary Nerve**

The second division, the *maxillary nerve* (V2 ), is sensory to the maxilla and associated structures, such as the teeth, the periosteum and the mucous membranes of the maxillary sinus and the nasal cavity, the soft and hard palate, the lower eyelids, the upper lip, and the side of the nose. The second division exits the cranium from the foramen rotundum and ultimately enters the face through the infraorbital canal; it terminates as the infraorbital nerve. The infraorbital nerve gives sensory branches to the lower eyelids, the side of the nose, and the upper lip.

The anatomy of the maxillary nerve is rather complicated because of its numerous branches. The first branch comprises 2 short sphenopalatine nerves to the pterygopalatine ganglion, also called the *Meckel ganglion* or the *sphenopalatine ganglion.* The next 2 branches of clinical importance are the nasopalatine and the greater (anterior) palatine nerves. The nasopalatine nerve arises from the pterygopalatine ganglion, courses down along the nasal septum, and is transmitted through the anterior portion of the hard palate by way of the anterior palatine canal. This canal is located in the midline approximately 10 mm palatally to the maxillary central teeth and immediately behind the incisors. The nasopalatine nerve is sensory to the most anterior portion of the hard palate and the adjacent gum margins of the upper incisors. This nerve is rarely blocked in clinical practice, except in dental operations (Fig. 33-2 *A*).

The anterior, or great, palatine nerve arises from the pterygopalatine ganglion and passes down through the posterior palatine foramen. The posterior palatine foramen is located 10 mm palatally to the third molar and the bicuspid teeth and intermingles with the nasopalatine nerve opposite the cuspid tooth. The greater palatine nerve is sensory to most of the hard palate, as well as the palatal aspect of the gingiva. It is rarely blocked in the emergency department (Fig. 33-2 *B*).

The next branch consists of the posterosuperior alveolar (PSA) nerve, which courses down the posterior surface of the maxilla for approximately 20 mm, at which point it enters 1 or several small posterosuperior dental foramina. This nerve supplies all the roots of the third and second molar teeth and 2 roots of the first molar tooth. A third branch consists of the middle superior alveolar (MSA) nerve, which branches off about midway within the infraorbital canal and then courses downward in the outer wall of the maxillary sinus. This nerve supplies the maxillary first and second bicuspid teeth and the mesiobuccal root of the first molar. The last branch consists of the anterosuperior alveolar (ASA) nerve, which branches off into the infraorbital canal approximately 5 mm behind the infraorbital foramen, just before the terminal branches of the infraorbital nerve emerge. This nerve descends in the anterior wall of the maxilla to supply the maxillary central, lateral, and cuspid teeth; the labial mucous membrane; the periosteum; and the alveoli on 1 side of the median line. There is an intercommunication among the ASA, MSA, and PSA nerves.

**Mandibular Nerve**

The third division, the *mandibular nerve* (V3 ), is the largest branch of the trigeminal nerve. It exits from the cranium through the foramen ovale and divides into 3 principal branches:

1.  The long buccal nerve branches off just outside the foramen ovale. It passes between the 2 heads of the external pterygoid muscle and crosses in front of the ramus to enter the cheek through the buccinator muscle, buccally to the maxillary third molar. The buccal nerve supplies sensory branches to the buccal mucous membrane and the mucoperiosteum over the maxillary and mandibular teeth. The cutaneous branch is the sensory nerve to the cheek.
2.  The lingual nerve courses forward toward the midline. It runs downward superficially to the internal pterygoid muscle to pass lingually to the apex of the mandibular third molar. It enters the base of the tongue at this point through the floor of the mouth and supplies the anterior two thirds of the tongue, the lingual mucous membrane, and the mucoperiosteum.
3.  The largest of the V3 branches is the inferior alveolar nerve. It is sensory to all of the lower teeth, although the central and lateral incisors and the buccal aspect of the molar teeth may receive additional sensory innervation. The nerve descends, covered by the external pterygoid muscle, and passes between the ramus of the mandible and the sphenomandibular ligament to enter the mandibular canal. It is accompanied by the inferior alveolar artery and vein and proceeds along the mandibular canal, innervating the teeth. At the mental foramen, the nerve bifurcates into an incisive branch, which continues forward to supply the anterior teeth. It gives off a side branch, the mental nerve, which exits from the mental foramen to supply the skin. The mental foramen is located approximately between the apices of the lower first and second bicuspids, or premolar teeth. This is a useful site at which to perform a nerve block, because the mental nerve is sensory to the integument of the chin and the skin and the mucous membrane of the lower lip.

# EQUIPMENT FOR DENTAL AND CRANIAL NERVE BLOCKS

One may easily give extraoral injections with standard injection equipment. Intraoral local anesthesia is conveniently administered with a Monoject aspirating dental syringe, which uses Carpule cartridges of anesthetic and disposable needles (Fig. 33-3) . A needle no smaller than 27 ga is recommended for deep block techniques due to the inability to perform aspiration with smaller needles. Generally, a long needle is used for block techniques, and a short needle is used for infiltrations.

The needle is screwed to the hub of the Monoject syringe, which in turn is attached to an adapter; the adapter may be removed for cleaning. When removing the disposable needle, one must take care not to remove and discard the adapter as well; this would render the syringe functionless (Fig. 33-4) . One pulls back the end of the syringe on its

**Figure 33-3** Local anesthesia--basic setup for intraoral application using an aspirating dental syringe.

spring, allowing room for the Carpule cartridge of anesthetic to be inserted (Fig. 33-5 *A*). The metal end of the Carpule cartridge is inserted, which engages the needle (Fig. 33-5 *B*). The handle of the syringe is then released and tapped to engage a barb into the rubber stopper of the cartridge (Fig. 33-5 *C*). One may then perform simple aspiration by retracting the handle, pulling on the rubber stopper within the Carpule.

To discard a Carpule cartridge, one should leave the needle in place on the syringe. The handle of the syringe is then withdrawn rapidly, disengaging the barb. If the needle has been removed, great care must be taken, because the negative pressure created in the Carpule cartridge upon withdrawal of the barb may cause shattering. Other adjuncts that are helpful in the administration of intraoral anesthesia include topical local anesthetic agents such as gels or sprays. It should be noted that dental syringes are not mandatory for intraoral local anesthesia but do make the procedure more simple. Reusable glass and disposable plastic aspirating syringes that do not use dental Carpule cartridges are also available.

The anesthetic agent most frequently used is 2% lidocaine with a vasoconstrictor, such as 1:100,000 or 1:50,000 epinephrine. Many other anesthetic agents, such as mepivacaine (Carbocaine) and Cetacaine (a combination of benzocaine, tetracaine, butamben, and benzalkonium), with or without vasoconstrictor agents, are also available. Bupivacaine (Marcaine) is a longer acting anesthetic that is often ideal for the procedures performed in the emergency department. When this agent is not available in Carpule cartridge form, a Carpule cartridge containing a different anesthetic may be emptied and bupivacaine drawn up in the evacuated cartridge. Because of the rich vascularity of the oral cavity, vasoconstrictors are important in sustaining the duration of anesthesia and should be used wherever possible in the absence of medical contraindications. Buffering with bicarbonate is not recommended for oral anesthesia.

## GENERAL PRECAUTIONS

Needles no smaller than 27 ga should be used for block techniques, since a higher gauge makes aspiration difficult, with resultant inadvertent intravascular injection. When an intraoral block procedure is performed, the needle should not be inserted to its full length at the hub. Should inadvertent breakage occur in such a situation, needle retrieval may be difficult. Furthermore, the direction of a needle should not be changed while the needle is deep in the tissue. Topical anesthetics can be placed on mucous membranes to make needle puncture painless. One should inject slowly to minimize pain and should always aspirate before injection. A warmed anesthetic solution is also more comfortable for the patient.

An important precaution for intraoral local anesthesia is that the injection should not be made into or through an infected area. This is especially important in inferior alveolar nerve blocks, in which tracking of an infection can be serious and difficult to treat. Trismus with inadequate oral access or direct extension of infection to parapharyngeal spaces can result. Therefore, local anesthesia should be only superficial before incision and drainage, unless a block can be performed far proximal to the site of infections.

## TECHNIQUE

### Topical Anesthesia

Most patients fear dental blocks greatly, and the anxiety and pain may be lessened considerably with the use of topical anesthetics that are applied to the mucous membranes before injection. The area to be injected is first thoroughly dried with gauze. A cotton-tipped applicator is generously coated with 20% benzocaine (Hurricaine, Beutlich, Inc., Niles, Ill.) or 5% to 10% lidocaine. Anesthesia results in 2 to 3 minutes. Note that rather concentrated topical anesthetics must be used, and poor results may be obtained with weaker preparations such as 2% viscous lidocaine.

### Supraperiosteal Infiltrations

The most common technique for intraoral local anesthesia of individual teeth is the supraperiosteal infiltration injection. This technique may supply complete relief of a toothache and is a useful emergency department procedure that can provide non-narcotic analgesia in the middle of the night. The area to be anesthetized is selected and dried with gauze. A topical anesthetic, such as 20% benzocaine or 5% lidocaine ointment, is applied as before. The mucous membrane of the area is grasped with a piece of gauze; the gauze is pulled out and downward in the maxilla and out and upward in the mandible to extend the mucosa fully and to delineate

the mucobuccal fold. The mucobuccal fold is then punctured with the bevel of the needle facing the bone. The area is aspirated, and approximately 1 to 2 mL of local anesthetic are deposited at the apex (area of the root tip) of the involved tooth (Fig. 33-6) (Figure Not Available) . It is helpful to place a finger against the *outer* aspect of the

lip overlying the injection site and apply firm and steady pressure against the lip as the local anesthetic is *slowly* injected into the supraperiosteal site.

The purpose of the injection is to deposit the anesthetic near the bone that supports the tooth. Because the anesthetic must penetrate the cortex of bone to reach the nerve of the individual tooth, the injection may fail if the solution is deposited too far from the periosteum, if the needle is passed too far above the roots of the teeth, or if the bone in the area is unusually thick or dense. If anesthesia is unsuccessful, one may also inject the palatal side. It may take 5 to 10 minutes to achieve full anesthesia with this technique, and the procedure may not be as effective for the posterior molars. Infiltration of the area around the maxillary canine and the first premolars will anesthetize the MSA and ASA nerves; lacerations of the upper lip can be treated by bilateral injection in the canine fossa areas.

## Infraorbital Nerve Block

### Anatomy

The infraorbital nerve block injection can be used to anesthetize the midface (Fig. 33-7) (Figure Not Available) . A solution of local anesthetic deposited at the infraorbital foramen anesthetizes not only the middle and superior alveolar nerves, but also the main trunk of the infraorbital nerve that innervates the skin of the upper lip, the skin of the nose, and the lower eyelid. The nasal mucosa is not anesthetized by this technique.

The infraorbital foramen is difficult to palpate extraorally and almost impossible to feel in the presence of facial swelling. It is found on the inferior border of the infraorbital ridge on a vertical (sagittal) line with the pupil when the patient stares straight ahead. Although 1 volunteer study found similar patient pain scale scores and overall preference in subjects receiving both intraoral and extraoral approaches, the intraoral approach seemed to provide nearly twice the duration of anesthesia. [4]

### Intraoral Approach

A topical anesthetic (e.g., 4% cocaine or 2% lidocaine) on a cotton-tipped swab is applied for 60 seconds prior to introducing the needle for the nerve block. [4] [5] When performing the intraoral approach, one keeps the palpating finger in place. The cheek is retracted, as in the supraperiosteal injection, and puncture is made in the mucosa opposite the upper second bicuspid (premolar tooth) approximately 0.5 cm from the buccal surface (Fig. 33-8 *A* and *B*). Topical anesthetic applied to the mucosa before injection decreases pain. The needle should be directed parallel with the long axis of the second bicuspid until it is palpated near the foramen, a depth of approximately 2.5 cm. If the entry is too acute initially, one will encounter the malar eminence before approaching the infraorbital foramen. In addition, if the needle is extended too far posteriorly and superiorly, the orbit may be entered (Fig. 33-8 *C*). Therefore, the procedure should be halted if the physician is unsure of the location of the needle or if patient cooperation is unsatisfactory.

When the location has been determined and aspiration has been performed, 2 to 3 mL of solution are injected *adjacent to, but not within, the foramen*. A finger should be held firmly on the inferior orbital rim to avoid ballooning of the lower eyelid with anesthetic solution. If one is not certain of the exact location of the infraorbital foramen, one may obtain anesthesia by performing a field block. For the latter technique, 5 mL of the anesthetic solution are infiltrated in a fan-like distribution in the upper buccal fold. This technique is not as precise as a discrete nerve block but usually produces the same effect. Massage of the tissue for 10 to 15 seconds following the injection also hastens onset of the anesthesia.

### Extraoral Approach

The infraorbital foramen may also be approached from an extraoral route (Fig. 33-9) (Figure Not Available) . The extraoral approach, of course, requires external preparation of the skin. In the extraoral approach, similar landmarks are used to locate the infraorbital foramen. The needle can be felt to pass through the skin, the subcutaneous (SQ) tissue, and the quadratus labii superioris muscle. After injection, the infiltrated tissue, usually visibly swollen, should be firmly massaged for 10 to 15 seconds.

Care must be taken not to anesthetize the facial artery and vein, because these may lie on either side of the needle. Vasoconstrictors should be avoided with this technique to avoid vasoconstriction of the facial artery. If severe blanching of the face occurs, warm compresses should be applied to the face immediately. Local phentolamine also may be administered (see Chapter 32) .

## Inferior Alveolar Nerve Block

In the setting of extreme dental pain, the emergency physician may find the use of the inferior alveolar nerve block and the lingual nerve block useful. This injection is somewhat more difficult than the other techniques described, and the emergency physician is advised to view demonstrations of this procedure before attempting it. The inferior alveolar nerve block provides anesthesia to all of the teeth on that side of the mandible and desensitizes the lower lip and the chin via block of the mental nerve. This technique is primarily useful for anesthetizing patients who have sustained severe dentoalveolar trauma; those with complaints of postextraction pain, dry socket, or pulpitis (toothache); or those with periapical abscess.

### Anatomy

The anatomy of the region should first be reviewed (Fig. 33-10 *A*). The patient can be seated either in a dental chair or upright with the occiput firmly against the back of the stretcher, so that when the mouth is opened, the body of the mandible is parallel to the floor. The physician should be ready for an unexpected quick jerk of the head when the anxious patient first feels the needle, despite the use of topical anesthesia. The physician stands on the side *opposite* the one being injected.

The technique first involves palpation of the retromolar fossa with the index finger or thumb. With this maneuver, the greatest depth of the anterior border of the ramus of the mandible (the coronoid notch) may be identified (Fig. 33-10 *B* and *C*). With the thumb in the mouth and the index finger placed externally behind the ramus (Fig. 33-10 *D* and *E*), the tissues are retracted toward the buccal (cheek) side, and the pterygomandibular triangle is visualized (Fig. 33-10 *F*). This technique also moves the operator's finger safely away from the tip of the needle.

**Approach**

The mucosa over the area to be injected may be coated with a topical anesthetic. When topical anesthesia has been obtained, the syringe should be held parallel to the occlusal surfaces of the teeth and angled so that the barrel of the syringe lies between the first and second premolars on the opposite side of the mandible (Fig. 33-10 *G* and *H*). If a large-barrel syringe is used, the corner of the mouth may hamper efforts to obtain the proper angle. The angle is facilitated by carefully bending the 25-ga needle about 30°. Puncture is made in the triangle, at a point that is 1 cm above the occlusal surface of the molars. If the needle enters too low (e.g., at the level of the teeth), the anesthetic will be deposited over the bony canal and prominence (lingula) that house the mandibular nerve, and not over the nerve itself.

The needle should be felt to pass through the ligaments and the muscles covering the internal surface of the mandible. One should stop when the needle has reached bone, which signifies contact with the posterior wall of the mandibular sulcus; bone *must* be felt with the needle. Failure to do so generally results from directing the needle toward the parotid gland (too far posteriorly) rather than toward the inner aspect of the mandible. The needle should then be withdrawn slightly and aspirated, and approximately 1 to 2 mL of solution should be deposited. Three to 4 mL may be required if needle positioning is suboptimal.

In children, the angulation is not parallel to the occlusal surfaces of the teeth; the barrel of the syringe must be held slightly higher, because the mandibular foramen is lower. One may anesthetize the lingual nerve by placing several drops of anesthetic solution while withdrawing the syringe. The anterior two thirds of the tongue can thus be anesthetized. In actual practice, the lingual nerve is consistently blocked with this procedure owing to the close proximity of both nerves. Following injection, it usually requires 3 to 5 minutes to achieve anesthesia.

**Complications**

Complications include inadvertent administration of anesthetic posteriorly in the region of the parotid gland, which will anesthetize the facial nerves (Fig. 33-10 *I*). This is an

annoying but relatively benign complication that will cause temporary facial paralysis (similar to a Bell's palsy) that affects the orbicularis oculi muscle and results in inability to close the eyelid. Should this occur, the eye must be protected until the local

anesthetic has worn off (approximately 2 to 3 hours), and the patient must be reassured. Anesthesia with bupivacaine (Marcaine) presents a more significant problem if this complication occurs, because bupivacaine anesthesia lasts from 10 to 18 hours in some patients.

## Mental Nerve Block

The mental nerve is blocked by the infiltration of local anesthetic about the nerve as it exits its bony foramen (Fig. 33-11) . Introduction of the needle in the mental nerve foramen is to be avoided, as the needle or injection of liquid into the foramen can produce neurovascular damage. Infiltration about the foramen will provide for anesthesia of the lower lip. Lacerations of the midline of the lips require administration of anesthetic about the mental nerve on each side of the face; this practice anesthetizes crossover fibers. Generally a 1.3-cm (0.5-in.), 25- or 27-ga needle on a 3-mL syringe is used.

### Anatomy

The mental nerve is a continuation of the inferior alveolar nerve, which innervates the mucosa and the skin of the lower lip of the ipsilateral side of the mandible, with limited crossover of midline fibers. The nerve emerges from the mental foramen below the second premolar.

### Approaches

Like the infraorbital nerve, the mental nerve may be blocked using an intraoral or an extraoral approach. Syverud and colleagues found that volunteers who received intraoral topical anesthetic followed by an intraoral injection considered the technique to be less painful than the extraoral approach. [5] Before using either approach, the mental foramen should be identified by palpation about 1 cm inferior and anterior to the second premolar. It is generally best to locate the foramen using a gloved finger placed into the labial area over the mandible. Generally the foramen will be just medial to the pupil along a sagittal plane.

When using the *extraoral* approach, the overlying skin is prepped and anesthetized with an anesthetic skin wheal. The mental nerve is approached through the overlying skin using a needle trajectory perpendicular or at a 45° angle to the foramen. As noted above, the local anesthetic is infiltrated about the nerve about 3 to 5 mm *outside* of the foramen. After placement of 1 to 2 mL of local anesthetic about the nerve, the needle is withdrawn and the area over the foramen massaged for 10 to 15 seconds to hasten anesthetic onset.

When using the *intraoral* approach, it is best to use topical anesthesia prior to infiltration. The lower labial fold adjacent to the first or second premolar is topically anesthetized. The mental foramen is again approached at about a 45° angle, and the area adjacent to the foramen is infiltrated with 1 to 2 mL of local anesthetic and the area massaged as

above.

## Scalp Block

Scalp blocks provide surgical anesthesia for the repair of scalp lacerations, drainage of superficial scalp abscesses, and exploration of scalp wounds.

### Anatomy

As shown in Figures 39-12 (Figure Not Available) and 39-13 (Figure Not Available) , the scalp receives its nerve supply from branches of the trigeminal nerve (fifth cranial nerve) and the cervical plexus. The forehead is supplied by the supraorbital and supratrochlear nerves. Both nerves are branches of the ophthalmic division of the trigeminal nerve. The temporal region receives its nerve supply from the zygomaticotemporal (a $V_2$ branch nerve), temporomandibular, and auriculotemporal nerves ($V_3$ branch nerves).

The posterior aspect of the scalp is innervated by the greater auricular and the greater, lesser, and least occipital nerves. The nerves that supply the posterior aspect of the scalp originate from the cervical plexus. All the nerves become superficial above a line drawn from the upper border of the external ear to the occiput and the eyebrows and converge toward the vertex of the scalp (see Fig. 33-12) .

Topographically, the nerves and vessels of the scalp are located in the SQ tissue above the epicranial aponeurosis. From this level they divide into small branches that extend to the deeper layers (epicranium and periosteum) (see Fig. 33-13) . [6]

### Approaches

A scalp block can be accomplished by individually blocking each nerve that supplies the scalp, but this approach is time consuming, difficult, and cumbersome. Because the nerves on the scalp are superficially located, the scalp block can easily be performed by injecting local anesthetic agents into the SQ tissue circumferentially around the area to be blocked. Injection of local anesthetic to the deeper levels is necessary only if bone is to be removed. Note that injection of local anesthetic agents only in the deeper layers without SQ infiltration results in an unsuccessful block and a greater amount of bleeding during surgical intervention. [7]

In preparation for the block, a band of hair may be clipped. (Some physicians prefer to shave the head, but this procedure is of unproven benefit.) A band 1 cm wide and 3 cm away from the wound can be circumferentially clipped. Local anesthetics are injected in the clipped area.

The skin is prepared using an antiseptic solution, and a skin wheal is raised at any point of the shaved skin using a 1.3-cm (½-in.), 25-ga needle. A 7.6-cm (3-in.), 22-ga needle is inserted through the skin wheal into the SQ tissue and advanced along the scalp circumferentially following the previously clipped area. An injection of 0.5% to 1% lidocaine or 0.125% to 0.25% bupivacaine with epinephrine (1:200,000) is used.

Epinephrine should be added to the local anesthetic agent to provide vasoconstriction and to prevent excessive blood loss and local anesthetic absorption. The total dose of the local anesthetic agents should not exceed the recommended dose for the particular agent. It may be useful to inject some local anesthetic solution into the temporalis muscle to prevent contraction of the muscle during the primary procedure.

Colley and Heavner demonstrated that when bupivacaine is used, the peak plasma local anesthetic concentrations occur within 10 to 15 minutes after injection. [8] Thus, the first 10- to 15-minute period after the injection is the most critical period for the occurrence of local anesthetic toxicity. Colley and Heavner also found that despite the scalp's high vascularity, the absorption of local anesthetics from the scalp is not excessive. Using the upper limit of the recommended

dose of bupivacaine without epinephrine (175 mg), they found that peak plasma bupivacaine concentrations were 0.8 mug/mL with a 0.125% solution and 1.2 mug/mL with a 0.25% solution. Considering that the toxic plasma threshold for bupivacaine is 4 mug/mL, these concentrations suggest that a scalp block using bupivacaine has a wide margin of safety, even without the use of epinephrine. When epinephrine is used with bupivacaine, its effect on absorption becomes more pronounced with concentrations of 0.125% than with those of 0.25%. This is probably because at low concentrations (0.125%), bupivacaine has a vasoconstrictor property. [9]

## Greater and Lesser Occipital Nerve Block

This relatively simple block may be useful in the emergency department for treating occipital neuralgia and tension headaches. For occipital neuritis, a long-acting corticosteroid, such as methylprednisolone (20 to 40 mg) may be combined with the local anesthetic.

### Anatomy

The posterior aspect of the head is supplied by the posterior rami of the cervical nerves. Two important branches of these nerves are the greater and lesser occipital nerves. The greater occipital nerve becomes superficial on each side at the inferior border of the obliquus capitis inferior muscle and runs superiorly toward the vertex over this muscle. The nerve is located medial to the occipital artery. The lesser occipital nerve is located approximately 2.5 to 3.5 cm lateral and 1 to 2 cm caudal to the greater occipital nerve (Fig. 33-14) . [10]

### Approach

It is not usually necessary to shave or clip the scalp prior to performing greater and lesser occipital nerve blocks. The greater occipital nerve can best be blocked at the nuchal line, which is in the middle of the external occipital protuberance and the mastoid process. The nuchal line is located between the insertion sites of the trapezius muscle and the semispinalis muscles. At this site, the greater occipital nerve is just medial to

the occipital artery.

The occipital artery is first palpated, and a 3.8-cm, 22-ga needle connected to a syringe that contains 5 mL of local anesthetic is inserted through the skin (see Fig. 33-14) . After obtaining paresthesia at the vertex, 5 mL of local anesthetic solution are injected. The lesser occipital nerve is blocked by a fan-like injection of a local anesthetic solution 2.5 to 3.5 cm lateral and 1 cm caudal to the point described for the greater occipital nerve. [10]

This procedure is not usually associated with any complications; however, intra-arterial injections should be avoided by careful aspiration.

## Ophthalmic (V$_1$ ) Nerve Block [3]

The lateral and medial branches of the supraorbital, supratrochlear, and infratrochlear nerves may be blocked by percutaneous local injection at the point where they emerge from the superior aspect of the orbit. Anesthesia of the

### Anatomy

The subtle supraorbital notch, which is in line with the pupil, may be palpated along the superior orbital rim. This landmark is the site of injection for blockage of the supraorbital nerves. The supratrochlear nerve is found 0.5 to 1.0 cm medial to the notch. The infratrochlear nerve is not usually blocked but is found in the most medial aspect of the superior orbital rim. If the anesthetic is placed on the forehead proper, this block may not produce complete anesthesia of the skin of the upper eyelid if the sensory branches to the eyelid are given off before the supraorbital nerve transverses the forehead.

### Approach

With the patient in the supine position, a skin wheal is raised. Paresthesias in the form of an electric shock sensation over the forehead are sought; these ensure a successful nerve block. One to 3 mL of anesthetic are placed in the area of the supraorbital notch. A finger or a roll of gauze should be held firmly under the orbital rim to avoid ballooning of anesthetic into the upper eyelid (Fig. 33-16) (Figure Not Available) .

If paresthesias cannot be elicited or if the nerve block is unsuccessful, a line of anesthetic solution placed along the orbital rim from the lateral to the medial aspect will ensure block of all of the branches of the ophthalmic nerve (Fig. 33-17) (Figure Not Available) .

Hematoma formation or swelling of the eyelid may occur but requires only local pressure. Occasionally, ecchymosis of the periorbital region will appear the next day, and the patient should be warned of this possibility.

Although this block is infrequently used, it is easily performed and is not associated with serious side effects. Its use should be considered when anesthesia of the forehead or

the anterior scalp is desired.

## CONCLUSIONS

Blocks about the head and neck are relatively painless when done carefully and slowly following topical mucosal anesthesia (for intraoral approaches) or local skin anesthesia (for extraoral blocks and approaches). Patients who appear anxious may benefit from sedation prior to attempting these blocks. These blocks should not be attempted in the uncooperative patient.

# Chapter 34 - Intravenous Regional Anesthesia

**James R. Roberts**

The clinical use of intravenous (IV) regional anesthesia has been well established  as a safe, quick, and effective alternative to general anesthesia in selected cases requiring surgical manipulation of the upper and lower extremities. Although often relegated to the operating room, the procedure is readily applicable to outpatient use. In the emergency department the technique provides quick and complete anesthesia, along with muscle relaxation and a bloodless operating field. The procedure is free from the troublesome side effects associated with other regional blocks, such as the axillary block. The procedure is easily mastered and has a very low failure rate, and consistently good results can be expected.

The first practical use of analgesia associated with IV injection of a local anesthetic agent was described by August Gustav Bier in 1908. [4] Colbern [5] has since proposed the eponym *Bier block*. Although the procedure has been in existence for many years, the need for special equipment and a safe anesthetic agent limited its use. However, the Bier block has now gained wide acceptance as a safe and effective procedure, and several papers extol its virtues. Although complications do exist, no reported fatalities directly attributable to the use of the Bier block have been reported. In this chapter, the techniques and complications are discussed according to their application in the emergency department.

## INDICATIONS AND CONTRAINDICATIONS

Indications for IV regional anesthesia include any procedure of the arm or leg that requires operating anesthesia, muscle relaxation, or a bloodless field. I have used the procedure for the reduction of fractures and dislocations, repair of major lacerations, removal of foreign bodies, debridement of burns, and drainage of infection. The procedure may be carried out on any patient of any age who is able to cooperate with the physician.

The only absolute contraindications are an allergy to the anesthetic agent and uncontrolled hypertension. Relative contraindications include an uncooperative patient and the recent ingestion of a large meal, both of which may only delay the procedure rather than contraindicate it.

## EQUIPMENT

The equipment required for IV regional anesthesia consists of the following:

> 1% lidocaine (Xylocaine), *without epinephrine,* to be diluted to a 0.5% solution ( *note:* 1 mL of 1% lidocaine equals 10 mg)

Sterile saline solution as a diluent

50-mL syringe/18-ga needle

Pneumatic tourniquet (single or double cuff) ( *note:* Do *not* use a standard blood pressure cuff)

IV catheters (18- or 20-ga) or a 21-ga butterfly needle

Elastic bandage/Webril padding

500 mL D5 W (5% dextrose in water) and IV extension tubing.

## PROCEDURE

The procedure should be explained in advance to the patient. If the patient is extremely apprehensive, premedication with midazolam (Versed), diazepam (Valium), or an opioid (e.g., morphine or fentanyl) may be helpful but need not be routinely used. The only painful portions of the procedure are the establishment of the infusion catheter and the exsanguination procedure. The procedure should not be done on patients who are intoxicated or obtunded or on those with a previous reaction to a local anesthetic.

The patient need not be free of oral intake (NPO) for a specific period of time before the procedure, but it is prudent to delay the procedure if the patient has just eaten a large meal. As a precaution, a large-bore catheter and IV line of D5 W are established in the unaffected extremity. Resuscitation equipment, including anticonvulsant drugs and oxygen, should be readily available.

While the patient is being prepared, the lidocaine solution is readied but withheld until the injured extremity is exsanguinated and the cuff is in place and reinflated as discussed below. The standard dose of lidocaine is 3 mg/kg injected as a 0.5% solution (1% lidocaine is mixed with equal parts of sterile saline in a 50-mL syringe). Farrell and associates have described a procedure termed the *minidose Bier block* using 1.5 mg/kg of lidocaine and reported a 95% success rate. [10] This lower dose may decrease the incidence of central nervous system side effects and is more desirable in the emergency department setting. (Additional lidocaine may be infused if the initial dose is inadequate.) Lidocaine with epinephrine should *not* be used. Plain lidocaine is also available as a 0.5% solution, and as such, it can be used directly to avoid the necessity of diluting the stronger solution.

A pneumatic tourniquet with cotton padding (to prevent ecchymosis) under the cuff is applied proximal to the pathology (Fig. 34-1) (Figure Not Available) . *It is strongly advised that one not use a regular blood pressure cuff*, because these often leak or rupture and are not designed to withstand high pressures for any length of time. A specially designed portable double-cuff pneumatic system, such as that marketed by OEC Zimmer Corporation, is ideal (Fig. 34-2) (Figure Not Available) .

The anesthetic is premixed in the syringe. The tourniquet is inflated, and a 20-ga plastic

catheter or a metal butterfly needle is placed in the superficial vein, as close to the pathologic site as possible, and is securely taped in place (Fig. 34-3) (Figure Not Available) . It is usually desirable to use a vein on the dorsum of the hand, but importantly the injection site should be at least 10 cm distal to the tourniquet to avoid injection of anesthetic proximal to the tourniquet. The hub remains on the catheter to avoid backbleeding or the syringe is attached to the butterfly tubing. This catheter will be the route of injection of the anesthetic agent.

The tourniquet is deflated, and the extremity is exsanguinated so that when the anesthetic agent is injected, it will fill the vascular system. Exsanguination may be accomplished by either of 2 methods. Simple elevation of the extremity for a few minutes may be adequate, but wrapping the extremity in a distal-to-proximal direction with an elastic bandage, being careful not to dislodge the infusion needle, enhances the exsanguination (Fig. 34-4) (Figure Not Available) . Wrapping may be painful; this step can be eliminated if it causes too much anxiety to the patient. If the wrapping procedure is not done, the extremity should be elevated for at least 3 minutes. During the wrapping procedure, care must be taken not to dislodge or infiltrate the infusion catheter.

With the extremity still elevated, the tourniquet is inflated to 250 mm Hg, the arm is then placed by the patient's side, and the elastic exsanguination bandage is removed. In a child, the tourniquet is inflated to 50 mm Hg above systolic pressure.

The 0.5% lidocaine solution is then slowly injected into the infusion catheter at the calculated dose. Note that the solution is placed in the arm in which circulation is blocked, *not* in the precautionary keep-open IV line on the unaffected side. At this point, blotchy areas of erythema may appear on the skin. This is not an adverse reaction to the anesthetic agent, but merely the result of residual blood being displaced from the vascular compartment, and it heralds success of the procedure.

In 3 to 5 minutes, the patient will experience paresthesia or warmth, beginning in the fingertips and traveling proximally, with final anesthesia occurring at the elbow. Complete anesthesia ensues in 10 to 20 minutes, followed by muscle relaxation. Note that adequate analgesia may exist even though the patient can still sense touch and position and has some motor function. If the "minidose" technique (1.5 mg/kg of lidocaine) does not provide adequate anesthesia, an additional 0.5 to 1 mg/kg may be infused at this time. Additional lidocaine was required in 7% of cases in one series using the minidose regimen. [10] The physician should be patient, however, and wait a full 15 minutes before infusing additional lidocaine. Alternatively, if analgesia is slow or inadequate an extra 10 to 20 mL of *saline* solution may be injected to supplement the total volume of solution to enhance the effect. *Do not exceed a 3 mg/kg total dose of lidocaine*. The infusing needle is then withdrawn, and the puncture site is tightly taped to prevent extravasation of the anesthetic agent. The surgical procedure or manipulation is performed, including postreduction x-ray films and casting or bandaging (Fig. 34-5) (Figure Not Available) .

Anesthesia from a fingertip-to-elbow direction seems to occur irrespective of the site of anesthetic infusion, but selecting an injection site near the site of pathology will provide more rapid anesthesia at a lower dosage. On completion of the procedure, deflation of

the tourniquet is *cycled* to prevent a bolus effect of any lidocaine that may remain in the intravascular compartment. The cuff is deflated for 5 seconds and reinflated for 1 to 2 minutes. *This action is repeated 3 or 4 times.*

If the tourniquet has been in place for <30 minutes, it is dangerous to deflate it, because adequate tissue fixation of the lidocaine probably has not occurred. This may result in a higher peak plasma lidocaine level, with increased side effects. If the surgical procedure is completed rapidly and the 3-mg/kg limit of lidocaine has been infused, the tourniquet should remain inflated until a full 30 minutes has elapsed, and only then should it be deflated using the cycling technique. It is reasonable to use a 20-minute cutoff if the minidose technique is used, because this dose is equal to a commonly administered IV bolus.

Sensation returns quickly when the tourniquet is removed, and in 5 to 10 minutes, the extremity returns to its preanesthetic level of sensation and function. After 20 minutes of observation, the patient is released (Table 34-1) (Table Not Available) .

If the procedure takes longer than 20 or 30 minutes, many patients complain of pain from the tourniquet, because the tourniquet is not inflated over an anesthetized area. The use of a double-cuff tourniquet alleviates the problem of pain under the cuff.

In the double-cuff system, there are 2 separate tourniquets placed side by side on the extremity. One is termed the *proximal cuff*, and the other is called the *distal cuff*. The proximal cuff is inflated at the beginning of the procedure, and anesthesia is obtained under the deflated distal cuff. When the patient begins to feel pain under the proximal cuff, the distal cuff is first inflated over an already anesthetized area, and the pain-producing proximal cuff is then deflated. One must be certain to inflate the distal cuff before the proximal cuff is released; otherwise, the anesthetic will rapidly diffuse into the general circulation.

## MECHANISM OF ACTION

Some of the anesthesia is undoubtedly related to the ischemia produced by the tourniquet, but most of the anesthesia
is secondary to the anesthetic agent itself. Although the exact mechanism by which anesthesia is produced is unknown, the site of action of the anesthetic may be at sensory nerve endings, neuromuscular junctions, or major nerve trunks. [11] Contrast studies have demonstrated that the anesthetic agent does not diffuse throughout the entire arm, yet anesthesia of the entire limb is obtained. For example, when the anesthetic agent is injected into the elbow and kept in that region with both distal and proximal tourniquets, anesthesia of the entire arm develops. [12] Evidence indicates that the local anesthetic does not simply diffuse from the venous system into the tissue but travels via vascular channels directly inside the nerve. Regardless of where the anesthetic is infused, the fingertips are the first area to experience anesthesia, suggesting that the core of the nerve is in contact with the anesthetic agent initially. Following release of the tourniquet, a considerable amount of the drug still remains in the injected limb for at least 1 hour. [13] This would suggest that at least a portion of the

anesthetic leaves the vascular compartment and becomes tissue fixed.

# PROCEDURAL POINTS

### Anesthetic Agent

Using 0.5% plain lidocaine at a dose of 1.5 to 3 mg/kg is preferred. Other agents have been used without demonstrable advantage and are not recommended. [14] Bupivacaine (Marcaine, Sensorcaine) is *contraindicated* because of the potential for serious cardiovascular and neurologic complications.

Dunbar and Mazze [7] showed that patients with IV regional anesthesia actually had significantly lower plasma lidocaine concentrations than patients with axillary block or lumbar epidural anesthesia for similar procedures. Peak plasma concentrations are reached 2 to 3 minutes after deflation of the tourniquet, and side effects are minimal if the deflation is cycled following the surgical procedure. The plasma half-life of lidocaine is approximately 60 seconds (see the excellent detailed discussion of pharmacokinetics by Covino [17] ), but the drug demonstrates a theoretical 3-compartment model similar to a direct IV infusion once the tourniquet is released. [18] Peak blood levels are related to the duration of vascular occlusion and to the concentration of the anesthetic. [17] [18]

Post-release peak plasma lidocaine levels decrease as the time of vascular occlusion (tourniquet time) increases. If the tourniquet is inflated for at least 30 minutes and the deflation-reinflation technique is used when the procedure is finished, plasma concentration of lidocaine should be approximately 2 to 4 mug/mL, below the 5 to 10 mug/mL level at which serious reactions occur. [7] Tucker and Boas demonstrated a peak plasma lidocaine level of 10.3 mug/mL following a 10-minute period of vascular occlusion, compared with 2.3 mug/mL if the tourniquet was inflated for 45 minutes. [18]

More dilute solutions of lidocaine are associated with lower peak lidocaine levels. When equal doses of lidocaine are used, the peak arterial plasma levels are 40% lower when the 0.5% solution is used than when the 1% solution is used. [18] For example, following 10 minutes of vascular occlusion, the peak plasma concentration of lidocaine has been demonstrated to reach 10.3 mug/mL with the 1% solution, compared with only 5.6 mug/mL when the drug was given under similar circumstances as a 0.5% concentration. [18]

### Exsanguination

Exsanguination of the extremity before injection of the anesthetic agent is considered essential for success by many physicians. Others do not believe that it is a critical factor. Exsanguination by simple elevation of the extremity should be done in all cases, but in certain cases one should consider avoiding the painful wrapping of the extremity with an elastic or Esmarch bandage. (Note that applying an Esmarch wrap over a fracture site is usually quite painful.) A pneumatic splint, such as the type used for out-of-hospital immobilization, is also a reasonable alternative to painful wrapping. The process of exsanguination is believed to allow for better vascular diffusion of the

anesthetic.

**Site of Injection**

Anesthesia is usually obtained no matter where the local anesthetic is injected, but some evidence indicates that the procedure is more successful when the anesthetic is injected distally. Sorbie and Chacho note the following failure rate associated with specific sites of anesthetic injection: antecubital fossa, 23%; middle of forearm or leg, 18%; and hand, wrist, or foot, 4%. [3] For most cases, a vein in the dorsum of the hand or foot is most often used. If local pathology precludes the use of the hand, the midforearm or antecubital fossa of the elbow are acceptable, albeit less desirable, alternatives as long as the infusion catheter is well below the tourniquet to avoid systemic injection.

Although most of the literature stresses the use of this technique on the upper extremity, it may also be used successfully in the leg. It cannot, however, be used for procedures above the knee. Tourniquet pain appears to be a limiting factor when the procedure is used on the leg. One must be certain to avoid damage to the peroneal nerve by using the tourniquet in the midcalf area only.

# COMPLICATIONS

Although IV regional anesthesia is both safe and simple, one should not be lulled into complacency, because complications do occur and are usually related to equipment failure or mistakes in the technique.

**Anesthetic Agent**

Serious complications seldom occur if proper attention is paid to technique. Reactions to lidocaine are rare and are usually systemic reactions from high blood levels. High levels may result from miscalculation of dosages, from too rapid release of the tourniquet before the anesthetic has become tissue fixed ("bolus effect") or, rarely, from advancement of the infusion catheter proximal to the tourniquet, resulting in direct IV infusion. [20] To emphasize the safety of this procedure it should be noted that the dose of lidocaine used in the "minidose" technique is similar to an IV bolus routinely given to patients with significant cardiovascular disease, in the presence of ventricular dysrhythmias.

Generally, the central nervous system effects of lidocaine are minor, resulting in mild reactions such as dizziness, lethargy, headache, or blurred vision. This should not occur in more than 2% to 3% of patients and requires no treatment. [7] Convulsions may occur but are extremely rare.

The most common complication relating to the anesthetic agent is rapid systemic vascular infusion, which occurs when a blood pressure cuff explodes or slowly leaks, resulting in both loss of anesthesia and high blood levels. [21] Similar complications may occur if the cuff is deflated before 30 minutes following the induction of anesthesia. Both

complications are the result of a bolus effect of the anesthetic, resembling an IV injection.

Van Neikerk and Tonkin reported 3 seizures in a series of 1400 patients. [19] Seizures are generally not recurrent and are treated with O2 and anticonvulsant drugs. Transient cardiovascular reactions, such as bradycardia and hypotension, are possible with large doses of lidocaine. Vasovagal reactions do occur. If resuscitation equipment is available and a precautionary IV line is started in the opposite arm, there should not be any serious sequelae.

One case of cardiac arrest for 15 seconds following the use of 200 mg of lidocaine has been reported, but the actual clinical scenario may have been a vasovagal reaction rather than a true cardiac arrest. [22]

**Additional Complications**

Thrombophlebitis can occur following IV administration of anesthetics, and the formation of insignificant amounts of methemoglobin with the use of prilocaine hydrochloride (Citanest) has been reported. [23] Methemoglobinemia also can theoretically occur with lidocaine but has not been reported.

A particularly bothersome problem has been the infiltration of the infusion catheter during exsanguination, resulting in tissue extravasation of the anesthetic agent. Also, there has been some leakage of anesthetic after the infusion needle has been removed. Both problems may result in poor anesthesia but may be minimized if a small, well-secured plastic infusion needle is used instead of a metal scalp vein ("butterfly") needle and if the puncture site is tightly taped following withdrawal of the catheter.

This procedure cannot be used in manipulations or operations in which the pulse must be monitored as a guide to reduction (e.g., supracondylar fractures of the humerus), because the tourniquet occludes arterial flow. The use of the Bier block in patients with sickle cell disease is not well documented. It should be used with caution until the ischemic effect of the tourniquet on the red blood cells of such patients has been clarified. In all patients the tourniquet time should not exceed 90 minutes. Ischemia for less than that amount of time is not associated with serious sequelae.

# Chapter 35 - Systemic Analgesia and Sedation for Procedures

**Kevin R. Ward, Donald M. Yealy**

Pain is a common complaint of patients who present to the emergency department (ED). In addition, many of the diagnostic or therapeutic procedures that are carried out in the emergency setting produce the full spectrum of pain and anxiety. The emergency physician has the task of providing acute pain relief and sedation to all age groups under all circumstances, unannounced. Unfortunately, pain in the emergent setting, especially during procedures, is often underrecognized, underprepared for, and undertreated. Fear of adverse effects, especially oversedation, has led many clinicians to underuse and sometimes withhold systemic analgesia and sedation. [4] Even patient ethnicity has been shown to influence analgesic regimens in the ED in some types of injuries. [5] Emergency physicians should be expert in providing sedation and/or analgesia and should make it a high priority that all patients receive pain relief and sedation commensurate with their individual needs during any procedure.

## BACKGROUND

Depending on the circumstance, the emergency physician may be required to produce analgesia, sedation, or a combination of both. The related terms used in this chapter are defined below :

1. Analgesia: Relief of pain without intentional production of an altered mental state such as sedation. An altered mental state may be a secondary effect of medications administered for this purpose.
2. Anxiolysis: A state of decreased apprehension concerning a particular situation in which there is no change in a patient's level of awareness.
3. Conscious Sedation: A medically controlled state of depressed consciousness that:
   a. Allows protective reflexes to be maintained.
   b. Retains the patient's ability to maintain a patent airway and adequate oxygenation and ventilation independently and continuously; and
   c. Permits arousal of the patient to physical or verbal stimulation
4. (*Note*: It is possible to vary the degree of conscious sedation in a dose-dependent fashion.)
5. Deep or Unconscious Sedation: A state in which sedation is profound enough to result in the loss of 1 or more of the 3 components of conscious sedation.
6. General Anesthesia: A state that produces the following 5 conditions: sensory, mental, reflex, and motor blockade and concurrent loss of all protective reflexes.

The major differentiation among the states of conscious sedation, deep sedation, and general anesthesia is the degree of intact protective reflexes. Short of maintaining the ability to sit up and cough, the value of various degrees of "protective" reflexes is unclear. In reality, the personnel providing sedation and analgesia are the patient's best

protective reflexes.

The progression from light analgesia and sedation to conscious and deep sedation to general anesthesia should be viewed as a continuum. The need to produce a state of general anesthesia outside the operating room is infrequent outside of brief inductions associated with rapid-sequence intubation (see Chapters 2 and 3) . However, creation of a state of conscious sedation is frequently required to permit the humane performance of procedures, with a state of deep sedation being less frequently required. Although this has not been studied, it is likely that some patients cross over between states without the provider's knowledge. This is especially likely in states of conscious and deep sedation. Therefore, careful monitoring must be instituted prior to the sedation process.

## PRINCIPLES OF SYSTEMIC ANALGESIA AND SEDATION

Common therapeutic mistakes resulting in inadequate analgesia and sedation include using the wrong agent, using the wrong dose, using the wrong route and frequency of administration, and poor use of adjunctive agents. With proper training and technique, adequate analgesia and sedation can be provided under almost any circumstance.

The correct agent (or combination of agents) and the route and timing of administration depend on the following factors: How long will the procedure last? Will it be seconds (e.g., simple relocation of a dislocated joint, incision and drainage of a small abscess, cardioversion) or minutes (e.g., complex fracture manipulation for reduction, breaking up loculations in a large abscess and then packing it)? Can local anesthesia be used as an adjunct? Does the patient only require sedation for a diagnostic study? How likely is it that the procedure will need to be repeated? The latter is mainly of import in acute fracture care, where repeat manipulation and reduction may take place.

No agents exist that are totally void of adverse effects. For the most part, agents chosen for systemic analgesia and sedation in the ED should act rapidly on the central nervous system (CNS), produce their effect reliably, last throughout the procedure, and allow for quick recovery and timely disposition of the patient.

Table 35-1 provides a list of agents and their properties used to provide analgesia and sedation for painful and diagnostic procedures. A dose-dependent continuum exists for all centrally acting agents. This continuum spans a range from simple relaxation and decreased anxiety to unconsciousness. Increasing doses of analgesics or sedatives are associated with a nonspecific sedative effect and less of the drug's specific effect.

*IV titration of agent(s) to patient response is the best method of obtaining rapid and safe analgesia and/or sedation.* For example, when using opioids, doses administered in 2- to 5-minute increments, observing for side effects such as miosis, somnolence, decreased responsiveness to verbal stimuli, minimally impaired speech, and diminished pain on questioning,

**TABLE 35-1** -- Agents for Analgesia and Sedation

| Drug | Class | Actions | Adult Dosing [*] | Pediatric Dosing [*] | Contraindications (Other Than Hypersensitivity) |
|---|---|---|---|---|---|
| Morphine | Opioid | Analgesia Sedation | 0.08-0.15 mg/kg IV | 0.1 mg/kg IV | Hemodynamic instability Active bronchospasm |
| Meperidine (Demerol) | Opioid | Analgesia Sedation | 1.0-3.0 mg/kg IV in divided doses | Usually administered IM as 2-4 mg/kg with 1 mg/kg of promethazine and chlorpromazine (DTP) | As plain meperidine: Hemodynamic instability Active bronchospasm *When used as DPT:* Age <6 mo Neurologic illness History of dystonic reaction |
| Fentanyl (Sublimaze) | Opioid | Analgesia Sedation | 1-5 mug/kg IV | 1-5 mug/kg IV 5-20 mug/kg transmucosally by oral route | Use with caution when age <6 mo Use with caution with nitrous oxide |
| Sufentanil (Sufenta) | Opioid | Analgesia Sedation | | 0.7-1.0 mug/kg nasally | |

| | | | | | |
|---|---|---|---|---|---|
| Ketamine (Ketalar) | Phencyclidine derivative | Dissociative anesthesia with: Analgesia Sedation Amnesia | 0.5-1.0 mg/kg IV 2-4 mg/kg IM | 1.0-1.5 mg/kg IV 2-5 mg/kg IM 50 mg/kg rectally 5-6 mg/kg orally Often used with atropine | Head injury Hypertension/CAD States of sympathetic depletion Active URI History psychosis Age <3 mo |
| Nitrous oxide ($N_2 O$) | Anesthetic gas | Analgesia Anxiolysis Sedation Some amnesia | 50:50 mix of $N_2 O$ and oxygen | 50:50 mix of $N_2 O$ and oxygen | Pregnancy (chronic exposure) Impaired mental status Uncooperative |
| Midazolam (Versed) | Benzodiazepine | Anxiolysis Sedation Amnesia | 0.02-0.1 mg/kg IV in divided doses | 0.05-0.15 mg/kg IV 0.05-0.15 mg/kg IM 0.5-0.7 mg/kg PO 0.2-0.5 mg/kg nasally | Concurrent CNS depression |
| Thiopental (Pentothal) | Barbiturate | Sedation Amnesia | 1-3 mg/kg IV | 1-3 mg/kg IV 25 mg/kg rectally | Hemodynamic instability Bronchospasm |
| Methohexital (Brevital) | Barbiturate | Sedation Amnesia | 1-3 mg/kg IV | 1-3 mg/kg IV 25 mg/kg rectally | Hemodynamic instability Bronchospasm |

| Etomidate (Amidate) | Imidazole-containing compound | Sedation Amnesia | 0.1-0.3 mg/kg IV | 0.1-0.3 mg/kg IV Not fully approved for ages <12 | See text |
|---|---|---|---|---|---|
| Propofol (Diprivan) | Isopropylphenol compound | Sedation Amnesia | 50-70 mug/kg/min IV continuous infusion | 50-70 mug/kg/min IV continuous infusion | Hemodynamic instability |
| Chloral hydrate | Sedative | Sedation | | 50-75 mg/kg PO or rectally | Renal or hepatic impairment |

CAD, Coronary artery disease; URI, upper respiratory infection

* For IV dosing, careful titration is recommended. Usual full therapeutic dose is shown.
Atropine dose 0.02 mg/kg IM.

are appropriate initial end points. If sedation is desired, similar end points such as ptosis (rather than miosis), somnolence, slurred speech, and gaze alteration should be sought. Repeated doses may be given in a titrated fashion based on the patient's response during the procedure. Careful titration of agent(s) and experience will help reduce the number of patients who complain of pain at the height of the procedure.

Oral, transmucosal, and intramuscular (IM) routes are more convenient means of administration but for the most part are much less reliable for timely dose titration to a desired response. The main advantage of the other routes is use in individuals (e.g., children) in whom IV access may be problematic or for procedures that may require only sedation in conjunction with the use of local anesthetics. These routes also may be advantageous for simple sedation for diagnostic procedures.

The oral and transmucosal (intranasal and rectal) routes of analgesics and sedatives are becoming increasing popular, especially in children. These routes may be useful in given situations if their limitations are understood. The first-pass effect of hepatic metabolism may increase the time of onset and decrease the ability to titrate effects or agents administered by the oral and rectal route.

With the exception of ketamine, agents administered IM have erratic absorption and a variable onset of action. As such, prolonged preprocedural and postprocedural observation may be necessary. When required because of limitations in obtaining IV access, the IM route offers little advantage over oral or transmucosal administration.

Another alternative route of analgesic/sedative administration is inhalation. The only available inhaled analgesic/sedative for use in the ED is nitrous oxide. It offers the advantage of being self-administered and somewhat titratable.

Because individual needs may vary widely, application of arbitrary ceiling doses of analgesic and sedative regimens is unwarranted. In most situations, the true ceiling dose of an agent is that dose that provides adequate pain relief or sedation without major side effects such as excessive and prolonged sedation. Side effects such as nausea, severe pruritus (mild nasal itching is common), and hypotension also should halt administration of the offending drug. However, these side effects are not strictly dose dependent and may be seen even with small doses.

When an expected response is not achieved after the administration of an agent, either a higher-than-anticipated need exists or the agent was not successfully delivered. The former is corrected by increasing the titrated dose of the agent, whereas the latter is corrected by ensuring that the correct dose was given and that the route of delivery is intact.

There are only 2 absolute contraindications to providing systemic analgesia or sedation for a procedure: the presence of severe clinical instability requiring immediate attention, and refusal by a competent patient. Relative contraindications include hemodynamic or respiratory compromise, altered sensorium, or inability to monitor side effects. However, even in many of these circumstances, appropriate agents can be given to provide analgesia and sedation while minimizing the chances for further deterioration. Although patients at the extremes of age represent monitoring challenges and require careful titration to effect, age is not a contraindication to analgesia and/or sedation.

**Risk Reduction**

An important aspect of successful administration of analgesia and sedation in the ED is choosing the correct modality for the patient. A presedation evaluation should be performed to identify patients who are at risk for complications or for reactions to certain agents. Although most patients in the ED have recently eaten, this factor is not an absolute contraindication to sedation. Others at risk for aspiration include pregnant and obese patients. In the event that deep sedation is needed, additional precautions should be considered. This may include delaying the procedure, placing a nasogastric tube, or special positioning of the patient.

Other important factors to be taken into account are the presence of recent alcohol and illicit drug ingestion. In addition, prescribed medication recently ingested, administered, or not taken as scheduled should be considered. This might include analgesics given

prior to the procedure or cardiovascular medications not taken as scheduled by the patient. Other important aspects of evaluation may include assessment of the patient's upper airway, assessment of the patient's ability to move the neck (should subsequent intubation be required), notation of dental prostheses, past medical and mental history, current medications, and the availability of a responsible escort at disposition.

Although no formal ED evaluation guidelines exist, incorporation of the American Society of Anesthesia (ASA) physical status classification might prove helpful in determining the need for consultation with an anesthesiologist. Table 35-2 lists the ASA physical status classification. However, it should be understood that the use of carefully titrated doses of analgesics and sedatives will not increase the risk of adverse events. In fact, the reverse is true. Untreated pain or anxiety can increase morbidity and mortality. Myocardial ischemia, dysrhythmias, cerebral ischemia, impaired host defenses, and impaired $O_2$ utilization are but a few complications that may result from poorly treated acute pain. [8] Some institutions require written informed consent for all patients undergoing elective conscious sedation in the ED.

## Pediatric Procedural Analgesia and Sedation

Sedation and analgesia of even infants will reduce the associated physiologic stress of clinical procedures. The principles of analgesia and sedation in children do not differ significantly from those outlined above. [7] [8] [9] [10] Variation in pediatric pharmacokinetics means that no one sedation or analgesic protocol will work for every child, or even for the same child in differing situations. As with the elderly, children may exhibit a paradoxical response to sedation, and the clinician should be prepared to deal with such events. Close attention must be paid to appropriate weight-based dosing. In addition, special consideration must be given to the route of administration, especially since more options exist and IV access may be more problematic. IM injections (with the exception of ketamine) should be avoided unless IV access is unobtainable and oral, nasal, and rectal routes are not practical.

Common pitfalls in pediatric analgesia and sedation include mixing 2 opioids or 2 sedatives, producing analgesia when only sedation is required, failure to choose agents that can be easily titrated, inexperience with pediatric airway management, and impatience. [9] Finally, because monitoring is more challenging and side effects are less predictable, additional care should be taken when administering analgesia and sedation to infants and neonates. The procedure should, of course, be thoroughly discussed with the caregiver prior to its being undertaken. Extreme care must be taken

| TABLE 35-2 -- American Society of Anesthesiologists Physical Status Classification | |
|---|---|
| Class I | A normal healthy patient |

| Class II | A patient with mild systemic disease (controlled chronic obstructive pulmonary disease [COPD], hypertension, diabetes mellitus, prior myocardialinfarction, etc.) |
|---|---|
| Class III | A patient with severe systemic disease that is not incapacitating (moderate to severe COPD, coronary artery disease with angina) |
| Class IV | A patient with severe systemic disease that is a constant threat to life (marked congestive heart failure, unstable angina) |
| Class V | A moribund patient not expected to survive without the operation. |

 in children with heart conditions (e.g., septal defects or other anomalies) or respiratory problems (e.g., severe asthma or cystic fibrosis).

# AGENTS

In the discussion below, elimination half-lives are given for the various agents. This should not be confused with the actual duration of clinical effect of the agent. Agents with a very high lipid solubility have long elimination half-lives but short clinical durations of action. This occurs because the highly lipid-soluble agents redistribute rapidly from their main site of action to other nonactive sites, making their clinical duration short but increasing the length of elimination from nonactive lipid stores, such as fat. Although redistribution from nonactive to active sites occurs, it usually does not do so in concentrations high enough to produce a secondary profound clinical response if used for short durations.

## Opioid Analgesics

### Morphine

#### Pharmacology.

The prototype opioid to which all other opioids are compared is morphine. It produces analgesia, euphoria, and sedation. Even low doses of morphine increase the pain threshold and can modify the perception of a noxious stimulus so that it is not experienced as pain. [11]

Because morphine penetrates the blood-brain barrier poorly, plasma concentrations following IV administration do not correspond well with its pharmacologic activity. Following IV administration, peak pain-relieving effects occur between 15 and 30 minutes, which correlate with cerebrospinal fluid (CSF) levels of morphine. It is estimated that <0.1% of morphine given IV enters the CNS at the time of peak plasma concentrations. Reasons for poor penetration include its low lipid solubility, protein binding, high degree of ionization at physiologic pH, and conjugation with glucuronic acid. Morphine is principally metabolized by conjugation with glucuronic acid in hepatic and extrahepatic sites (principally the kidney). It does not undergo significant first-pass uptake by the lung. Its elimination half-life is 114 minutes, with its "active" half-life much less. [11]

**Adult dosage and use.**

If the goal is production of long-lasting analgesia, morphine is an ideal and inexpensive agent. When it is given in 0.08- to 0.15-mg/kg (IV) increments every 5 to 10 minutes, most adults will experience good pain relief at a total dose of 10 to 20 mg, although higher doses may be needed. [11] Because of delays in achieving peak CSF levels, morphine is not a good choice for short procedures. Morphine should be administered cautiously in critically ill patients in whom volume status is unknown.

**Pediatric dosage and use.**

Morphine may be used in the same manner in infants and children as in adults. It is best used as a long-lasting agent for continuous pain in 0.1-mg/kg increments (IV). [6] [10] As in adults, it should be administered cautiously in children with a tenuous or unknown volume status.

**Adverse effects.**

Morphine can cause nausea and pruritus, especially around the nose. Although morphine is not a direct myocardial depressant, it may cause hypotension by a number of mechanisms, including reducing sympathetic nervous system tone to peripheral veins, resulting in venous pooling; drug-induced bradycardia via stimulation of the vagal nucleus in the medulla; and histamine release. [14] [15] Histamine release may also cause bronchospasm. These effects can be greatly reduced by optimizing intravascular volume prior to administration and giving the agent slowly.

**Meperidine**

**Pharmacology.**

Meperidine (Demerol) is a synthetic opioid agonist derived from phenylpiperidine and is one tenth as potent as morphine. It is the most commonly administered opioid agent for

acute pain in the United States. [11]

Meperidine is rapidly cleared, with about 90% metabolized in the liver. However, its active metabolite, normeperidine, has one half the analgesic effect but twice the seizure-producing properties, although seizures are rare in the acute setting. Unlike morphine, plasma levels of meperidine correlate reasonably well with levels of analgesia. [16] Peak effect begins 10 to 20 minutes after IV administration. Absorption of meperidine by the IM route is extremely erratic, making it a poor choice to be administered in this manner. [11] Despite a longer elimination half-life (180 to 264 minutes) than morphine, the duration of effect of meperidine is shorter.

**Adult dosage and use.**

Most adults undergoing a painful procedure will require a total IV meperidine dose of 1.0 to 3.0 mg/kg or more in divided doses, depending on the duration and intensity of the procedure. [17] The clinical duration of the analgesia and sedation produced by adequate doses may be longer than needed for short outpatient procedures.

**Pediatric dosage and use.**

Because of a pharmacodynamic profile similar to morphine, meperidine offers no advantage over morphine as an IV titratable agent for acute, long-lasting pain. Because it cannot be easily titrated, it is not an appropriate agent for titration during painful procedures.

The popular combination of meperidine (2 mg/kg), promethazine (Phenergan) (1 mg/kg), and chlorpromazine (Thorazine) (1 mg/kg), better known as DPT, has been a time-honored IM "package" of analgesia and sedation. In addition to its 2:1:1 mixture, it can also be used in a 4:1:1 ratio. [6] [10] Although it provides moderate sedation and analgesia, the onset and duration of the effect are delayed and unpredictable and cannot be titrated. When this was carefully studied, only 70% of patients were moderately to well sedated after receiving this regimen. In addition, the mean time to release home approached 5 hours, with return to normal behavior averaging up to 19 hours. [18] Because of these many factors, the Agency for Health Care Policy and Research (AHCPR) Task Force on Acute Pain Management has discouraged the use of DPT. [19]

**Adverse effects.**

In equipotent doses, meperidine will produce as much euphoria, sedation, nausea, and ventilatory depression as morphine. Meperidine possesses direct myocardial depressant effects and produces a histamine release similar to that produced by morphine, making it a poor choice in hemodynamically sensitive patients. [14] [20] Seizures may be seen in patients who develop high levels of its active metabolite, normeperidine.

**Fentanyl**

This agent is from a family of opioid agonists that also includes alfentanil and sufentanil; all are

related to meperidine. Fentanyl is 75 to 125 times more potent than morphine and is capable of causing profound sedation but has no intrinsic anxiolytic or amnestic properties. [11] A single dose given IV has an extremely rapid onset (<30 seconds), with a peak at 2 to 3 minutes and shorter clinical duration of action (20 to 40 minutes) than morphine or meperidine. This increase in potency and onset of action is in part related to its greater lipid solubility, which facilitates its passage across the blood-brain barrier. Most (75%) of a fentanyl dose undergoes first-pass pulmonary uptake. When large doses are used and lung sites are saturated, a prolonged effect may result. Fentanyl is rapidly metabolized into inactive forms and is excreted into bile and urine. Despite its shorter duration of action, the actual elimination half-life of fentanyl is 185 to 219 minutes, longer than that of morphine.

**Adult dosage and use.**

Because of its rapid onset and relatively short duration of action when administered IV, fentanyl is an ideal agent when analgesia is required for painful procedures, because it can be easily and rapidly titrated. [21] It has commonly been described as a "20-minute drug for a 20-minute procedure," highlighting this opioid's excellent utility for the ED. Doses between 1 and 5 mug/kg are usually sufficient, especially if a sedative is also used. However, *it is paramount that vigilant respiratory monitoring be performed when using this drug.* When fentanyl was given in a bolus dose of 2.0 mug/kg to healthy adult volunteers, half developed hypoxemia ($SaO_2$ <90%) and significant depression of ventilatory response to $CO_2$ , but not apnea. When 0.05 mg/kg of midazolam was given concomitantly, almost all patients developed hypoxemia, and half developed transient apnea. [22] When fentanyl was given in a titrated fashion during a painful procedure, the incidence was much less. [21]

In the absence of significant ethanol intoxication, hypovolemia, or concomitant drug ingestion, hypotension is rare, with even large doses of fentanyl. Because of its safe hemodynamic profile, fentanyl is an ideal analgesic agent for use in critically ill or injured patients. During acute painful procedures in which fentanyl is used, most patients will benefit from the addition of a pure sedative. The exception to this may be when it is used as an analgesic supplement to local anesthesia when application of the local anesthetic may be extremely painful, such as to the ear or genitalia (e.g., prior to drainage of a Bartholin's abscess). With fentanyl, nausea and vomiting are rare, in distinction to analgesia with morphine or meperidine.

**Pediatric dosage and use.**

The same IV dosing regimen may be used in pediatric patients. [6] [10] However, because fentanyl is capable of causing impressive sedation in children, many opt to use the agent alone, without a pure sedative, for painful procedures or as a supplement to local anesthesia. However, when used in this manner, amnesia to the procedure may not occur. In addition to the potential complication of chest wall rigidity, described below, infants and children are at greater risk for bradycardia when fentanyl is given rapidly at higher doses.

In addition to IV administration, fentanyl may be administered as a transmucosal preparation. Oral transmucosal fentanyl (OTF) in the form of a lollipop may decrease anxiety and pain in a child prior to a painful procedure, such as application of a local or regional anesthetic. [23] At doses of 10 to 20 mug/kg, adequate analgesia and sedation occur within 30 minutes in 85% of patients. The frequency of nausea and vomiting (20 to 47%) increases with the dose. The role of this agent in the ED is promising but awaits further study.

**Adverse effects.**

A commonly observed patient reaction to administration of fentanyl is pruritus, often of the face (patients invariably scratch their nose). Fentanyl causes little nausea, vomiting, hypotension or other cardiovascular changes, and virtually no histamine release, making it an ideal agent for the critically ill patient or patient with bronchospastic pulmonary disease. [15] However, in the neonate, fentanyl may produce bradycardia secondary to central vagal nucleus stimulation and a prolongation of the refractory period at the AV node. A rare side effect in rapidly administered doses (5 mug/kg) is chest wall rigidity, which may not be readily reversible with naloxone and may require assisted ventilation and, occasionally, pharmacologic paralysis. [6]

**Sufentanil and Alfentanil**

**Pharmacology.**

Sufentanil, an analogue of fentanyl, is 5 to 10 times more potent than fentanyl, which is reflected in its greater affinity for opiate receptors. [11] Despite its high lipid solubility, its volume of distribution is smaller because of protein binding. Sufentanil is metabolized rapidly, with an elimination half-life slightly less than that of fentanyl. Compared with similar doses of fentanyl, sufentanil produces a longer period of analgesia with less ventilatory depression, although once apnea occurs, there is no significant difference in time of return of spontaneous respiration.

Alfentanil is another fentanyl analogue but is one fifth to one tenth as potent as fentanyl and has just one third the duration. The onset of action is rapid, peaking at 1 to 2 minutes, because 90% of it exists in a nonionized form at physiologic pH. Its volume of distribution is 4 to 6 times smaller than fentanyl, because it is slightly less lipophilic and more protein bound. [26] The actual elimination half-life is 70 to 98 minutes.

Although there may be some pharmacodynamic advantages to the use of these drugs for painful outpatient procedures, a large experience with them has not been reported. They may not have a significant advantage over fentanyl in terms of clinical effectiveness and cost-effectiveness in the adult population.

**Pediatric dosage and use.**

Despite its limited application in adults, sufentanil has been used in children as a nasally administered agent. When used nasally at a dose of 0.7 to 1.0 mug/kg, its onset is 5 to 15 minutes, and its duration is 30 to 60 minutes. [27] It is effective when used for the same indications as fentanyl. Nasally administered midazolam (0.2 mg/kg) may be used concomitantly. When this is done, the dose of nasal sufentanil should be limited to 0.7 mug/kg. [10]

**Adverse effects.**

These agents have a complication profile similar to that of fentanyl.

## Nonopioid Analgesics

A large variety of nonopioid analgesic agents are available for use in the treatment of acute pain. These include nonsteroidal anti-inflammatory drugs (NSAIDs), the dissociative anesthetic ketamine, and the inhalational analgesic nitrous oxide. Although NSAIDs aid in decreasing the need and amount of other analgesics during and after many procedures, most are available only in oral or rectal form and require 1 hour or longer for peak effects. This limits their

utility for performing emergent procedures of short duration. Although ketorolac can be given parenterally, it offers little practical benefit and is relatively costly. In addition, it is not approved for use in children.

### Ketamine

**Pharmacology.**

Ketamine may be the most common anesthetic agent used throughout the world. Its safety profile has permitted its use in rather primitive surgical settings. The drug is a phencyclidine derivative and is unique among sedative, hypnotic, and analgesic agents because of its ability to produce all three states. Its actions are thought to be caused from a dissociation between the cortical and limbic systems. The result is a seemingly awake patient (catatonic) who is dissociated from the environment but in whom intense analgesia and sedation is present. [28] [29] Its analgesic and anesthetic effects are likely mediated by different mechanisms and probably involve interaction with opioid receptors as well as antagonizing *N*-methyl-ᴅ-aspartate (an excitatory amino acid). Ketamine is a water-soluble molecule but is highly lipophilic. It is hepatically metabolized, with an

elimination half-life of 1 to 2 hours but a much shorter clinical half-life secondary to its rapid redistribution.

Ketamine can be given IV, IM, orally, transmucosally, and rectally. The pharmacodynamics depend on the route of administration. Clinical effects occur within 1 minute when given IV at a dose of 1 mg/kg with return of coherence in 15 to 20 minutes. When given IM, the same effect is achieved with 4 to 5 mg/kg but with an onset of 5 minutes and recovery in 20 to 30 minutes. [28] [29]

**Adult dosage and use.**

Ketamine is an effective agent for use in acute fracture or joint care, burn and wound debridement and dressing, large laceration repair, large abscess incision and drainage, and analgesia for other painful procedures during the resuscitation of acutely ill or injured patients who are hemodynamically unstable but not sympathetically depleted. An initial IV loading dose of 0.5 to 1.0 mg/kg should be given over 30 to 60 seconds while observing for signs of dissociation. Additional 5- to 10-mg doses may be given as needed to maintain adequate analgesia and sedation. The IM route (2 to 4 mg/kg) may be used for those in whom IV access is problematic.

For adults, concomitant benzodiazepine administration is recommended. Lorazepam at a dose of 1 to 4 mg IV just prior to use is reported to be very effective in preventing emergence phenomena. [30] [31] Our experience demonstrates that 0.01 to 0.05 mg/kg of IV midazolam given in divided doses during the procedure will also suffice. In nonintubated patients, the use of an anti-sialagogue is also recommended. Since atropine is a centrally acting agent and may potentially aggravate emergence delirium, glycopyrrolate should be used.

**Pediatric dose and usage.**

The safety and efficacy of ketamine has been documented in over 11,000 children. Indications for use in children are essentially the same as for adults. [32] [33] In addition, it may be used as a sedative agent for children undergoing diagnostic procedures. Ketamine is also valuable for use in repair of intraoral lacerations. Doses of 1.0 to 1.5 mg/kg IV or 2 to 5 mg/kg IM are effective. As with adults, the routine use of an anti-sialagogue is recommended. However, since emergence phenomena is rarer in children, especially those <10 years of age, atropine is most commonly used. The addition of a benzodiazepine such as midazolam (0.025 to 0.05 mg/kg) is recommended in children older than 10 years. The IM route of administration is popular in children, and if this is used, atropine and midazolam may be mixed with ketamine, and all 3 given as 1 injection.

Ketamine may also be administered to children by other routes, including nasal, oral, and rectal. [6] Effective anxiolysis and moderate sedation can be achieved with an oral dose of 5 to 6 mg/kg for outpatient procedures. Rectal ketamine in a 50-mg/kg dose is capable of producing a dissociative state in 4 minutes without cardiorespiratory

depression.

**Adverse effects.**

Compared to equipotent doses of other agents, ketamine has by far the best safety profile in terms of cardiorespiratory complications. Spontaneous respiration and upper airway tone are preserved, making intubation unnecessary, even in states of deep sedation. Protective airway reflexes such as swallowing and coughing are not depressed and may be slightly exaggerated. Ketamine may also cause hypersalivation and bronchorrhea. For these reasons, laryngospasm is occasionally produced with ketamine administration, especially in those with an active upper respiratory tract infection. The bronchorrhea and hypersalivary effects of ketamine can be reduced with prior administration of atropine (0.01 mg/kg to a maximum of 0.5 mg) or glycopyrrolate (0.005 mg/kg to a maximum of 0.25 mg).  Profound respiratory depression is rare, except after rapidly administered doses in children or in those with central nervous system injuries. Although vomiting is relatively common with ketamine, intact protective reflexes generally prohibit aspiration.

Although ketamine is a direct myocardial depressant, this is counteracted by its sympathomimetic actions, which are caused in part by its effect in inhibiting catecholamine uptake. This usually results in a net increase in heart rate and blood pressure after IV administration and is in part why it is valuable as an induction agent in hypovolemic patients. By the same token, because of this sympathomimetic effect, its use in those who may be catecholamine depleted or in those with severe coronary artery disease is cautioned.

Ketamine also has been inconsistently noted to increase intracranial pressure. The usual result, however, is a net increase in cerebral perfusion pressure because of increases in mean arterial pressure. Consideration of these factors should take place prior to its administration in those patients with potential head injury. Ketamine occasionally causes a transient erythematous papular rash involving the face and neck, but this is of no clinical significance.

The major concern with the use of ketamine has been its ability to cause hallucinations on emergence, even at subanesthetic doses. The true incidence of this occurrence is unknown, but maximum incidence has been estimated to be 50% in adults and 10% in children. Risk factors may include age >10 years, female gender, personality disorders, history of frequent dreaming, excessive stimulation during recovery, and rapid IV administration. The incidence of emergence phenomena can be reduced with IM or slow IV administration; a quiet environment for recovery; and, most importantly, concomitant administration of a benzodiazepine. Adjunctive benzodiazepine use can virtually eliminate emergence phenomena, although no exact dose has been studied in the outpatient setting. The precise mechanism for this

effect is not known but may be due in part to increased time of ketamine metabolism and prolonged recovery.

**Nitrous Oxide**

**Pharmacology.**

Nitrous oxide-$O_2$ mixtures provide anxiolysis and moderate analgesia when inhaled. [34] In U.S. EDs, it is used as a 50:50 mix at or near sea level or a maximum of 60:40 at higher elevations. In this concentration alone, general anesthesia cannot be provided. In its pure form, nitrous oxide is colorless, sweet smelling, heavier than air, and nonflammable. When mixed with $O_2$, however, it supports combustion. Nitrous oxide is highly soluble in plasma (30 times greater than nitrogen) and quickly diffuses across biologic membranes. This accounts for its rapid onset of action (30 to 60 seconds). Maximum effect occurs after about 5 minutes. Cortical function is depressed rapidly, and basically all sensations are affected. [34] These include decreases in taste, smell, hearing, and sensation to touch, pain, and temperature. The cardiovascular system, as well as the respiratory system and airway reflexes, are not significantly altered.

Inhalation of a 50:50 nitrous oxide-$O_2$ mixture has been reported to produce analgesia equivalent to 10 to 20 mg of morphine. However, patients experience a wide range of levels of relief. About 25% report marked pain relief, with 40 to 50% reporting mild to moderate relief, and the remaining 15 to 25% experiencing no relief. Because of this variable response, when extremely painful procedures are performed, carefully titrated doses of an opioid may be required to supplement analgesia. Conversely, nitrous oxide may be used as a means to reduce the amount of opioid analgesics required for procedures.

**Equipment.**

In the ED, nitrous oxide is self-administered via a hand-held mask or mouthpiece attached to a demand valve (Fig. 35-1) . A negative pressure of 3 to 5 cm $H_2O$ must be produced within the mask or mouthpiece to activate the flow of gas. This provides for patient-controlled delivery in cooperative adults or children above the age of 8 years and acts as a fail-safe control, because if the patient becomes too somnolent, delivery will not continue.

In the United States, a double-tank system is used to deliver the nitrous oxide and $O_2$ mixture. The system relies on a mixing valve preset to deliver a fixed 50:50 ratio and will only deliver gas when $O_2$ is flowing. The double-tank system contains a fail-safe device that automatically stops the flow of nitrous oxide when the $O_2$ supply is depleted. In addition, through special reference pressures, the unit provides automatic $O_2$ enrichment at very shallow breathing rates.

**Adult and pediatric use.**

The use of nitrous oxide is helpful as an adjunct during local or regional anesthesia administration prior to repair of a variety of soft tissue and orthopedic injuries. It may also be administered during difficult pelvic examinations, for cardiac- and

noncardiac-related chest pain, during attempts at difficult IV access, and as initial pain treatment of sickle cell pain crisis and renal colic. In general, nitrous oxide is ideal for procedures or conditions that are associated with a continuous baseline pain level, but it may afford insufficient analgesia when a particularly painful procedure (e.g., abscess drainage) is performed. As mentioned above, the addition of an opioid may be required for patients who do not receive moderate relief with nitrous oxide, or for more prolonged procedures. [9] Although the patient self-administers the gas, the clinician must be certain that the mask is actively held in place by the patient so that the mask will be released and gas flow stopped should the patient begin to lose consciousness.

**Adverse effects and precautions.**

A scavenging system must be in place to collect exhaled nitrous oxide when it is used. Scavenging devices can be placed in line with the delivery system to collect exhaled nitrous oxide, resulting in levels far below the 1200 ppm considered hazardous for use in the ED. A number of generally minor adverse effects may be seen even with therapeutic dosing (Table 35-3) . Because of concern about increased rates of spontaneous abortions, pregnant ED staff should avoid repeated exposures to the agent. However, its use in pregnant patients for short procedures is considered safe.

Because of its property of high diffusibility, nitrous oxide must be used with caution in the presence of closed-space disease such as bowel obstructions, pneumothorax, or pneumocephaly. The potential exists to greatly increase the size of the closed space, although when nitrous oxide is used in a 50:50 mixture for <30 minutes, increases are likely to be small. [41] Another concern with the use of nitrous oxide is the potential for *diffusion hypoxia*. When nitrous oxide is stopped, diffusion of nitrous oxide back from the blood into the alveoli may displace $O_2$ and produce a state of hypoxia. However, diffusion hypoxia does not occur with a self-administered 50:50 mixture.

Caution is advised in the use of nitrous oxide in the patient with chronic obstructive pulmonary disease (COPD), because the gas is mixed with 50% $O_2$ , which might cause depression of the hypoxic drive. However, 1 study found no increase in $Pa_{CO_2}$ in COPD patients given a 50:50 mixture. [42]

Although the potential for abuse by ED staff exists, such abuse should be rare if several simple steps are taken. As with other agents, a strict protocol of accountability should be in place. A simple locking device can be added to the cylinders of gas. In addition, the delivery valve or mouthpiece may be locked in the same place in the department as the opioids.

## Sedatives

### Benzodiazepines

**Pharmacology.**

Benzodiazepines are a group of highly lipophilic agents that possess anxiolytic,

amnestic, sedative, hypnotic, muscle relaxant, and anticonvulsant properties. [43] These agents lack any direct analgesic properties. Their use in conjunction with analgesics usually results in the need to administer less of the analgesic during the procedure. Diazepam and midazolam are the most common benzodiazepines

| TABLE 35-3 -- Side Effects of Nitrous Oxiide Analgesia | |
| --- | --- |
| Drowsiness | Dizziness or vertigo |
| Giddiness | Dysphoria or panic |
| Nausea | Voice change |
| Amnesia | Inappropriate laughing out loud |
| Paraesthesias | |

used as adjuncts with other agents, such as opioids, for painful procedures. Benzodiazepines in appropriate doses produce 2 helpful effects: anxiolysis and amnesia. Anxiolysis can be produced with relatively low doses. The amnesia produced by benzodiazepines is primarily anterograde in nature and usually dose dependent. Obviously, when used with appropriate doses of opioids, a smaller benzodiazepine dose will be needed to produce amnesia to any perceived pain. The anxiolytic and amnestic properties of benzodiazepines also help reduce the incidence and degree of emergence delirium associated with the use of ketamine.

An additional theoretical benefit for the use of benzodiazepines as an adjunct to painful procedures is their ability to produce muscle relaxation. Preventing the patient from consciously applying counterforces by providing proper sedation is likely the true benefit (e.g., during joint relocation).

Diazepam is insoluble in water and is dissolved in an organic solvent for administration. It is metabolized primarily by the liver and has a long elimination half-life (21 to 37 hours). In contrast, midazolam is water soluble, making IV administration less painful and absorption from the IM or mucosal route more rapid. It also undergoes primary

hepatic metabolism but has a greatly reduced elimination half-life of 1 to 4 hours. Midazolam is approximately 2 to 4 times more potent than diazepam, and its time to peak effect is 2 to 3 minutes (slightly faster than diazepam) when given IV. These differences primarily reflect midazolam's greater affinity for benzodiazepine receptors.

**Adult dosage and use.**

Diazepam offers no advantage over midazolam if a need exists to produce a titratable short state of anxiolysis or sedation. Midazolam given at a dose of 0.02 to 0.04 mg/kg (1 to 2 mg in most adults) every 3 to 5 minutes to doses up to 5 to 7 mg will produce adequate sedation in most patients, especially if opioid analgesics are also used. It may be used IM, but it is difficult to titrate in this manner. Midazolam is most commonly used for sedation for diagnostic procedures and as an anxiolytic and sedative adjunct to opioids during painful procedures. It also is used as an adjunct to ketamine anesthesia in adults and children over the age of 10 years in order to reduce the occurrence of emergence delirium.

Unlike barbiturates, etomidate, and propofol, it is difficult to use midazolam as a sole agent during rapid performance of painful procedures. This is secondary to its slower onset of action and prolonged duration. If midazolam used in doses equipotent (0.3 mg/kg) to those of barbiturates, ketamine, propofol, or etomidate, the onset of action is as rapid, but the duration of action is greatly prolonged. In addition, apnea is likely to occur.

**Pediatric dosage and use.**

Midazolam may be used for the same indications as in adults in an IV dose of 0.05 to 0.15 mg/kg. Some children require much larger doses, and rarely a child will exhibit a paradoxical response (i.e., hyperexcitability). Midazolam is used more frequently for sedation as an adjunct to local anesthesia for laceration repair and as a sedative for diagnostic procedures. Because of its pharmacology and the unique problems with medication administration in children, it has become popular to administer midazolam orally (0.5 to 0.7 mg/kg) and intranasally (0.2 to 0.5 mg/kg). When used intranasally, the technique of administration is as important as the dosing. Care should be taken to slowly apply the agent in a dripping fashion and to position the patient in such a way as to put

as much of the agent in contact with the mucosa as possible. If used in combination with intranasal sufentanil, the dose should be limited to 0.2 mg/kg. Note that midazolam's variable onset, when given orally or intranasally, may render this route inappropriate for many emergency procedures.

**Adverse effects.**

Because of its profile, midazolam has become the agent of choice in emergency medicine to supplement analgesics in the performance of painful procedures. It is associated with a dose-dependent decrease in ventilation that is greatly enhanced in the

presence of ethanol or other depressive drugs, especially opioids. These effects are exaggerated in the elderly. More than 80 deaths from respiratory arrest have been associated with the use of midazolam, and many of these occurred when an opioid was concomitantly used. [22] Careful attention to monitoring should take place when benzodiazepines and opioids are used together. When slowly administered and carefully titrated, these agents are usually void of any serious hemodynamic complications.

**Barbiturates**

**Pharmacology.**

Barbiturates are a class of highly alkaline and highly lipophilic drugs that work at the gamma-aminobutyric acid (GABA) complex and are capable of causing profound sedation, hypnosis, amnesia, and anticonvulsant activity in a dose-dependent fashion. [45] They possess no inherent analgesic properties and in low doses may produce a state of hyperesthesia. The major use of barbiturates has been as induction agents for the production of general anesthesia. In the ED, they have mainly been used as part of rapid-sequence endotracheal intubation protocols.

Thiopental and methohexital are the barbiturates most commonly used IV and are termed *ultrashort-acting*. Because of their lipid solubility, they are also effective when given rectally. When given IV, both produce effects within 1 minute. Clinical recovery in terms of protective reflexes and wakefulness is rapid and reflects the rapid redistribution of these agents from the central nervous system (CNS). The elimination half-lives of methohexital and thiopental are 3.9 and 11.6 hours, respectively. Their rapid redistribution half-lives, however, are similar at 5.6 and 8.5 minutes, respectively. [45] Both agents produce residual sedation.

**Adult dosage and use.**

Barbiturates such as thiopental or methohexital may be useful in such procedures as rapid relocation of a large joint; simple, rapid reduction of a fracture; or incision and drainage of an abscess. Because they provide no analgesia in the doses used, the procedure should be extremely short, so that the duration of acute pain from the procedure is minimized. The use of these agents for emergent cardioversion is not recommended because of the high probability of worsening an already compromised hemodynamic status. Incremental IV titrated doses of methohexital (1 to 3 mg/kg) or thiopental (1 to 3 mg/kg) usually produces sedation within 1 to 2 minutes that lasts for 5 to 10 minutes. Rarely is >2 to 3 mg/kg of either agent required.

**Pediatric dosage and use.**

Thiopental and methohexital may be used in the same manner as in adults and at the same dose. In addition to its IV use, excellent longer lasting sedation for pediatric diagnostic procedures occurs when either agent is given rectally at a dose of 25 mg/kg. Sedation is produced within 8 minutes, and apnea has not been reported to be a

problem. This route and dose also may be used when sedation is required for laceration repair in addition to local anesthetics.

**Adverse effects.**

Use of barbiturates can be associated with major adverse effects, especially if patients are not chosen correctly and dosing and monitoring are not carefully performed. These effects include hypotension in overtly or occultly hypovolemic patients, through a number of mechanisms. Even in normovolemic patients, transient 10- to 20-mm Hg drops in blood pressure and increases in heart rate may occur. [46] Because barbiturates also cause histamine release, airway tone can increase (including laryngospasm) during barbiturate administration. Barbiturates also cause a dose-dependent decrease in ventilatory drive and maintenance of protective reflexes. Unfortunately, the window between light sedation, deep sedation, and general anesthesia produced by these agents can be narrow.

**Etomidate**

**Pharmacology.**

Etomidate is an imidazole derivative that is able to produce sedation, anxiolysis, and amnesia equal to that of barbiturates, but with significantly fewer adverse hemodynamic effects. [28] It has most commonly been used to induce anesthesia in hemodynamically compromised patients, as well as to reduce intracranial pressure while avoiding decreases in mean arterial pressure. Its onset of action and recovery are similar to those of thiopental and methohexital.

**Adult dosage and use.**

The main indication of etomidate may be during the performance of short painful procedures, such as cardioversion, in those individuals who are hemodynamically compromised or have a history of severe coronary artery disease. Doses of 0.1 to 0.3 mg/kg slowly administered IV are usually adequate. As with barbiturates, some residual sedation exists.

**Pediatric dosage and use.**

Etomidate has not been approved for use in children under the age of 12 years. [6] Although experience with this agent as a sedative for painful pediatric procedures is limited, its use in patients with a tenuous hemodynamic status may still be warranted.

**Adverse effects.**

Etomidate is associated with nausea on recovery and benign myoclonic jerks. This latter effect may limit its usefulness as a sedative during diagnostic procedures requiring a motionless patient. Transient reduction in cortisol production has also been documented

after the use of etomidate, but the clinical significance of this is unclear.

**Propofol**

**Pharmacology.**

Propofol is a substituted isopropylphenol. It is highly lipophilic and capable of producing profound sedation, hypnosis, and amnesia, but it is without innate analgesic properties. [28] The characteristic that sets this drug apart from others is its rapid metabolic clearance and redistribution, which is approximately 10 times faster than that of thiopental. Although the elimination half-life is 0.5 to 1.5 hours, the clinical recovery is extremely rapid (within 4 to 8 minutes), even after prolonged administration, and with little residual sedation. Recovery is even faster when subinduction doses are used. Onset after IV administration is rapid, with a "one arm-to-brain" circulation time of 15 to 30 seconds. Propofol has the added advantage of having intrinsic antiemetic effects. [47]

**Adult and pediatric dosage and use.**

Propofol is indicated for use in the same conditions as barbiturates. Experience with propofol in the ED is limited. [48] With proper guidance and sufficient practice, propofol may become the

agent of choice. This may be particularly true in cases where procedures must be repeated within a short time frame (e.g., remanipulation of a fracture after initial reduction is shown on radiographs to be inadequate).

When propofol is used, a carefully titrated dose of 50 to 70 mug/kg/min as a continuous infusion is recommended. [49] [50] [51] [52] At this dose it produces a sleep-like state with minimal respiratory depression and easy arousability to verbal commands, with recovery occurring in 2 to 2.5 minutes after stopping the infusion. If short-acting opioids are needed as an analgesic adjunct, the dose of propofol should be reduced.

**Adverse effects.**

The major drawbacks to the use of propofol for painful procedures are its potent respiratory and cardiac depressant effects. As with barbiturates, patients must be chosen carefully to prevent sudden profound hypotension (i.e., it should not be used when cardiovascular compromise is suspected). A narrow window exists between conscious and deep sedation. When it is used as a constant infusion, patients are awake 2 to 2.5 minutes after stopping the infusion. [47] Recovery is likely to be just as rapid with cessation of a continuous infusion as with administration of flumazenil or naloxone to reverse the effects of benzodiazepines or opioids, respectively. Lower doses may suffice, depending on the procedure. Adjunctive use of short-acting opioids such as fentanyl may be required for supplemental analgesia, depending on the type of procedure and its duration.

**Chloral Hydrate**

Chloral hydrate is a pure sedative hypnotic agent without analgesic properties. Its major use is for sedation as an oral or rectal agent in children during diagnostic procedures or as an adjunct with local anesthetics for painful procedures. [6] It is generally administered in a dose of 50 to 75 mg/kg, given orally or rectally. The average time to peak sedation is 40 minutes, with a recovery time of an additional 40 to 60 minutes and with residual effects (mild sedation) lasting up to 24 hours. [53] Higher doses (e.g., up to 100 mg/kg) are more effective but begin to carry risk similar to that of more potent regimens, without adding any practical benefit. Chloral hydrate is best used in healthy children for scheduled outpatient studies [54] and is generally of little use in emergency patients.

## Adjunctive Agents

Clinicians have long sought agents that would safely potentiate the effects of opioids. Agents that have been used for this purpose include phenothiazines (promethazine, prochlorperazine, and chlorpromazine) and antihistamines (mainly hydroxyzine). However, the major theoretical benefit of concurrent phenothiazine use is for treatment of the nausea that occasionally accompanies the use of opioids. No analgesic benefit has been demonstrated from the combination of opioids and phenothiazines, although the level of sedation is increased. The sedation due to these agents cannot be reversed using opioid antagonists.

Hydroxyzine (Vistaril) is an antihistamine with minor sedative and antiemetic properties. Controversy exists as to its ability to augment the analgesic properties of opioids. One study found that 100 mg of hydroxyzine provided analgesia equal to 8 mg of morphine in addition to providing significant anxiolytic effects. The major disadvantage to this agent is that it is recommended only for IM use.

## Reversal Agents

Reversal agents are generally not required following the use of opioid or benzodiazepine agents for analgesia and sedation. However, patients with underlying pulmonary or cardiovascular disease may benefit from a more rapid reversal, as do those patients who demonstrate apnea not responding to stimulation. Specific agents exist for reversal of both opioids and benzodiazepines.

**Opioid Reversal**

**Pharmacology.**

Naloxone and nalmefene are opioid antagonists that competitively inhibit opioid agents at opiate receptors. They rapidly reverse mainly the respiratory depressant effects of opioids. Both agents may be administered IV, IM, or even sublingually if needed.

**Adult and pediatric use.**

Naloxone at an initial dose of 2.0 mg in adults and 0.1 mg/kg in children will generally reverse the effects of opioids. Doses may be repeated every 2 to 3 minutes to a total dose of 10 mg in both adults and children. Nalmefene at an initial dose of 0.1 mg/70 kg, up to a total dose of 1.5 mg/70 kg in adults, produces the same reversal as naloxone, with the potential added advantage that it has twice the opioid receptor blockade at 8 hours, making resedation with commonly used opioids less likely. Nalmefene's efficacy in children has not yet been demonstrated. For patients with expected continued pain or those with preexisting physiologic dependence, the reversal should be more controlled (see below).

**Adverse effects.**

The opioid reversal agents will *not* induce systemic opioid withdrawal symptoms in a patient without preexisting physiologic dependence. However, some patients will experience nausea with opioid reversal, and those patients with persistent pain following their procedure will be quite uncomfortable. Nonopioid analgesia may benefit these latter patients (i.e., nonsteroidal anti-inflammmatory drugs [NSAIDs]). Rapid reversal also may lead to return of anxiety and sympathetic stimulation. In these situations it is preferred to partially reverse opioid sedation by administering small aliquots of naloxone (e.g., 0.2 to 0.4 mg per dose in an adult; 0.01 mg in a child) every 1 to 2 minutes until the desired level of alertness or respiratory drive is achieved. Close monitoring of respiratory effort will be required as the naloxone wears off.

**Benzodiazepine Reversal**

**Pharmacology.**

Flumazenil is a benzodiazepine antagonist that competitively inhibits benzodiazepine agents at the GABA receptor. It is capable of prompt reversal of benzodiazepine-induced respiratory and CNS depression.

**Adult and pediatric use.**

Flumazenil is effective in adults and children when given in a dose of 0.02 mg/kg in children or 0.1 to 0.2 mg in adults at 60-second intervals up to a total dose of 1 mg.

**Adverse effects.**

Blockade of the GABA receptor may lead to repetitive seizures. Flumazenil should be used with extreme caution in individuals using benzodiazepines for chronic seizure control, in persons with a cyclic antidepressant overdose, and in those with an elevated intracranial pressure. Rapid reversal also may lead to return of anxiety

and sympathetic stimulation. Administering flumazenil in small aliquots (e.g., 0.1 mg/dose) over an extended period of time (i.e., 1 to 2 minutes per dose) will result in a more gradual reversal and will ameliorate some of the benzodiazepine reversal anxiety or hyperactivity seen with rapid bolus injections.

# SEDATIVE-ANALGESIC COMBINATIONS

Patients undergoing procedures primarily associated with anxiety, such as wound repair after local anesthetic infiltration or diagnostic imaging studies, are best managed using pure sedatives. However, most painful procedures that will last more than a few seconds may benefit from the use of both a sedative and an analgesic agent to maximize patient cooperation and comfort. The clinician must use judgment, incorporating the patient's emotional and physical state and the anticipated duration of the procedure and associated pain, to determine the degree of analgesia and sedation required. In all circumstances, careful monitoring and titration of drug doses to effect are required. For most agents, the upper limit of dosing is guided by patient response. Below are some examples of effective combinations for painful procedures.

### Fentanyl and Midazolam

#### Indications

This combination has become a commonly used regimen for painful procedures in both adults and children. The combination is close to ideal, because both agents have approximately the same time to peak action, as well as the same clinically effective duration. In addition, both agents can be reversed if required. Since the combination can easily produce apnea, careful monitoring is required. The combination is effective for fracture reduction, relocation of joints, abscess drainage and packing, chest tube placement, wound and burn debridement, and other painful procedures. In addition, when given in small and frequent aliquots, the combination provides effective analgesia and sedation in victims of multisystem trauma or other patients with critical illness who are intubated and require a prolonged diagnostic workup in the ED.

#### Adult Use

After obtaining written informed consent that is specific for elective conscious sedation (where mandated), and after initiation of proper monitoring (see Monitoring During Use of Analgesia and Sedation), sedation should first be initiated by administering 1 to 2 mg of midazolam over 30 to 60 seconds. Repeat doses should be administered every 3 minutes until mild sedation and anxiolysis are produced. This occurs in the vast majority of individuals with 3 to 5 mg. Fentanyl should then be administered in 100-mug (2-mL) increments every 3 to 5 minutes until adequate analgesia and sedation are obtained (as evidenced by slurred speech, ptosis, and drowsiness, but purposely responsive to painful and verbal stimuli). The best judge of adequate analgesia will be the patient's response in the initial stages of the procedure. In most cases a dose of 4 to 5 mug/kg is

sufficient.

Some clinicians prefer to reverse the above order, administering fentanyl first. With this alternate approach, an adequate analgesic dose is first estimated, and its effects will persist even if the operator chooses to reverse the midazolam portion of the sedation. This approach may be less satisfactory in those procedures where the intensity of pain increases significantly during the procedure. A detailed description of the approach to the combined midazolam/fentanyl sedation/analgesia appears in Table 35-4 .

**Pediatric Use**

The above regimen may be performed in children undergoing painful procedures. Midazolam is given in 0.03- to 0.05-mg/kg increments, and fentanyl is given in 1 mug/kg doses until the desired effect is achieved. As mentioned above, if IV access is problematic, a similar state can be produced with a combination of nasally administered sufentanil (0.7 mug/kg) and midazolam (0.2 mg/kg).

## Midazolam with Other Opioids

**Indications**

Midazolam can be used in combination with other opioids such as morphine and meperidine for analgesia and sedation during painful procedures. However, opioids other than those in the fentanyl family have peak actions in the 15- to 20-minute range, making their rapid titration with midazolam difficult and increasing the necessary period of recovery. If used, sedative agents should be given 10 to 20 minutes after opioid administration in order to take advantage of peak analgesic effects. The main advantage of this regimen may be cost.

**Adult Use**

After initiation of appropriate monitoring (see Monitoring During Use of Analgesia and Sedation), the opioid agent is titrated to light sedation. Generally, morphine sulfate is administered in 1- to 2-mg increments every 3 to 5 minutes until the patient is comfortable and eyelids are heavy or speech is slightly affected ("thick"). Another 15 to 20 minutes are allowed to pass for peak opioid effect to occur. Then, just prior to the procedure, midazolam in 1- to 2-mg increments is used to supplement the sedation until the patient is somnolent or speech is slurred. Asking the patient to count backward from 100 is a useful tool. Often the patient will begin to skip numbers or count repetitively when the appropriate degree of sedation is reached.

## Ketamine and Midazolam or Lorazepam

This combination is ideal in settings similar to those in which fentanyl and midazolam are used. Details of the dosing of this regimen, including use of anti-sialagogues, are

provided in the preceding section on ketamine administration.

## Nitrous Oxide and Fentanyl

### Indications

Nitrous oxide has both sedative and analgesic properties, the degree of which is somewhat unpredictable. However, because of the ease of administration and low incidence of respiratory depression, its use as an adjunct to short-acting

| **TABLE 35-4** -- Procedure for Safe and Successful Conscious Sedation with Midazolam/Fentanyl | |
|---|---|
| **Caveats** | |
| 1. | Don't consider this procedure if you do not have experience with the drugs or the time to perform it properly (15-20 min). Do not attempt the procedure if the pulse oximeter is not working, the IV is not secured, or the room is too small. |
| 2. | This is a 2-person procedure, 1 to monitor the patient and 1 to administer medication and perform the procedure. |
| 3. | Individual response to the drugs is variable and dependent upon the patient's underlying physiologic state and the presence of concomitant drugs/medication. |
| 4. | Maximum drug effect may take 2-3 min following administration. Proceed slowly and be patient, allowing the medication to take full effect before giving more. |
| 5. | Have naloxone and flumazenil at the bedside. Label all syringes with the full names of the drug (not with 1-letter abbreviations). |

| | |
|---|---|
| 6. | If the patient seems overly sedated, perform the procedure. The pain of the procedure often stimulates respiration and lessens sedation. |
| 7. | Give written instructions at the time of release, since even a seemingly awake patient who has received midazolam may not remember anything. |
| 8. | The order of the drugs is one of personal preference. Reversal of the drugs is not routine. If flumazenil alone is used for reversal, the patient awakens, and the analgesic effect of fentanyl remains. |
| 9. | Where appropriate, obtain written informed consent specific for conscious sedation. |

**Protocol**

| | |
|---|---|
| 1. | Establish an IV infusion of normal saline (18-ga catheter preferred in adults) in the supine patient with the bed rails in the up position. |
| 2. | Pulse, respiratory rate, blood pressure, and level of consciousness should be recorded intially, *and every 5 to 10 min throughout the procedure.* |
| 3. | Continuous monitoring of oxygen saturation with a pulse oximeter probe (to maintain at >95% or no less than 3% to 5% less than the initial value) must be performed. Supplemental oxygen via nasal prongs is given at a rate of 4 to 6 L/min. ECG monitoring is optional but suggested in the elderly or those with a cardiac history. |
| 4. | A resuscitation cart with a bag-valve-mask, oral and nasal airways, endotracheal tubes, and a functioning laryngoscope must be nearby. Suction equipment and naloxone and flumazenil should be at the bedside. |

| | |
|---|---|
| 5. | Administer 1 mg midazolam over 30 to 60 sec; if, after 3 to 5 min, there is no evidence of mild sedation (subjective relaxation by the patient with mild drowsiness and normal or minimally altered speech), additional 1-mg doses can be administered in a similar fashion, up to a maximum of 0.1 mg/kg. * The goal is *mild sedation and anxiolysis,* achievable in most patients with 2 to 4 mg of midazolam. |
| 6. | Reassess clinical status (see 2). |
| 7. | Administer fentanyl 100 mug (2 mL) over 60 sec; this may be repeated in 0.5- to 1.0-mug/kg (50- to 100-mug) increments every 3 to 5 min until adequate analgesia and sedation have been obtained (slurred speech, ptosis, drowsy but responsive to painful and verbal stimuli, and good analgesia with initial stages of procedure). The maximal total dose recommended is 5 to 6 mug/kg. * |
| 8. | Administer local anesthesia if indicated (this often serves to help gauge effectiveness or systemic analgesia). |
| 9. | Perform the procedure. Additional doses of fentanyl may be required based on the response and length of the procedure. |
| 10. | If hypoxemia, deep sedation, or slowed respirations unresponsive to external stimuli are seen during or after procedure, ventilation should be assisted with a bag-valve- mask and flumazenil and/or naloxone should be administered. Naloxone should not be given routinely at the termination of procedures, since it will abruptly reverse all analgesia. |
| 11. | Continue close observation until the patient is awake and alert, and release the patient with a friend or relative only after a minimum 1 hr of further observation. Instruct the patient not to drive or operate dangerous machinery for at least 24 hr. |

* For children, fentanyl alone is suggested in 0.5-mug/kg increments, up to a maximum total dose of 2 to 3 mug/kg.
Sublimaze 50 mug/mL.

analgesics such as fentanyl is desirable. Concurrent use of nitrous oxide reduces the

amount of IV analgesic needed.

**Adult and Pediatric Use**

After several minutes of breathing the 50:50 nitrous oxide-$O_2$ mixture, adults and children may receive IV administration of fentanyl in doses of 1 mug/kg given every 3 to 5 minutes until adequate analgesia is produced or the patient cannot coordinate the use of the mask and the demand valve. Rapid reversal takes place with administration of naloxone, removal of the mask, or both.

## Propofol and Fentanyl

**Indications**

Despite a sparsely published use in the emergency medicine literature, this combination may become common in adults and children with no underlying cardiorespiratory problems. The major advantage of this combination is when a painful procedure may have to be repeated, such as with complex fracture manipulation and reduction.

**Adult and Pediatric Use**

For adults and children, fentanyl can be given in a priming dose of 1 to 2 mug/kg over 1 minute. Propofol is then administered as a constant IV infusion at a dose between 50 and 70 mug/kg/min. When the patient is adequately oxygenated, respiratory depression, when recognized, can be reversed within 30 to 60 seconds by cessation of the propofol infusion, without the occurrence of hypoxemia.

# MONITORING DURING USE OF ANALGESIA AND SEDATION

## General Indications

Monitoring has two goals: ensuring adequate analgesia and sedation and detecting adverse effects. While adverse effects

will undoubtedly occur regardless of how much care is taken, most serious adverse effects can be averted when detected at the earliest possible time with well-chosen and applied monitoring. Clinical monitoring of vital signs is required whenever analgesic or sedative agents are administered. The use of adjunctive equipment should be guided by local hospital policy. Generally, $O_2$ saturation and rhythm monitoring are used when conscious sedation is the therapeutic goal. Adjuncts such as expired $CO_2$ monitoring and automatic blood pressure monitoring should be determined by patient status, planned analgesic and sedative agents, and anticipated duration and nature of the procedure.

## Interactive Monitoring

As important as the actual monitoring equipment are the personnel involved in the procedure and monitoring. Regardless of how much monitoring equipment is used, interaction between the clinician and the patient must take place. Monitoring techniques can therefore be divided into interactive and mechanical methods. Interactive monitoring involves contact between the person providing monitoring and the patient.

Assessment of the patient's baseline status is the first step of interactive monitoring. This includes measurement and recording of basic vital signs, such as blood pressure, respiratory rate, and heart rate. Baseline and interval assessment of the patient's level of consciousness should be performed and based on the stimulus required to evoke 1 of the following responses: awake and spontaneously conversant, somnolent but responsive to verbal or painful stimuli, or unresponsive to any stimuli.

Prior to administration of any agent to adults or children over the age of 8 years, some attempt should be made to assess pain. In clinical practice, the use of a simple numeric scale from 1 (barely perceptible pain) to 10 (worst pain imaginable) may work best. For research purposes, visual analog scales are optimal. When appropriate, the patient should be informed of what clinical actions will be taken for various subjective pain scores (a score >3 will be treated with additional analgesia). Ongoing interactive monitoring should take place in at least 5-minute intervals. In addition, certain side effects, such as dyspnea, chest pain, nausea, dizziness, and visual or auditory changes, should be regularly inquired about.

Patients should be carefully monitored until recovery is complete. Depending on the particular agent and dose used, interactive monitoring should take place at frequent intervals for at least 20 minutes or longer after the end of the procedure or the last dose of agent. During the recovery phase, repeat assessments at 5- to 10-minute intervals should continue until the patient is awake and conversant, with appropriate alarms set on mechanical monitoring devices. Reversal of analgesia and sedation for purposes other than hemodynamic or respiratory compromise is generally unnecessary and is discouraged. Rapid reversal may lead to return of pain, anxiety, and sympathetic stimulation.

**Monitoring Equipment**

Monitoring techniques should be chosen based on factors such as the patient's age and general health status, as well as on the procedure being performed and the agents used. Currently all available general monitoring systems used in the operating suite are available for use in the ED. These include electrocardiographic (ECG) monitoring (including monitors with ST segment monitoring capabilities), noninvasive blood pressure monitors, pulse oximetry, and capnography. Although none of these modalities have been shown to have an effect on outcome, because they are used during similar procedures in other parts of the hospital (mainly in the operating room), their use should be considered for involved cases.

Because the most common complication of systemically administered analgesia and sedation is bradypnea or apnea leading to hypoxemia, it would be prudent in most

situations to include pulse oximetry and capnography (see Chapter 6) as part of the monitoring regimen. Several caveats regarding pulse oximetry are important. For a reading to be valid, the oximeter should concurrently indicate the patient's heart rate. The best monitors will include a plethysmograph to ensure proper signal detection. If blood pressure monitoring is used, the pulse oximeter should be placed on a different extremity to prevent interruption of monitoring and unnecessary alarm activation. Both saturation and pulse alarms should be used during and especially after the procedure to ensure detection of adverse events.

Capnography may be helpful for several reasons. Application of supplemental $O_2$ during these procedures is the standard of care, and for this reason most patients will have a functional residual lung capacity that is high in $O_2$ , possibly approaching 100% in some individuals. Because pulse oximetry is not sensitive at this level of the oxyhemoglobin dissociation curve, patients may become apneic for up to 5 minutes before desaturation is detected and reported by the pulse oximeter. It also is difficult to continuously monitor respiratory rate and quality during many procedures, especially if drapes are required over the head or thorax as part of the procedure. Capnography using specially designed nasal cannulae with side-stream sampling capabilities can act as an immediate indicator of bradypnea or apnea and give some indication as to the quality of ventilation. If capnography is not included in the patient's monitoring, an argument could be made to withhold supplemental $O_2$ in order to detect desaturation and reverse it at its earliest time. Combined pulse oximetry, capnography, ECG, and blood pressure units are commercially available. These units may be particularly helpful in certain situations (e.g., pediatric sedation for diagnostic procedures such as computed tomography [CT] or magnetic resonance imaging [MRI]) in which the patient must be "remotely" monitored and interaction is minimized.

Automated blood pressure monitors should be used in the elderly and when certain agents or combinations of agents are used in which hypotension is a risk. These devices can ease demands by eliminating the cumbersome tasks of repeated manual measurements, but the initial reading as well as subsequent suspect readings should be confirmed with a traditional manual blood pressure check.

Although rhythm disturbances are a rare late complication from the agents discussed here, ECG monitoring is recommended in the elderly or in any patient with a known or suspected history of coronary artery disease. Although not studied, the use of ST segment ECG monitoring during the painful procedure may prove to be helpful in patients with certain risk factors.

Whatever monitoring modalities are chosen, proper interval

recording and documentation should be performed. Figure 35-2 is an example of a continuous flow sheet that should be used from the beginning of the procedure until the time of disposition. The clinician should resist the temptation to routinely reverse sedation or analgesia for the sole purpose of reducing the time and personnel required or the recovery time, or to allow for earlier discharge.

## Adjunctive Safety Measures

Formal deep sedation is a 2-person procedure. Because of the complexity of the procedure, 1 operator should be solely responsible for administering the drugs and monitoring the patient's status (vital signs, respiratory function, level of consciousness, etc). If opioids and benzodiazepines are used, it is recommended that naloxone and flumazenil be *available at the bedside.* All syringes should be clearly labeled with the full name of the drug they contain. Simply using initials on the syringes may lead to an "F" solution (fentanyl) being administered inadvertently as a "flush" solution. It is preferred to have an IV bag hung for flushing the line after each drug is administered. Some clinicians prefer to routinely administer $O_2$ during sedation rather than waiting for evidence of desaturation. The operator should be skilled at advanced airway maneuvers and airway equipment, including

| **TABLE 35-5** -- Postsedation and Analgesia Disposition Criteria |
|---|
| 1. Return to baseline verbal skills. * |
| 2. Return to age- and condition-appropriate vital signs. |
| 3. Return to baseline muscular control function (infants able to sit unattended; child or adult able to walk unassisted). * |
| 4. Return to baseline mental status. |
| 5. Patient able to take fluids by mouth prior to release. |
| 6. Pain controlled with oral pain medications. |
| 7. Responsible adult present who understands specific sedation and analgesia emergency department disposition criteria. |

* These items may take into account the patient's specific age and injuries

suctioning devices, which should be within reach of the patient's bedside (see Chapters 1 and 2) .

## Disposition

Prior to disposition, minimum motor, mental status, and cognitive criteria should be met based on age. Suggested disposition criteria are displayed in Table 35-5 . Both children and adults receiving analgesia and sedation should be accompanied home by a responsible adult who understands the disposition instructions. In addition, the patient's pain

---

**TABLE 35-6** -- Sample Adult Disposition Instructions after Systemic Analgesia and Sedation

1. Do not drive or operate heavy machinery for the next 24 hr.

2. Eat a light diet for the next 24 hr.

3. Take only your prescribed medications as needed, including any pain medication you were discharged with. Do not drink alcohol.

4. Do not make any important decisions or sign important documents for the next 24 hr. You may be forgetful due to medications that were administered.

5. If you experience any difficulty breathing or persistent nausea and vomiting, call or return to the emergency department or dial 911.

6. You should have a responsible person with you for the rest of the day and during the night.

---

should be controllable with oral pain medications prior to discharge. Sample

patient/guardian instruction sheets for adults and children are provided in Tables 35-6 and 35-7 .

**Policies**

Ideally, guidelines for administering systemic analgesia and sedation should be developed in a multidisciplinary fashion involving emergency physicians, anesthesiologists, pediatricians, nurses, and pharmacists, with attention paid to the special needs of the emergency patient and the ED environment. All have a vested interest and duty to alleviate acute pain and anxiety, and patients will benefit most when all cooperate to achieve this goal. The Joint Commission on Accreditation of Healthcare Organizations (JCAHO) requirements are sparse concerning analgesia and sedation administered by nonanesthesia personnel. [64]

The JCAHO does provide standards for cases in which a reasonable expectation exists that loss of protective reflexes will occur in a significant group of patients after analgesia and sedation is given. Written informed consent that is specific for elective conscious sedation is required by some institutions. Accepted standards include a preanesthesia assessment of patients, a plan for anesthesia, discussion of the

| **TABLE 35-7** -- Sample Pediatric Disposition Instructions after Systemic Analgesia and Sedation |
| --- |
| Your child has been given medicine for sedation and/or pain control. These medicines may cause your child to be sleepy and less aware of his/her surroundings, making it easier for accidents to happen as he/she walks or crawls. Because of these side effects, your child should be watched closely for the next 8 hr. In addition, we also suggest the following: |
| 1. No eating or drinking for the next 2 hr. If your child is an infant, you may resume half normal feedings in 1 hr. |
| 2. No playing for the next 24 hr that requires normal coordination, such as bike riding or jungle gym activities. |
| 3. No playing for 8 hr without an adult to watch and supervise. |

| |
|---|
| 4. No baths, showers, cooking, or use of potentially dangerous electrical appliances for 8 hr unless supervised by an adult. |
| If you notice anything unusual about your child or if your child cannot take fluids within the next 4 to 6 hr, please call the emergency department. |

anesthesia and consent prior to the procedure, appropriate monitoring of the patient's physiologic status during anesthesia, and appropriate disposition of the patient. A flow sheet should be used to document the procedure and recovery period. All of these requirements can be easily met by constructing policies accordingly, obtaining and using interactive and mechanical monitoring, and providing documentation

# Chapter 36 - Principles of Wound Management

**Richard L. Lammers**

Acute traumatic wound management is one of the most common procedures in the practice of emergency medicine. There are many areas of controversy in the medical literature; the clinician must be careful to keep the final outcome in focus and not become concerned with minor or clinically insignificant theoretical details.

Wound care involves much more than closure of divided skin. The primary goal of wound care is not the technical repair of the wound; it is providing optimal conditions for the natural reparative processes of the wound to proceed. Primary wound healing is not an inevitable process. For centuries, victims of wounds commonly experienced inflammation, infection, and extreme scarring; in fact, these processes were considered part of normal wound repair. Only in the late 19th century did surgeons first realize that sepsis could be separated from healing. [1]

The cornerstones of wound care are cleaning, debridement, closure, and protection. The primary objectives in wound care are as follow:

1. Preserving viable tissue
2. Restoring tissue continuity and function
3. Optimizing conditions for the development of wound strength
4. Preventing excessive or prolonged inflammation
5. Avoiding infection and other impediments to healing
6. Minimizing scar formation

This chapter reviews current strategies for achieving these goals.

## BACKGROUND

**Wound Healing**

Emergency physicians should have a basic understanding of the process of wound healing. Highlights of this complex phenomenon as they relate to clinical decision making are presented.

Wounds extending beneath the epithelium heal by forming scar tissue. Inflammation, epithelialization, fibroplasia, contraction, and scar maturation constitute the stages of this nonspecific repair process. [1] [2] [3] Inflammation is a beneficial response that serves to remove bacteria, foreign debris, and devitalized tissue--a biologic debridement. Polymorphonuclear and mononuclear leukocytes concentrate at the site of injury and phagocytose dead and dying tissue, foreign material, and bacteria in the wound. [4]

As white blood cells die, their intracellular contents are released into the wound. In excessive amounts, they form the purulence characteristic of infected wounds. Some

exudate is expected even in the absence of bacterial invasion; however, infection with accumulation of pus interferes with epithelialization and fibroplasia and impairs wound healing. Wounds contaminated with significant numbers of bacteria or foreign material may undergo a prolonged or persistent inflammatory response and not heal. Granuloma formation surrounding retained sutures is an example of chronic inflammation. [5]

As white blood cells remove debris within the wound, epithelial cells at the surface of the wound begin to migrate across the tissue defect. In most sutured wounds, the surface of the wound develops an epithelial covering impermeable to water within 24 to 48 hours. Eschar and surface debris impair this process. The epithelium thickens and grows downward into the wound and along the course of skin sutures. Although there is some "adhesiveness" to the wound edges during the first few days, this is lost because of fibrinolysis.

By the fourth or fifth day, newly transformed fibroblasts in the wound begin synthesizing collagen and protein polysaccharides, initiating the stage of scar formation known as *fibroplasia.* Collagen is the predominant component of scar tissue. Wound strength is a balance between the lysis of old collagen and the synthesis of new collagen "welding" the wound edges together. The amount of scar tissue is influenced by physical forces (e.g., the stresses imposed by movement) acting across the wound. In contrast, a wound that heals by secondary intention closes by contraction. *Contraction* is the movement of skin edges toward the center of the defect, primarily in the direction of underlying muscle.

Significant gains in tensile strength do not begin until approximately the fifth day following the injury. Strength increases rapidly for 6 to 17 days, more slowly for an additional 10 to 14 days, and almost imperceptibly for as long as 2 years (Fig. 36-1) . The strength of scar tissue never quite reaches that of unwounded skin. Although the process of collagen formation is essentially completed within 21 to 28 days, the scar widens for another month, and collagen continues to remodel and strengthen the wound for up to 1 year. [1] [5]

Decisions regarding the optimal time for suture removal and the need for continued support of the wound with tape are influenced by (1) wound tensile strength, (2) the period of scar widening, and (3) the cosmetically unacceptable effect of epithelialization along suture tracks. The wise physician will remind the patient that scars are quite red and noticeable at 3 to 8 weeks following closure. However, the appearance of a scar should not be judged before the scar is well into its remodeling phase. Therefore, any scar revision should be postponed until 6 to 12 months after injury.

Zitelli states, "The most important factor in predicting the cosmetic result is wound location. In general, wounds on concave surfaces heal with better cosmetic results than wounds on convex surfaces....Besides location, other factors such as *skin color, wound size*, and *wound depth* are helpful in predicting the cosmetic results of wounds healing by secondary intention."

Small, superficial wounds in lax, light-colored skin, especially in areas in which the skin is thin, result in less noticeable scars. Wounds on convex surfaces look better after

primary closure than following secondary healing. Repigmentation occurs over 3 to 5 years, even in large wounds that heal by secondary intention.

# INITIAL EVALUATION

The approach to the management of a particular wound depends on information gathered during history taking and on the results of physical examination. The decision on whether to close a wound immediately or after a period of observation is based on a variety of factors that affect the risk of infection. Some wounds may appear benign but conceal extensive and devastating underlying tissue damage. The discovery that an extremity wound was produced by a roller or wringer device, a high-pressure injection gun, high-voltage electricity, heavy and prolonged compressive forces, or the bite of a human or a potentially rabid animal radically alters the overall management of the affected patient. The American College of Emergency Physicians' "Clinical Policy for the Initial Approach to Patients Presenting with Penetrating Extremity Trauma" provides a useful approach to the evaluation of all wounds. [7]

## History

In the initial evaluation of a wound, the physician should identify all of the extrinsic and intrinsic factors that jeopardize healing and promote infection. These include the mechanism of injury, the time of injury, the environment in which the wound occurred, and the patient's immune status.

### Wound Age: The "Golden Period"

The likelihood of wound infection increases with the time that elapses before definitive wound care. A delay in wound cleaning is the most important variable, and may allow bacteria contaminating the wound to proliferate. A delay in treatment of a contaminated wound for as little as 3 hours can result in infection. However, there is evidence suggesting that wounds in highly vascular regions such as the face and scalp can be closed without increased risk as long as 24 hours after injury. [12] Contrary to popular belief, the "golden period"--the maximum time after injury that a wound may be safely closed without significant risk of infection-- *is not a fixed number of hours*.[13]

Many factors affect infection risk, and closure decisions should not be based solely on temporal considerations. Peacock points out that "a clean razor slice of highly vascular skin of the face might be closed safely 48 hours after injury, whereas a stable-floor-nail penetration of the foot of an elderly person might not be closed safely one minute after injury." [3] Berk and colleagues concluded that there is little change in wound infection rates in most areas of the body for up to 19 hours after a variety of traumatic injuries, and infection rates of simple wounds involving the head are essentially unaffected by the interval between injury and repair. [12] Hence, all of the data accumulated in the initial evaluation, both historical and physical, must be considered when making the decision to close a wound in a particular patient. In addition, the techniques of wound care in themselves may extend the golden period; a skillful clinician can often convert a

dangerously contaminated wound into a clean wound that can be safely closed. [3]

**Other Historical Factors**

Other factors that affect wound healing or the risk of infection include the patient's age and state of health. Patient age appears to be an important factor in host resistance to infection; those individuals at the extremes of age--young children and the elderly--are at greatest risk. [14] Infection

rates are reported to be higher in patients with medical illnesses (e.g., diabetes mellitus, immunologic deficiencies, malnutrition, anemia, uremia, congestive heart failure, cirrhosis, malignancy, alcoholism, arteriosclerosis, arteritis, collagen vascular disease, chronic granulomatous disease, smoking or chronic hypoxia, or liver failure), in obese patients, and in patients taking steroids or immunosuppressive drugs or those receiving radiation therapy. Shock, remote trauma, distant infection, bacteremia, denervation, and peripheral vascular disease also increase wound infection rates and slow the healing process. [4] [15] [16]

Additional information pertinent to decision making in wound management includes the following:

*Present medications* (specifically, anticoagulants and immunosuppressive drugs)

*Allergies* (especially to local anesthetics, antiseptics, analgesics, antibiotics, and tape)

*Tetanus immunization status*

*Potential exposure to rabies* (in bite wounds and mucosal exposures)

*Potential for foreign bodies* embedded in the wound, especially when the mechanism of injury is unknown or was associated with breaking glass or vegetative matter [17]

*Previous injuries and deformities* (especially in extremity and facial injuries)

*Associated injuries* (underlying fracture, joint penetration)

*Other factors* (availability for follow-up, patient understanding of wound care or compliance)

## Physical Examination

All wounds should be examined for amount of tissue destruction, degree of contamination, and damage to underlying structures. A common error in wound management is to assume that a traumatic wound is already contaminated and then,

during the examination, to contaminate it further. Despite the fact that all traumatic wounds are contaminated to some degree, these injuries should be examined using aseptic technique. It is prudent for the examiner to wear clean or sterile gloves and avoid droplet contamination from the mouth by maintaining distance or, preferably, by wearing a mask. [18] Traditionally, sterile gloves are used by the operator, but in many countries, this luxury is not available. There are no data quantifying a presumed increase in infection rate when nonsterile gloves are worn.

**Mechanism of Injury and Classification**

of Wounds

The magnitude and direction of the injuring force and the volume of tissue on which the force is dissipated determine the type of wound sustained. Three types of mechanical forces produce soft tissue injury: shear, tension, and compression forces. The resulting disruption or loss of tissue determines the configuration of the wound. Wounds may be classified into six categories:

1. *Abrasions.* Wounds caused by forces applied in opposite directions, resulting in the loss of epidermis and possibly dermis (e.g., skin grinding against road surface).
2. *Lacerations.* Wounds caused by shear forces that produce a tear in tissues. Tensile and compressive forces also cause separation of tissue. Little energy is required to produce a wound by shear forces (e.g., a knife cut). Consequently, little tissue damage occurs at the wound edge, the margins are sharp, and the wound appears "tidy." The energy required to disrupt tissue by tensile or compressive forces (e.g., forehead hitting a dashboard) is considerably greater than that required for tissue disruption by shear forces, because the energy is distributed over a larger volume. These lacerations have jagged, contused, "untidy" edges.
3. *Crush wounds.* Wounds caused by the impact of an object against tissue, particularly over a bony surface, which compresses the tissue. These wounds usually contain contused or partially devitalized tissue.
4. *Puncture wounds.* Wounds with a small opening and whose depth cannot be entirely visualized. Puncture wounds are caused by a combination of forces.
5. *Avulsions.* Wounds in which a portion of tissue is completely separated from its base and is either lost or left with a narrow base of attachment (a flap). [19] Shear and tensile forces cause avulsions.
6. *Combination wounds.* Wounds with a combination of configurations. For example, stellate lacerations caused by compression of soft tissue against underlying bone create wounds with elements of crush and tissue separation; missile wounds involve a combination of shear, tensile, and compressive forces that puncture, crush, and sometimes avulse tissue. [18]

**Contaminants (bacteria and foreign material).**

Numerous factors affect the risk of wound infection, but the primary determinants of infection are the amount of bacteria and dead tissue remaining in the wound. [20] Also of

importance is the ability of the patient's immune system to respond to bacterial invasion and the presence of local tissue ischemia or hypoxia. [21]

Essentially all traumatic wounds are contaminated with bacteria to some extent. The number of bacteria remaining in the wound at the time of closure is directly related to the risk of infection. A critical number of bacteria must be present in a wound before a soft tissue infection develops. In experimental wounds produced by shear forces, an inoculum of $10^6$ aerobic bacteria per gram of tissue inevitably produces wound infection in time. When the mechanism of injury involves a compressive force, the infective dose of bacteria is $10^4$ bacteria per gram of tissue. If bacterial counts after injury (or after wound management) are below this level, the wound has a very low probability of becoming infected.

Surgical operations are categorized on the basis of the relative levels of bacterial contamination of the wounds. Most traumatic wounds fall into one of two categories:

1. *Contaminated wounds.* Traumatic wounds <12 hours old (the most common type of wound seen in emergency departments).
2. *Dirty wounds.* Wounds heavily contaminated with pathogenic organisms, those with significant numbers of bacteria associated with large amounts of devitalized tissue, or traumatic wounds older than 12 hours. [21] Infection rates in series of contaminated wounds of all types range from 1.1 to 21%; rates in series of dirty wounds range from 7 to 38%. [14] The nature and amount of foreign material contaminating the wound often determine the type and quantity of bacteria implanted. The presence of undetected reactive foreign bodies in sutured wounds almost

   guarantees an infection. Although bullet or glass fragments by themselves rarely produce wound infection, these foreign bodies may carry particles of clothing, gun wadding, or soil into the wound. Minute amounts of organic or vegetative matter, feces, or saliva carry highly infective doses of bacteria. The bacterial inoculum from human bites often contains 100 million or more organisms per milliliter of saliva. [28] Inorganic particulate matter, such as sand or road surface grease, usually introduces few bacteria into a wound and has little chemical reactivity; these contaminants are relatively innocuous. However, soil containing a large proportion of clay particles readily promotes infection. Presumably because of their marked chemical reactivity, clay particles damage local tissue defenses. [29] Soils with a high organic content, such as those in swamps, bogs, and marshes, also have a high infection potential. [30] Most wounds encountered in the practice of emergency medicine have low initial bacterial counts. If wound cleaning and removal of devitalized tissue are instituted before bacteria within the wound enter their accelerated growth phase (3 to 12 hours following the injury) and if one uses aseptic technique in examining and managing these wounds, bacterial counts will remain below the critical number needed to initiate infection.

**Wound Location**

The anatomic location of the wound has considerable importance in the risk of infection.

Bacterial densities on the skin surface range from a few thousand to millions per square centimeter. [30] Areas with endogenous microflora in numbers sufficient to infect a wound ($>10^5$ bacteria/cm$^2$ ) include the hairy scalp, the forehead, the axilla, the perineum, the foreskin of the penis, the vagina, the mouth, intertriginous areas, and the nails. In other regions, skin bacteria are sparse ($10^2$ to $10^3$ bacteria/cm$^2$ ) and are not a source of infection. [18] Wounds in regions of high vascularity, such as the scalp and the face, more easily resist bacterial incursions. The high vascularity of the scalp probably accounts for extremely low infection rates with scalp injuries, despite the large numbers of endogenous microflora. Distal extremity wounds, in contrast, are more at risk for the development of wound infections than are injuries of most other parts of the body. [14] Wounds in ischemic tissue are notoriously susceptible to infection. [31]

### Devitalized Tissue

Identifying devitalized tissue is an important part of the examination of a wound. Tissue damage lowers the resistance of the wound to infection. Devitalized or necrotic tissue enhances the possibility of infection in a wound by providing a culture medium in which bacteria proliferate, by inhibiting leukocyte phagocytosis, and by creating an anaerobic environment suitable for certain bacterial species.

### Underlying Structures

Identification of injury to underlying structures such as nerves, vessels, tendons, joints, bones, or ducts may lead the emergency physician to forgo wound closure and consult a surgical specialist. Procedures such as joint space irrigation, reduction and debridement of compound fractures, neurorrhaphy, vascular anastomosis, and flexor tendon repair are best accomplished in the controlled setting of the operating room, in which optimal lighting, proper instruments, and assistance are available.

## CLEANING

The wound should be cleaned as soon as possible after evaluation. Although most wounds are contaminated initially with less than an infective dose of bacteria, given time and the appropriate wound environment, bacterial counts may reach infective levels. The goals of wound cleaning and debridement are the same: (1) to remove bacteria and reduce their numbers below the level associated with infection, and (2) to remove particulate matter and tissue debris that would lengthen the inflammatory stage of healing or allow the growth of bacteria beyond the critical threshold. [16]

### Patient Preparation

Prior to examining, cleaning, exploring, or repairing wounds, medical procedures should be explained to patients to allay fears and to engender patient cooperation. In general, all wound care should be performed *with the patient in a supine position,* since fainting is a common occurrence once wound preparation has commenced (Fig. 36-2) . Even the most hardy or brave patient may faint at the sight of a needle, scalpel, or blood. Patient falls are a serious source of comorbidity and litigation. Likewise, relatives and

friends should be allowed to stay with the patient only after their propensity for fainting has been assessed and they have been properly cautioned. The wise physician will insist that any significant others who remain in the room to support the patient sit during the procedure and report any perceived dizziness or nausea.

## Wound Handling

Anyone cleaning, irrigating, or suturing wounds should wear protective eyewear and a mask, as virtually any patient may be seropositive for the human immunodeficiency virus (HIV). Although mucosal exposure to blood or tissue products that are contaminated by HIV is considered a relatively low risk for subsequent infection, universal precautions are currently recommended.

Thorough cleansing of bacteria, soil, and other contaminants from a wound cannot be accomplished without the patient's cooperation. Scrubbing most open wounds is painful, and the patient's natural response is withdrawal. Therefore, *local or regional anesthesia often must precede the examination and cleaning of a wound.* Approaches to wound anesthesia are discussed in detail in Chapters 31 , 32 , 33 , and 34 .

Despite adequate anesthesia, the patient may be unable to cooperate because of apprehension. The physician should explain the wound cleansing procedure to the patient and should provide assurance that everything possible will be done to minimize pain. Reassurance may not alleviate the fears of young children, and both sedation and physical restraining devices must be used. Approaches to sedation using parenteral sedative-hypnotics and narcotic agents and the use of inhaled nitrous oxide are discussed in Chapter 35 .

The two primary methods of wound cleaning are mechanical scrubbing and irrigation. Soaking a wound in a saline or antiseptic solution before the arrival of a physician

**Figure 36-2** What is wrong with this picture? Although the patient stated that blood and needles did not bother her, she fainted shortly after this picture was taken, nearly falling to the floor. The cautious physician prepares and repairs wounds only when the patient is recumbent (preferably supine).

is of little value. Indeed, soaking a wound in saline may actually increase bacterial counts. [32] The methods of scrubbing and irrigation are reviewed below.

## Mechanical Scrubbing

Initially, a wide area of skin surface surrounding the wound should be scrubbed with an antiseptic solution to remove contaminants that in the course of wound management might be carried into the wound by instruments, suture material, dressings, or the physician's gloved hand. Minimal aseptic technique requires the use of gloves during the cleaning procedure. It is important to remove all nonabsorbable particulate matter; any such material left in the dermis may become impregnated in the healed tissue and result in a disfiguring "tattoo" effect. [5] However, scrubbing the internal surface of a wound is controversial. Although scrubbing a wound with an antiseptic-soaked sponge

does remove foreign particulates, bacteria, and tissue debris, an abrasive sponge may inflict more damage on tissue and provoke more inflammation. [33] [34]

Some physicians reserve mechanical scrubbing for "dirty" wounds contaminated with significant amounts of foreign material. If irrigation alone is ineffective in removing contaminants from a wound, the wound should be scrubbed. Because the amount of damage inflicted on tissues by scrubbing is correlated with the porosity of the sponge, a fine-pore sponge (e.g., Optipore sponge [90 pores per linear inch]) should be used to minimize tissue abrasion. [33] [35] Detergents have an advantage over saline because they minimize friction between the sponge and tissue, thereby limiting tissue damage during scrubbing. Detergents also dissolve particles, helping to dislodge them from the wound surface. Unfortunately, many of the available detergents are toxic to tissues. [33] [36]

## Antiseptics During Cleaning

For many years, antiseptic solutions have been used for their antimicrobial properties in and around wounds (Table 36-1) . Studies of antiseptics in wounds demonstrate that there is a delicate balance between killing bacteria and injuring tissue. [37] Intact skin can withstand strong microbicidal agents, whereas leukocytes and the exposed cells of skin and soft tissue can be damaged by these agents. [16]

Many antiseptic solutions have been used for cleaning wounds. Povidone-iodine (Betadine) is widely available as a 10% stock solution. Although studies on the efficacy and safety of povidone-iodine solution have shown variable results, [41] it appears that dilute povidone-iodine solution in concentrations of <1% is both safe and effective for use in contaminated traumatic wounds. The precise concentration that provides the most benefit is unknown. In contrast, povidone-iodine surgical scrub (Betadine scrub) and hexachlorophene (pHisoHex) both contain anionic detergents that are harmful to tissues and that increase infection rates when used in wounds. Aqueous iodine is irritating and corrosive to tissue and should not be used in any wound. [38]

In vitro studies have demonstrated the toxicity of chlorhexidine gluconate-alcohol (Hibiclens) to the cellular components of blood. [41] Hydrogen peroxide is used by some clinicians for its effervescent effect in cleaning wounds. However, because peroxide is hemolytic, it is best to use it only to clean surrounding skin encrusted with blood and coagulum or to soak off adherent blood-soaked dressings.

| Agent | Biologic Activity | Tissue Toxicity | Systemic Toxicity * | Potential Uses | Comments |
|---|---|---|---|---|---|

**TABLE 36-1** -- Summary of Agents Used for Wound Care

| | | | | | |
|---|---|---|---|---|---|
| Povidone-iodine surgical scrub (Betadine 7.5%) | Virucidal; strongly bactericidal against gram-positive and gram-negative organisms | Detergent can be toxic to wound tissues | Painful to open wounds; other reactions extremely rare | Hand cleanser | Iodine allergy possible; systemic absorption of iodine from burns, open wounds; not routinely used in open wounds |
| Povidone-iodine solution (Betadine 10%) | Same as povidone-iodine scrub; virucidal, bactericidal | Minimally toxic to wound tissues at full strength; 1% solution has no significant tissue toxicity | Extremely rare | Wound periphery cleanser; diluted to 1% for wound irrigation | Probably the safest and most effective product currently available; iodine is active agent, povidone is carrier molecule; iodine allergy possible; systemic absorption of iodine from burns, open wounds; dilute 10:1 (saline:Betadine) if used to irrigate wounds |
| Chlorhexidine gluconate (Hibiclens) | Strongly bactericidal against gram-positive organisms, less strong against gram-negative bacteria | Ionic detergent can be toxic to tissue/cellular components; eye and inner ear toxicity | Extremely rare | Hand cleanser | Generally avoid use in open wounds; not for use in eye/ear |

| Polaxamer 188 (Shur-Clens; Pluronic F-68) | No antibacterial or antiviral activity | None known; does not inhibit wound healing | None known | Wound cleanser (particularly useful on face) | Nonionic detergent used for cleansing properties; nontoxic even with IV use; will not damage eye/cornea; lack of antibacterial properties limits use |
|---|---|---|---|---|---|
| Hexachlorophene (pHisoHex) | Bacteriostatic against gram-positive bacteria, poor activity against gram-negative bacteria | Detergent can be toxic to wound tissues | Possibly teratogenic with repeated use | Alternative hand cleanser; not used on open wounds | Systemic absorption causes neurotoxicity |
| Hydrogen peroxide | Very weak antibacterial agent | Toxic to tissue/red cells | Extremely rare | Wound cleanser adjunct; very weak antiseptic properties | Breaks down to water and oxygen; foaming activity useful to remove debris/coagulated blood |

* Based largely on in vitro studies/animal data

Peroxide should not be used on granulation tissue, because oxygen bubbles lift newly formed epithelium off the wound surface. [42] Hexachlorophene was used as an antiseptic for a time but was found to be neurotoxic and teratogenic through skin absorption. Its narrow antimicrobial spectrum makes it no more effective than ordinary soap and water. [43] Quaternary ammonium compounds are less toxic to tissue but have a limited antimicrobial spectrum; gram-positive organisms are more susceptible to these solutions than are gram-negative bacteria. Benzalkonium chloride (Zephiran) is inactivated by soaps, detergents, blood, and other organic matter. Furthermore, *Pseudomonas* has been known to proliferate in stored solutions. Consequently, use of benzalkonium chloride has fallen into disfavor. [35]

Nonantiseptic nonionic surfactants are attractive alternatives to these toxic cleansing agents. In contrast to antiseptic solutions, these preparations cause no tissue or cellular

damage, leukocyte inhibition, or impairment in wound healing. The solutions also cause no corneal injury, conjunctival irritation, or pain on contact with the wound. [41] [44] Poloxamer 188 (Pluronic F-68, Shur-Clens, and Pharma Clens) is nontoxic, even when administered intravenously, and nonallergenic. This pluronic polyol has no antibacterial activity, but scrubbing experimental wounds with poloxamer has reduced infection rates, proving its ability to cleanse a wound effectively and atraumatically. [33]

From a clinical standpoint, for most simple wounds, there may be little practical difference between many irrigation and cleaning solutions. The above cited concerns about toxicity of solutions are largely theoretical, suggested by in vitro studies, or inferred from animal data. A survey of wound practice by Howell and Chisholm suggests that a wide range of solutions and techniques are in clinical use in the United States. [45] However, it is important to distinguish

between skin antiseptics and irrigating solutions. As a general rule, commercially available antiseptics should be used *only to clean intact skin,* and one should avoid introduction of significant amounts of these products into open wounds. It is recommended that most open wounds be irrigated with copious amounts of saline or a dilute (1%) povidone-iodine solution. Pluronic polyols may be considered when the wound is near mucous membranes and dilute povidone-iodine may be particularly irritating.

**Irrigation**

Properly performed irrigation is effective in removing particulate matter, bacteria, and devitalized tissue that is loosely adherent to the edges of the wound and trapped within its depths. Rodeheaver and colleagues studied the effect of irrigating experimental wounds contaminated with 20 mg of soil. Irrigating wounds with 400 mL of fluid at 1 psi removed 48.6% of the soil, whereas increasing the pressure to 15 psi removed 84.8%, reducing the infection rate from 100% to 7%. [46] *The effectiveness of irrigation is determined primarily by the hydraulic pressure at which the irrigation fluid is delivered.*[47] [48] Bulb syringes or gravity flow irrigation devices deliver fluid at low pressures and are ineffective in ridding wounds of small particulate matter or in lowering wound bacterial counts. The flow rate of irrigation fluid delivered through IV tubing with a blood pressure cuff inflated around a collapsible plastic IV bag provides <10 psi of irrigation pressure.

The pressure that can be delivered with a syringe varies with the force exerted on the plunger of the syringe and with the internal diameter of the attached needle. A simple irrigation assembly consisting of a 19-ga plastic catheter or needle attached to a 35-mL syringe produces 25 to 40 psi when the barrel of the syringe is pushed with both hands. [48] This high-pressure irrigation system removes significant numbers of bacteria and a substantial amount of particulate matter from the wound surface (Fig. 36-3) . Commercial irrigation systems with a ring-handled syringe and a one-way valve that connects into a standard IV solution are available (Travenol pressure irrigation set, code number 2D2113, or Irrijet, Ackrad Laboratories, Garwood, NJ).

Pulsatile jet irrigation of wounds creates pressures of 50 to 70 psi but does not appear to drive significant amounts of bacteria or surface contaminants into the soft tissues of

the wound. [49] [50] Although jet irrigation can damage tissue defenses and spread fluid laterally into loose areolar tissue, [51] it is more effective in cleaning wounds, less traumatic to tissues, and less likely to produce edema than conventional scrubbing with a brush. However, this technique should generally be reserved for use in heavily contaminated wounds in which syringe irrigation proves to be ineffective. The jet irrigation system used by many investigators was a Water Pik unit (Teledyne Aquatic Corp.) with a sterile tip nozzle held approximately 4 cm from the wound. [35] [50] [51]

Minimum recommended volumes of irrigation fluid vary, but for average-sized wounds, 100 to 300 mL should be used. [35] Greater volumes may be required for larger or heavily contaminated wounds. Irrigation should continue until all visible, loose particulate matter has been removed. Approximately 20% of open, undated bottles of "sterile" saline and water are contaminated; only solutions in bottles opened within the past 24 hours should be used to irrigate wounds. [52] [53] A potential complication of wound irrigation is that infectious material can be splashed into the face of the physician, even when the tip of the irrigation device is held below the wound surface. A plastic cup device that fits on the end of a syringe (ZeroWet Splashshield) can be used to contain the splatter (Fig. 36-4) . The wound should be positioned to allow continuous drainage of fluid during irrigation by any method. [54]

**Antibiotic Solutions for Irrigation**

Antibiotic solutions have been instilled directly into wounds or used as irrigation solutions. Halasz reviewed the studies of several investigators who analyzed the technique of irrigating wounds with antibiotic solutions, including ampicillin, a neomycin-bacitracin-polymyxin combination; tetracycline; penicillin; kanamycin; and cephalothin. He concluded that "organisms in the wound can be exposed to adequate concentrations of antibiotics, and that the concentration of these drugs in the wound remains in the bactericidal range for long periods of time, far exceeding that obtainable by systemic administration." [55] The use of antibiotics in irrigation solutions in lieu of antiseptic solutions avoids the tissue destruction of the antiseptics but theoretically risks topical sensitization of the patient to the antibiotic and the development of toxic tissue levels of the antibiotic. To date, there have been no reports of these complications. Within 3 hours of injury, a proteinaceous coagulum forms within the wound, surrounding the bacteria and probably preventing their contact with topical or systemic antibiotics. Therefore, the wound should be scrubbed before irrigation with an antibiotic solution. [35] [56] The indications for antibiotic solutions in cleaning wounds have not been defined, and this practice is not considered standard, although a decrease in wound infection has been demonstrated in several studies.

**Recommendations for Cleaning the Wound**

The prerequisites of any wound-cleaning technique are a calm or sedated patient, satisfactory anesthesia, and a thorough scrub of the skin surface adjacent to the wound. The importance of ridding the wound of major contaminants and infective doses of bacteria is unquestionable. Two strategies to accomplish the goals of wound cleaning are apparent from this discussion. The contaminated or "dirty" wound can be irrigated or both scrubbed and irrigated with a 1% povidone-iodine solution (Betadine preparation,

*not* Betadine scrub). This should be followed by flushing with a 0.9% saline solution. As an alternative, the wound can be scrubbed with pluronic polyols and irrigated with an antibiotic-normal saline solution. *Only pluronic polyols or saline should be used near the eyes.* All scrubbing should be performed with a soft, fine-pore sponge, and high-pressure techniques should be used for all irrigation. The use of hydrogen peroxide on open wounds is discouraged. Gentle scrubbing with poloxamer and normal saline high-pressure irrigation both appear to be satisfactory methods for cleaning minimally contaminated wounds.

Some clinicians routinely irrigate wounds, especially extremity wounds, with tap water instead of sterile saline. The technique has not been studied extensively, but given that patients almost always irrigate their wound under the tap prior to presentation and that the practice is widespread in hospitals, it is unlikely to be of major harm. Angeras and coworkers demonstrated that tap water produced a lower infection rate than saline (5.4 vs 10.3%). [59] Even though bacteria were cultured from the faucets, these organisms did not account for a single wound infection. The obvious advantage of using tap water is the large volume of irrigant that can be quickly applied to an open wound.

**Preparation for Wound Closure**

Before debridement or wound closure, the wound must be prepared and draped. Body hair should generally be left intact, since wounds that are shaved demonstrate *higher* infection rates. For wounds in hair-bearing areas, hair should be removed by clipping only if the hair interferes with the procedure. [61] Stubborn hairs that repeatedly invade the wound during suturing can be coated with petrolatum jelly or water-soluble ointments to keep them out of the field. Eyebrows should not be shaved, because critical landmarks needed for exact approximation would be lost. Although shaved eyebrows will grow back eventually, shaving produces an undesirable cosmetic effect.

The skin surface adjacent to the wound (not the wound itself) should be disinfected with a standard 10% povidone-iodine or chlorhexadine gluconate (Hibiclens) solution. The solution is painted widely on the skin surrounding the wound but should not cover the interior of the wound itself. After hand washing, the physician and any assistants involved in the procedure must wear gloves. Visible talc or other powders on the gloves should be rinsed before touching the patient's wound. [30]

Face masks are recommended and are mandatory for any clinician with a bacterial upper respiratory infection. Because droplets of saliva may leak even from around the edges of a face mask, talking in proximity to the wound must be avoided. [63]

A single fenestrated drape or multiple folded drapes are placed over the wound site. For hand wounds, a sterile glove may be applied to the patient to provide a sterile field in lieu of a fenestrated drape. The area to be sutured can be exposed by cutting the glove, and the extremity can be placed on a sterile towel. This technique provides a clean field without the need to continually adjust the drape or to operate through a small opening. If the wound has not yet been anesthetized, anesthesia can be provided at this time (see Chapters 31 , 32 , 33 , and 34 for details).

The entire depth and the full extent of every wound should be explored in an attempt to locate hidden foreign

bodies, particulate matter, bone fragments, and any injuries to underlying structures that may require repair. The clinician should avoid the temptation to initially explore wounds with a finger in search of a foreign body or to assess wound characteristics. Embedded glass, metal fragments, or sharp pieces of bone may cut the physician, exposing him or her to contamination with the patient's blood. Exploration with a metal probe and the liberal use of radiographs, combined with direct vision and good lighting, is a much safer approach. Lacerations through thick subcutaneous (SQ) adipose tissue are treacherous, because large amounts of particulate matter can be totally obscured in deeper folds of tissue. Unless a careful search is undertaken, these contaminants may be left in the depths of a sutured wound, and infection usually follows. Some physicians are reluctant to extend lacerations to properly clean or explore them; however, opening the wound to permit adequate visualization may be needed for successful wound preparation.

### Debridement

Debridement is of undisputed importance in the management of the contaminated wound. With this technique, the physician can remove tissue impregnated with foreign matter, bacteria, and devitalized tissue that otherwise impairs the ability of the wound to resist infection and prolongs the period of inflammation. [20] Debridement also creates a tidy, sharp wound edge that is easier to repair and results in a more cosmetically acceptable scar.

If the wound already is clean and the edges are viable, sharp debridement may not improve the outcome. Irregular wounds have greater surface areas than do linear lacerations. Because skin tension is distributed over a greater length, the scar width is usually less in jagged wounds than if the wound is converted to an elliptical defect with tidy edges. If the edges are devitalized or contaminated, the wound edges must be debrided. To avoid a wide scar in this situation, the wound can be undermined.

### Excision

If significant contamination occurs in areas in which there is a laxity of tissues and if no important structures, such as tendons or nerves, lie within the wound, the entire wound may be excised. [3] This technique is the most effective type of debridement, because it converts the contaminated traumatic wound into a clean surgical wound (Fig. 36-5) . Complete excision of grossly contaminated wounds such as animal bites allows primary closure of such wounds with no greater risk of infection than in relatively uncontaminated lacerations. [35] Wounds of the trunk, the gluteal region, or the thigh are amenable to this technique. If necessary, the clinician can judge the adequacy of the excision by coloring the wound surface with a vital dye. The clinician then creates a new wound by excising all dyed tissue. [20] Most traumatic wounds can be excised with an elliptical excision. A lenticular configuration should be marked superficially around the

wound with the blade of a No. 15 scalpel by cutting only the epidermis.

If a puncture wound is being excised, the axis of the excision should parallel a wrinkle, a skin line, or a line of dependency or facial expression, and the long axis should be three to four times as great as the short axis. The clinician may plan this type of excision by premarking the skin with a surgical marking pen. Tension should be placed on the surrounding skin with a finger or a skin hook. With the clinician's hand steadied on the table or on the patient, the No. 15 blade is used to cut through the skin at right angles or at slightly oblique angles to the skin surface. If complete excision of the entire depth of the wound is not necessary, the tissue scissors may be used to cut the edge of the wound, following the path premarked in the epidermis by the scalpel blade. If a complete excision is desired, the incision on each wound edge should be carried past the deepest part of the wound (see Fig. 36-5) .

Excision should be planned carefully; excessive removal of tissue can create a defect that is too large to close. In hair-bearing areas of the face, particularly through the eyebrows, the incision should be angled parallel to the angle of hair follicles to avoid linear alopecia (Fig. 36-6) (Figure Not Available) . The wedge of excised tissue should be removed carefully, without contaminating the fresh wound surface. [64] [65]

Puncture wounds of the feet may contain foreign bodies that are difficult to remove. Contaminated punctures or those likely to contain foreign bodies (e.g., nail punctures through shoes) can be excised by removing a small cone of surrounding tissue with a scalpel or with a 4-mm disposable punch biopsy and the wound irrigated a second time. [66] [67]

**Figure 36-6** (Figure Not Available) Excision through an eyebrow. Use an angled incision to remove tissue in the eyebrow, thus avoiding further injury of hair follicles. *(From Dushoff IM: A stitch in time. Emerg Med 5(1):2, 1973. Reproduced by permission.)*

**Selective Debridement**

Complete excision is impossible for most wounds because of insufficient skin elasticity, and selective debridement must be used. [3] [20] Simple excision of a wound of the palm or the dorsum of the nose will make approximation of the resulting surgical wound edges difficult. Stellate wounds and wounds with an irregular, meandering course have greater surface areas and less skin tension per unit length than do linear lacerations. In some cases excision of an entire wound would result in the loss of too much tissue (i.e., produce a gaping defect and excessive tension on the wound edges when closed). This problem can be avoided with selective debridement and approximation of the irregular wound edges. This technique involves sharp debridement of devitalized or heavily contaminated tissue in the wound piece by piece and eventual matching of one edge of the wound with the other. Selective debridement is time consuming but preserves more surrounding tissue.

Identifying devitalized tissue in a wound remains a challenging problem. Tissue with a narrow pedicle or base, especially distally based, narrow flaps on extremities, is unlikely to survive and should be excised. Sometimes a sharp line of demarcation distinguishes

devitalized skin and viable skin, but in most wounds there is usually only a subtle bluish discoloration. The comparison of capillary refill in wounded tissue with that in adjacent skin is probably the most practical test for tissue viability available to the emergency physician. [68] If circulation is adequate, viable tissue becomes hyperemic following the release of a proximal tourniquet.

In heavily contaminated wounds, especially those with abundant adipose tissue, all exposed fat and all fat impregnated with particulate matter should be removed. The SQ adipose tissue attached to large flaps or to avulsed viable skin should be debrided before reapproximation of the wound; removal of this fatty layer allows better perfusion of the flap or the graft.

Contaminated bone fragments, nerves, and tendons are almost never removed. Every effort should be made to clean these structures and return them to their place of origin, because they may be functional later. [69]

Instruments usually required for debridement include two fine single- or double-pronged skin hooks, a scalpel with a No. 15 blade, tissue scissors, hemostats, and a small tissue forceps. The jagged wound edges are stabilized with skin hooks or forceps, and the scalpel or scissors are used to cut away devitalized tissue from one end of the wound to the other. Fascia and tendons perform important functions despite potential loss of viability. If they can be cleaned adequately, these tissues should not be debrided. They may be left in wounds as free grafts and covered by viable flaps of tissue. [70]

Following debridement or excision, the wound should be irrigated again to remove any remaining tissue debris.

**Control of Hemorrhage**

Wound exploration or cleaning sometimes induces bleeding. Hemostasis is essential at any stage of wound care. Not only does persistent bleeding obscure the wound and hamper wound exploration and closure, but also hematoma formation in a sutured wound separates wound edges, impairs healing, and risks dehiscence or infection. If bleeding is not a problem before wound debridement, it frequently becomes a complication once the wound edges are excised.

Several practical methods of achieving hemostasis are available to the emergency physician. Sustained direct pressure with gloved fingers, gauze sponges, or packing material, combined with elevation, is usually effective in immediately controlling a single bleeding site or a small number of sites until cut ends of vessels constrict and coagulation occurs. In a patient with multiple injuries and several urgent problems, hemorrhage can be controlled temporarily with a compression dressing. Several absorptive sponges are applied directly over the bleeding site, and these are secured in place with an elastic bandage (e.g., Ace wrap) or elastic adhesive tape (Elastoplast). Pressure is provided by the elasticity of the bandage. The bleeding part should be elevated. Wound care can then be deferred while the physician attends to more

pressing matters.

Although simply crushing and twisting the end of a small vessel with a hemostat avoids the introduction of suture material into the wound, this method provides unreliable hemostasis. Ligation of the vessel with fine absorbable suture material is preferred. Bleeding ends of vessels are clamped with fine-point hemostats, providing immediate hemostasis. Because nerves often course with these vessels, all clamping should be done under direct visualization. The tip of the hemostat should project beyond the vessel to hold a loop of a ligature in place (Fig. 36-7) . While an assistant lifts the handle of the hemostat, a synthetic absorbable 5-0 or 6-0 suture is passed around the hemostat from one hand to the other (Fig. 36-8) (Figure Not Available) . The first knot is tied beyond the tip of the hemostat. Once the suture is securely anchored on the vessel, the hemostat is released. [65] [71]

In practice, the emergency physician seldom has an assistant available to ligate vessels by this method. MacDonald describes a technique that enables a single operator to maintain tension on the ligature while removing the hemostatic clamp. [72] A needle holder is used to grasp one tail of the ligature; the other end is held by the third, fourth, and fifth fingers of the left hand. As the clamp held in the right hand is removed from the vessel, the needle holder is moved

away from the left hand by extending the thumb and the index finger, maintaining tension on the ligature. The right hand can discard the clamp, grasp the needle holder, and complete the tie (Fig. 36-9) (Figure Not Available) . [72] Three knots are sufficient to hold the ligature in place. The ends of the suture should be cut close to the knot to minimize the amount of suture material that is left in the wound.

Physicians can make the mistake of spending excessive time attempting to tie off small bleeding vessels while the patient slowly exsanguinates. In highly vascular areas, such as the scalp, it is sometimes best to suture the laceration following wound exploration and irrigation, despite active bleeding; the pressure exerted by the closure will usually stop the bleeding. If bleeding is too brisk to permit adequate wound evaluation and irrigation, hemorrhage can often be controlled by clamping and everting the galea or dermis of each wound edge using hemostats. Lemos and Clark propose the use of scalp clips as an alternative. [71]

Cut vessels that retract into the wall of the wound may frustrate attempts at clamping and ligation. Bleeding should be controlled first by downward compression on the tissue. A suture is passed through the tissue twice, using a figure-of-eight or horizontal mattress stitch, and then tied. The double thread will constrict the tissue containing the cut vessel (Fig. 36-10) . The disadvantage of this method is that the tissue constricted by the ligature may necrose and leave devitalized tissue in the wound.

Vessels with diameters greater than 2 mm should be ligated. Those smaller than 2 mm that bleed despite direct pressure can be controlled by pinpoint electrocautery. A dry field is required for an effective electrical current to pass through the tissues; if sponging does not dry the field, a suction-tipped catheter should be used. Trauma is minimized by using fine-tipped electrodes to touch the vessel or by touching the active electrode of

the electrocautery unit to a small hemostat or fine-tipped forceps gripping the vessel. [1] The power of the unit should be kept to the minimum level required for vessel thrombosis. Bipolar coagulation (such as that provided by the Bovie unit) is preferred over monopolar coagulation because it produces approximately one third less necrosis of surrounding tissue. [35] If the amount of tissue cauterized is kept to a minimum, wound healing is no more compromised by this technique than by ligation. Cauterization of medium- and small-sized vessels can quickly provide hemostasis. Self-contained, sterilizable, battery-powered coagulation units also are available. Vessels are cauterized by the direct application of a heated wired filament. Although these units may damage more surrounding tissue than electrocautery units, they are compact and simple and thus well suited for use in the emergency center (Fig. 36-11) .

Epinephrine is an excellent vasoconstrictor. Topical epinephrine (1:100,000) on a moistened sponge can be applied to a wound to reduce the bleeding from small vessels. Combined with local anesthetics, concentrations of 1:100,000 and 1:200,000 prolong the effect of the anesthetic and provide some hemostasis in highly vascular areas. Because vasoconstrictors theoretically may increase the risk of wound infection, they should be restricted to situations in which widespread small vessel and capillary hemorrhage in a wound is not controlled by direct pressure or cauterization.

Fibrin foam, gelatin foam, and microcrystalline collagen may be used as hemostatic agents. Their utility is limited in that vigorous bleeding will wash the agent away from the bleeding site. Their greatest value may be in packing small

**Figure 36-9** (Figure Not Available) *A,* Ligature tension is maintained with one hand by grasping one tail of the suture with a clamp and keeping its base between the thumb and the forefinger. *B,* As the pedicle clamp is removed with the other hand, the ligature is tightened by extending the flexed thumb and index finger to the desired tension. Then the pedicle clamp is discarded from the right hand, and further knots are applied in the usual fashion. *(Modified from MacDonald RT: Maintenance of ligature tension by a single operator with simultaneous removal of a hemostatic clamp. Am J Surg 143:770, 1982. Reproduced by permission.)*

cavities from which there is a constant oozing of blood. [65] In most simple wounds with persistent but minor capillary bleeding, apposition of the wound edges with sutures provides adequate hemostasis.

**Tourniquets**

If bleeding from an extremity wound is refractory to direct pressure, electrocauterization, or ligation, or if the patient presents with exsanguinating hemorrhage from the wound, a tourniquet can be used to control the bleeding temporarily. Tourniquets also are helpful in examining extremity lacerations by providing a bloodless field. However, they can cause injury in three ways:

**Figure 36-10** Ligation of a retracted, bleeding vessel. *A,* Horizontal mattress technique. *B,* Figure-of-eight technique.

1. They can produce ischemia in an extremity.

2. They can compress and damage underlying blood vessels and nerves.
3. They can jeopardize the survival of marginally viable tissue.Although problems rarely develop from tourniquets used in routine wound care, potential problems can be minimized if (1) there is a limit placed on the total amount of time that an extremity is confined by a tourniquet, and (2) excessive tourniquet pressures are avoided. It is also imperative that all tourniquets be removed prior to releasing the patient. A tourniquet may be overlooked if a bulky dressing is applied over the tourniquet.A single-cuff tourniquet (sphygmomanometer cuff) placed around an arm or a leg effectively stops distal venous or arterial bleeding without crushing underlying structures. The length of time that a tourniquet may remain in place is

**Figure 36-11** Battery-powered cautery.

limited by the development of pain underneath and distal to the tourniquet. This occurs within 30 to 45 minutes in a conscious patient, well within the limits of safety. [73] Before application of the tourniquet, the injured extremity should be elevated and then manually exsanguinated to prevent bothersome venous bleeding. An elastic bandage (e.g., Ace wrap or Esmarch) may be wrapped circumferentially around the extremity, starting distally and moving in a proximal direction. A cuff that is 20% wider than the diameter of the limb is placed around the arm proximal to the wound and inflated to 250 to 300 mm Hg, and the tubing is clamped; the bandage is then removed and the extremity lowered. [74] Some experts recommend a cuff pressure 70 mm Hg higher than the patient's systolic blood pressure. [75] Because tourniquets impair circulation and may produce neurapraxia, their use in the emergency department should be limited to a maximum of 1 hour.Tourniquets on digits have a greater potential for complications. The maximum tourniquet time that is safe for a finger may easily be exceeded inadvertently. Also, finger tourniquets can exert excessive pressures over a small surface area at the base of the finger and injure digital nerves or cause pressure necrosis of digital vessels. For this reason, rubber bands should *not* be used as tourniquets. Tourniquet pressures up to 250 to 300 mm Hg are safe in digits, but pressures of only 150 mm Hg are needed for hemostasis.A 0.5-inch Penrose drain placed around the base of a finger and stretched to no more than two thirds of its circumference provides safe and effective hemostasis. Some physicians wrap the entire finger with a Penrose drain in the fashion of a miniature Esmarch bandage to exsanguinate the digit. The wrap is unraveled from distal to proximal, leaving 2 or more turns around the proximal part of the finger to serve as a tourniquet. This technique generates excessive pressures, ranging from a minimum of 300 mm Hg to >800 mm Hg. [76] A finger can be exsanguinated with a Penrose drain, but a separate drain should be used as a tourniquet (Fig. 36-12) . A few millimeters of difference in total stretch makes a large difference in the pressure applied by this type of tourniquet. [76] Alternatively, a finger can be exsanguinated with a moistened piece of gauze opened to its fullest length, folded in half, and rolled tightly around the elevated finger from tip to base. A Penrose drain is stretched around the base of the finger and secured with a hemostat, and the gauze is removed.A latex rubber surgical glove placed over a patient's hand also can serve as a finger tourniquet. The tip of the glove covering the injured digit is removed, and the latex rubber is then rolled proximally along the patient's finger to form a constricting band at the base (Fig. 36-13) . Another advantage of this technique is that contamination of the wound during closure is less likely. Rolled surgical gloves produce pressures ranging from 113 to 363 mm Hg, depending on the

thickness, the amount of glove finger removed, the number of rolls, and the size of the glove in relation to the size of the patient's hand. [76] Pressure under a Penrose drain ranges between 100 and 650 mm Hg, but it can be more easily controlled. [77] Commercial ring-shaped exsanguinating digit tourniquets are available (Tourni-cot [Mar-Med Company]) (Fig. 36-14) . There is a danger of forgetting to remove such a small tourniquet and of accidentally incorporating it in the dressing.These techniques provide bloodless fields in which to examine, clean, and close extremity wounds. The maximum tourniquet time on a finger should not exceed 20 to 30 minutes. [76] [77] Debridement of questionably devitalized tissue in a wound is best accomplished *without* a tourniquet or pharmacologic vasoconstriction, because bleeding from tissues is often an indication of their viability.

# CLOSURE

The various techniques of wound closure are presented in Chapter 37 . The remainder of this chapter addresses issues related to wound management (e.g., secondary closure, wound dressings, antibiotic use, aftercare instructions, and suture removal).

### Open Vs Closed Wound Management

Wounds that heal spontaneously (i.e., by secondary intention) undergo much more inflammation, fibroplasia, and contraction than those whose edges are reapproximated by wound closure techniques. [5] [78] During wound healing, contraction covers the defect, yet it may have undesirable consequences--notably, deformity (contracture) or loss of function. Left to itself, the healing process may be unable to close a defect completely in areas in which surrounding skin is immobile, such as on the scalp or in the pretibial area.Exposed tendons, bone, nerves, or vessels may desiccate in an open wound. If the patient is careless with an otherwise adequate dressing that covers an open wound, the wound may be further contaminated. [79] The advantages of surgical closure of wounds are apparent: this procedure minimizes inflammation, fibroplasia, contracture, scar width, and contamination.

On the other hand, risks are incurred when wounds are closed. Closure of contaminated wounds increases the probability of wound infection, with impaired healing, dehiscence, and sepsis as possible complications. After cleaning and debridement, wounds left unsutured appear to have a higher resistance to infection than do closed wounds.

Sutures in themselves are detrimental to healing and increase the risk of infection. Each suture inflicts an intradermal incision, damaging surface epithelium, dermis, SQ fat, blood vessels, small nerves, lymphatics, and epithelial appendages such as hair follicles, sweat glands, and ducts. These appendages, once divided and separated by a stitch, usually undergo inflammation and resorption.  Each suture is another piece of foreign material that provokes inflammation. [5] When a suture is removed, bacteria that have settled on the exposed portion of the suture are pulled into the suture track and deposited there. [82] Raised pretibial flap lacerations in elderly patients often necrose when sutured but survive and heal well by secondary intention if taped back into

position. [84]

If the wound is judged to be clean or is rendered clean by scrubbing, irrigation, and debridement, it may be closed. If the wound remains contaminated despite the best of efforts, it must be left open to heal by secondary intention. If the status of the wound is uncertain, the physician must make an educated guess about the risk of infection. Another option available is delayed primary closure.

## Delayed Primary or Secondary Closure

There is a common misconception that all wounds must either be sutured within a few hours or be left open and

relegated to slow healing and an unsightly scar. If there is a substantial risk that closure of a particular wound might result in infection, the decision to close or to leave the wound open can be postponed (Fig. 36-15) (Figure Not Available) . The condition of the wound after 3 to 5 days will then determine the best strategy. Although cleaning and debridement should be accomplished as rapidly as possible, there is no urgency in closing a wound. Edlich and coworkers point out that "the fundamental basis for delayed primary closure is that the healing open wound gradually gains sufficient resistance to infection to permit an uncomplicated closure." [18] Despite its effectiveness, delayed primary closure is a technique that is unappreciated and likely underused by most physicians.

Open wound management is usually an outpatient procedure. The technique consists of the usual careful cleaning and meticulous debridement, followed by packing of the wound with sterile, saline-moistened, fine-mesh gauze. The packed wound is covered by a thick, absorbent, sterile dressing. Depending on the specifics of the wound and the ability of the patient to perform his or her own wound care, the packing may be changed daily at home or in the emergency department, or the wound may be left undisturbed for several days. Sterile saline-soaked packing is standard, and there is no need to impregnate wounds with antiseptics. Prophylactic antibiotics are occassionally prescribed, but their use is neither mandatory nor of proven benefit. On the fourth postoperative day, the wound is reevaluated for closure. If no evidence of infection is present, the wound margins can be approximated (delayed primary closure), or the wound can be excised and then sutured (secondary closure) with minimal risk of infection. Because the wound is closed before the proliferative phase of healing, there is no delay in final healing, and the results are indistinguishable from those of primary healing.

Certain wounds should almost always be managed open or by delayed closure. These include wounds that are already infected and those heavily contaminated by soil, organic matter, or feces. Also included in this category are wounds associated with extensive tissue damage (e.g., high-velocity missile injuries, explosion injuries of the hand, or complex crush injuries) and most bite wounds. Physicians disagree as

**Figure 36-15** (Figure Not Available) Incidence of wound infection over time when delayed closure is performed. Delayed closure is best accomplished on the fourth or fifth day to avoid infection. *(From Edlich*

to which bite wounds may be closed initially. Most would suture cosmetically deforming injuries, including facial bites, and bite wounds that can be completely excised. [35] Others would suture nonextremity dog bites. [85] In severe soft tissue injuries, delayed closure allows time for nonviable tissue to demarcate from uninjured tissue. Debridement can then be accomplished with maximal preservation of tissue.

# PROTECTION

## Dressings

At the conclusion of wound repair, dried blood on the skin surface should be wiped away gently with moistened gauze, and the wound should be covered with a dressing. Depending on the specifics of the wound and the type of repair, a dressing can consist of a simple dry gauze pad or a complex multilayer dressing. Some wounds, such as sutured scalp lacerations, do not routinely require any dressing. Although a variety of specialized (and expensive) dressings are available, there are little hard data to support their use over readily available, properly applied gauze dressings. Therefore, much of the following discussion is theoretical.

### Function of Dressings

Dressings serve a variety of functions. They protect the wound from contamination and trauma, absorb secretions from the wound, immobilize the wound and the surrounding area, exert downward pressure on the wound, and improve the patient's comfort. Occlusive dressings on burns or abrasions prevent painful exposure of the wound to the air and dehydration of the wound surface. [88] Sutured wounds are susceptible to infection from surface contamination during the first 2 days after wound repair. Dressings effectively protect the wound from contamination during this vulnerable period.

One of the primary functions of a gauze dressing is to absorb the serosanguineous drainage that exudes from all wounds. Absorbent dressings also reduce the development of stitch abscesses to some extent. Surface sutures produce small indentations at their points of entrance; tiny blood clots and debris overlie these indentations, allowing bacterial growth at the site. Small "stitch abscesses" can develop; these are initially undetectable but are nevertheless destructive to epithelium. Stitch abscesses rarely infect the entire wound but can slightly increase the width of the scar and produce noticeable, punctate suture marks. [13]

The most common type of dressing is constructed in three layers: a nonadherent contact layer, an absorbent layer, and an outer wrap. [89] Ideally, this dressing provides nonadherence without maceration.

**Contact Layer: Dry, Semiocclusive, and Occlusive Dressings**

Petrolatum gauze (e.g., Adaptic, Xeroform, Betadine, Aquaflo) can be applied next to the wound surface to prevent the wound from sticking to the dry gauze in the absorbent layer and to protect the regenerating epithelium (Table 36-2) . (Nonadherent material should always be used to cover skin grafts.)

Coarse weaves of gauze, usually available in the form of multilayered pads, absorb blood and exudate, but the dressing

| **TABLE 36-2** -- Advantages of Occlusive Dressings<br>*Eaglstein WH: Effect of occlusive dressings on wound healing. Clin Dermatol 2:107, 1984.* |
|---|
| 1. More rapid healing |
| 2. Less pain from air exposure |
| 3. Better cosmetic results |
| 4. Fewer dressing changes |
| 5. Better protection from bacteria |

will adhere if the interstices of the fabric are relatively large. Capillaries, fibrin, and granulation tissue will penetrate and become enmeshed in the material. If the proteinaceous exudate from the wound dries by evaporation, the scab usually clings to the dressing. [89] [90] Some clinicians use this effect to "debride" the wound when the gauze is removed. However, it also destroys healing tissue, particularly the new epithelium. Debridement of the wound with wet-to-dry dressings is quick, but debridement with surgical instruments is more controlled and less traumatic.

Adherence to the wound can be avoided if the dressing is nonabsorbent, occlusive, or finely woven. Some physicians use fine mesh gauze (41 to 47 warp threads per square inch) rather than petrolatum gauze on abrasions, especially on those wounds that are

heavily contaminated, because removal of this type of dressing debrides only the small tufts of granulation tissue that become fixed in the mesh pores, leaving a clean, even surface. Once a healthy, granulating surface is present and re-epithelialization is proceeding, nonporous dressings can be used. [90] Fine mesh gauze also is used next to exposed tissue in wounds being considered for delayed primary closure; a protective and absorptive bulky dressing is placed on top of the wound.

Wounds covered with permeable dressings such as plain gauze tend to dry out. Drying of the wound surface damages a shallow layer of exposed dermis, which impedes epidermal resurfacing of abrasions, burns, and incisions. [87] Wound desiccation results in further epidermal necrosis, crust formation, and increased inflammation. [90] [91] If the wound is kept moist by covering it with an occlusive film soon after wound management and if the film is left in place for at least 48 hours, the epidermis will migrate over the surface of the dermis up to 100% faster than when a dry scab is allowed to form. [92] [93] [94] In one study, the occluded half of a surgical incision produced a more linear, less pigmented scar. [95] Protection of wounds that are healing by secondary intention with occlusive or semiocclusive dressings has several advantages, [6] including more rapid healing, less pain from air exposure, better cosmetic result, few dressing changes, and protection from bacteria.

This occlusive effect is achieved with various polyurethane-derived membranes, such as Epilock (Derma-Lock Medical Corporation), Op-Site (Smith and Nephew, Ltd.), Tegaderm (3M), Bioclusive (Johnson & Johnson), and Primaderm (ACCO, Inc.); those with soluble collagen or gelatin backing, such as DuoDerm (Convatec) and Biobrane (Woodroof Laboratories); and products with hydrogels, such as Vigilon.

One of the fears of using occlusive dressings is that microorganisms will proliferate in the moist environment beneath the occlusive film and increase wound infection rates. Occlusive dressings such as DuoDerm actually serve more as a barrier to external pathogenic bacteria [97] ; although *surface* bacteria under occlusive dressings can multiply, [98] chronic wounds, usually contaminated with large numbers of bacteria, are routinely treated with occlusive dressings successfully. [99]

A paint-on collodion dressing over a wound closed with a buried subcuticular stitch provides considerably greater resistance to infection than wounds closed by the same technique but with no dressing. The use of collodion obviates the need for a gauze dressing, frequent dressing changes, and uncomfortable dressings in areas such as the groin, the axilla, and the neck. [100] However, the collodion does not allow drainage of the wound and so is rarely used.

Another concern of physicians is that occlusive dressings will macerate underlying skin. Optimal wound appearance under a dressing is a moist red surface with capillary and epithelial growth. Collagen sponge dressings provide this appearance (if they are not accidentally dislodged), whereas both DuoDerm and Op-Site adhere to the wound site, macerate it, and produce a thick eschar that may be difficult to remove. However, underneath the eschar the surface is epithelialized. [101] Wounds covered with certain occlusive dressings or with silver sulfadiazine (Silvadene [Marion Laboratories]) applications appear to be blanketed with pus; this exudate actually represents the

beneficial proliferation of macrophages and polymorphonuclear leukocytes. [94] [102]

Adhesive-backed dressings (e.g., DuoDerm and Op-Site) have a tendency to adhere to and remove new epidermis, and they do not allow exudate to drain out the edges of the dressing. Between dressing changes, the wound should be coated with petrolatum or an antibiotic ointment before these products are applied. [6] Epilock has the advantage of thermally insulating the wound by virtue of its thickness, but unlike Tegaderm and Op-Site, it is opaque and does not allow inspection of the underlying wound surface. [102] Because Epilock allows drainage of exudate, it is better tolerated by patients if the overlying gauze bandage is changed daily.

Other nonadherent-type dressings include Adaptic, Xeroform, Betadine, Dermicel, and the nonabsorbent Telfa. Petrolatum gauze does not appear to enhance epidermal healing. [103]

### Absorbent Layer

In dressing wounds with considerable drainage, sufficient gauze should be used to cover the wound and to absorb all of the drainage. Dressings on such wounds can be changed daily, which is frequent enough to avoid bacterial overgrowth beneath the dressing. [5] [90] Once a dressing becomes moist, pathogens can pass through the dressing to the underlying wound. [87] Any dressing should be changed whenever it becomes soiled, wet, or saturated with drainage. Fluid accumulating under an occlusive dressing should be aspirated or the dressing changed every 1 to 2 days during the first week or until the exudate no longer accumulates. [104] A dressing that is used to absorb exudate or debride the wound must be changed more frequently than one designed solely to occlude.

### Outer Layer

Dressings and bandages can serve as surface splints (as can surgical tape) by reducing mechanical stresses on the wound during the early phases of healing. Even when subcuticular stitches have been placed, these "external splints" are useful

in relieving tension across the wound. They are most needed between the 7th and 42nd days, the time of collagen synthesis and remodeling. [13]

Compressive dressings may be helpful in preventing hematoma formation and eliminating dead space within a wound. They are particularly useful in wounds that have been undermined extensively and in facial wounds, in which SQ capillary bleeding and swelling can exert tension on fine skin sutures and jeopardize skin closure (Fig. 36-16) . Pressure dressings should be used to immobilize skin grafts. Surgical tape can serve as a pressure dressing in areas such as fingertips on which bandages cannot be easily applied. However, a pressure dressing should not be used as a substitute for good hemostasis. [5]

Pressure dressings should be applied to all ear lacerations to prevent hematoma

formation and subsequent deformation and destruction of cartilage. The ear should be enveloped in the dressing so that pressure from the outer bandage is distributed evenly across the irregular surface of the pinna. Moistened cotton is packed into the concavities of the pinna until the cotton is level with the most lateral aspect of the helical rim. Square pieces of gauze cut to fit the curvature of the ear are placed *behind* (medial to) the pinna. Several more gauze squares are placed on the lateral surface of the ear; the packing is then secured in place with a circumferential head bandage. The bandage must not encompass the opposite ear, because it would just as easily cause pressure necrosis of that ear if left unprotected. Application of a pressure dressing for the ear is discussed further in Chapter 68

Traumatic wounds are bandaged to compress or immobilize the wound or to secure and protect the underlying dressing. Most bandaging is performed on extremities, on which dressings are difficult to secure with tape alone. Rolls of cotton (Kerlix; Kling stretch gauze) are well suited for this purpose. The bandage is wound around the extremity, advancing proximally with circular, overlapping turns. Care should be taken to avoid allowing wrinkles in the bandage, which will later create pressure points, or making loose turns that shorten the effective life of the dressing. When joint surfaces are crossed, the cotton is anchored distally with several turns, unrolled obliquely across the joint several times in a figure-of-eight pattern, and anchored proximally by two complete turns. This process is repeated until the bandage is securely in place. The ends of the bandage are fastened to the skin by strips of adhesive tape.

Bandages over the forearm and the lower extremities are particularly prone to slippage because of the constant motion of these parts and because of the marked changes in extremity diameter over a short distance. The roll of bandage can be rotated 180° after each circular turn, producing a reverse spiral and reducing the bandage's mobility (Fig. 36-17) (Figure Not Available) . A "tube" of elastic cotton netting (e.g., Surgifix, Tubex, Surgitube, HygiNet) pulled over the bandage or unrolled from a metal applicator frame effectively stabilizes the entire dressing in these areas (Fig. 36-18) (Figure Not Available) . Another useful technique consists of placing strips of tape on opposite sides of the extremity, leaving the ends free. The bandage is wrapped around the dressing, covering the portions of the tape that are attached to the skin. The free ends of tape are then incorporated in the bandage (Fig. 36-19) (Figure Not Available) . [105]

Certain chemically treated wide-mesh weaves have the properties of cling and stretch, holding snugly in place but expanding if edema develops. [89] An elastic cotton roll (Kerlix) allows the bandage to conform to body contours, provides some mobility to bandaged joints, and permits the wound to swell without the circumferential bandage constricting the extremity. The inelastic Kling bandage better immobilizes the part. Rigid immobilization with plaster splints or braces is needed to protect wounds in mobile areas, such as around large joints.

Most scalp wounds do well when left uncovered. If a dressing is necessary, it must be held in place by a bandage. There are many techniques for bandaging heads. Stavrakis described the following method:

The assistant tightly holds a strip of bandage three inches wide and three feet long over

the patient's head in a frontal plane [Fig. 36-20 (Figure Not Available) *A*]. While one person maintains tension on the first strip of bandage, the second person starts bandaging the head with the main bandage at the forehead level in a horizontal plane, using a full-length gauze bandage [see Fig. 36-20 (Figure Not Available) *B*]. (The 'Kling'-type bandage is preferred.) After several turns are made to stabilize the main bandage, it is passed near the patient's ear, then wrapped around the short strip of bandage in a full turn [see Fig. 36-20 (Figure Not Available) *C*]. The main bandage is then taken across the front of the head, wrapped full-turn around the other side of the short bandage, then brought around the back of the head and wrapped around the first side again. This maneuver is repeated, alternating front and back until the head is covered by overlapping passes of bandage [see Fig. 36-20 (Figure Not Available) *D*]....Several turns of the bandage across the forehead in a horizontal plane stabilize the dressing. The dressing is...secured by tying the ends of the short strip under the chin [see Fig. 36-20 (Figure Not Available) *E*]. Removal of this dressing can be accomplished easily by untying the chin straps and gently pulling both ends upward. [106]

Methods of bandaging wounds in other locations of the body are described in detail in other texts. [68] [86]

Dressings vary in their absorbency, adhesiveness, occlusiveness, opacity, and insulating properties. Further research may identify types of dressings best suited for different phases of the healing wound. Currently, a two- or three-layer dressing is used for most traumatic wounds; the choice of material for the contact layer is determined by the characteristics of the individual wound. [107]

**Splinting and Elevation**

Although splints are readily applied to orthopedic or soft tissue injuries, immobolization of wounds and sutured lacerations is often neglected, despite the fact that these techniques

**Figure 36-20** (Figure Not Available) *A- E,* Technique for bandaging the head. *(From Stavrakis P: A better head dressing. Res Staff Physician 26:88, 1980. Reproduced by permission.)*

may enhance healing and provide patient comfort. Immobilization of an injured extremity promotes healing by protecting the closure and by limiting the spread of contamination and infection along lymphatic channels. Wounds overlying joints are subjected to repeated stretching and movement, which delays healing, widens the scar, and could possibly disrupt the sutures. [18] Splints are almost always required for lacerations that overlie joints and are frequently necessary for protection of wounds involving fingers, hands, wrists, the volar aspects of forearms, the extensor surfaces of elbows, the posterior aspects of legs, the plantar surfaces of feet, and the extremities when skin grafts have been applied. Splinting is often underused by the emergency physician in the treatment of lacerations.

A plaster or aluminum splint may be incorporated into a bandage to reduce the mobility of the part. Splinting techniques for extremities are explained more fully in Chapters 51 ,

Elevation of injured extremities is important in all but trivial injuries. Elevation limits edema formation, an expected sequela of trauma and inflammation, and allows more rapid healing. [18] Elevation also reduces throbbing pain. Patients given this information are often more motivated to elevate the extremity as instructed. Slings can be used to elevate wounds involving the forearm or the hand.

## Ointments

The safety and efficacy of topical antibiotic preparations used on wound surfaces are still debated. Some investigators warn of skin sensitization by preparations containing neomycin [83] and others, of the emergence of resistant strains of bacteria with any topical antibiotic. [108] Other studies have shown that use of a triple-antibiotic preparation containing neomycin, bacitracin, and polymyxin provides a broad spectrum of protection against infection in *abrasions* without systemic absorption and toxicity or the emergence of resistant strains of bacteria. Unless this topical antibiotic ointment is used repeatedly or on inflamed skin, there is a relatively low risk of allergic sensitization. [109] There is evidence that the active agents in Neosporin ointment and Silvadene cream, as well as their inert bases and vehicles, improve wound healing. In a prospective, randomized, double-blind study, Dire and colleagues found that bacitracin and Neosporin ointments reduced the infection rate over that seen with plain petrolatum ointment. [112]

Ointments can be used to reduce the formation of a crust that covers and separates the edges of the wound. Lacerations surrounded by abraded skin are especially predisposed to coagulum formation. In such cases the patient can be instructed to cleanse the wound frequently and to follow the cleansing with an application of ointment during the first few days. [18] Ointments also prevent the dressing from adhering to the wound. [3] Some researchers recommend using bacitracin applied in a thin coating, not for protection against infection, but for prevention of these mechanical problems.

Note that the stronger topical corticosteroids have detrimental effects on healing. Application of 0.1% triamcinolone acetonide in an ointment retards healing in wounds by as much as 60%, whereas hydrocortisone probably does not interfere with epithelialization. [93] [103] Some physicians believe that single and low doses of oral corticosteroids probably have no effect on wound healing but that repeated, large doses of steroids (40 mg of prednisone per day) inhibit healing, particularly if used before the injury or during the first 3 days of the healing phase. There is some evidence that topical vitamin A may reverse some of the anti-inflammatory and immunosuppressive effects of corticosteroids. [115] The exact value of ointments in the treatment of lacerations has yet to be determined. However, their routine use after wound cleaning does encourage patient inspection of the wound.

## Systemic Antibiotics

Most traumatic soft tissue injuries sustain a low level of bacterial contamination. [79] The standard wound infection rate in unselected emergency department wounds is 2 to 5%. In a number of clinical studies of traumatic wounds, prophylactic antibiotics administered

orally and intramuscularly in a variety of regimens did not reduce the incidence of infection.

In experimental models of contaminated incisions, antibiotics have no therapeutic value >3 hours after the injury. [121] [122] When the wound is contaminated with greater than $10_9$ bacteria per gram of tissue (such as wounds in contact with pus or feces,) infection will develop despite antibiotic treatment. [35] Most clinical investigations of antibiotic use in emergency patients have omitted heavily contaminated wounds in their series. Studies of antibiotic prophylaxis for animal bite wounds have produced variable results, and no large study providing stratification of the many prognostic factors has been done. [123]

Indications for antibiotics vary among physicians, and

because of limited scientific data, there is no clear practice standard. In most soft tissue wounds where the level of bacterial contamination after cleaning and debridement is low, antibiotics have not been proven beneficial. Antibiotics may have marginal benefit when the level of contamination is overwhelming or if the amount of questionably viable tissue left in the wound is considerable (e.g., with crush wounds). Antibiotics should be considered for extremity bite wounds, puncture-type bite wounds in any location, intraoral lacerations that are sutured, orocutaneous lip wounds, wounds that cannot be cleaned or debrided satisfactorily, and highly contaminated wounds (e.g., those contaminated with soil, organic matter, purulence, feces, saliva, or vaginal secretions). They also should be considered for wounds involving tendons, bones, or joints; for wounds requiring extensive debridement in the operating room; for wounds in lymphedematous tissue; for distal extremity wounds when treatment is delayed for 12 to 24 hours; for patients with orthopedic prostheses; and for patients at risk of developing infective endocarditis. [18] [117] If systemic antibiotics are considered necessary, they should be given intravenously or intramuscularly in the earliest stages of wound management.

The choice of antibiotic, particularly for bite wound prophylaxis, is as controversial as the indications for usage. Many species of bacteria cause bite wound infections, making complete coverage impossible. [28] [124] [125] Some of the antibiotic regimens that have been recommended for bite wounds include dicloxacillin or cephalexin for high-risk dog bite wounds, dicloxacillin or cephalexin plus penicillin for human or cat bite wounds, and amoxicillin-clavulanic acid or cefuroxime for any domestic animal bite. Irrigation with topical antibiotics may be of benefit in these situations. The duration of antibiotic prophylaxis also is in question. It is common practice to provide antibiotics for 72 hours, although data from surgical studies indicate that antibiotics administered beyond the first postoperative day provide no additional protection. [126] Short courses of antibiotics do not seem to increase the incidence of resistant strains of organisms. [127] In all cases, the use of antibiotics should remain subordinate to careful cleaning and debridement. If the infection risk is high enough to warrant antibiotics, secondary closure should be considered.

It is often difficult for physicians to accept that there are *no data to support the routine use of prophylactic antibiotics for the majority of wounds encountered in the emergency department*. [128] Although the theoretical arguments for antibiotic use have been

outlined above, hard data demonstrating benefit are generally lacking. Antibiotics should not be used as a substitute for proper wound preparation or a measure to overcome factors suggesting delayed wound closure. The downsides of antibiotic use include needless expense; potential side effects (e.g., rash, anaphylaxis, diarrhea, vomiting); and the development of resistant bacteria, both in the wound and in general. If antibiotics are used, they should be given as soon as possible after wounding and continued for only 2 to 3 days in the absence of a developing infection.

## Immunoprophylaxis

Although tetanus is rare, it still occurs in the United States (about 50 cases per year) and is a preventable disease. Therefore, any wound should be assessed for its potential to cause tetanus, and prophylaxis should be considered in the emergency department. Gergen and colleagues demonstrated that about 70% of Americans >6 years of age had protective levels of tetanus antibodies. [130] Levels declined as age increased, and elderly women had the lowest levels of protection. Hispanics (and likely other immigrants) were most likely to have inadequate immunity. Hence, efforts at preventing tetanus should be especially addressed in immigrants and the elderly.

Recommendations for tetanus prophylaxis have evolved since the 1980s. The guidelines published by the Public Health Services Advisory Committee on Immunization Practices, Centers for Disease Control and Prevention, differ slightly from those of the American College of Surgeons in the use of tetanus immune globulin.

Many cases of tetanus develop despite prior immunization; tetanus frequently results from chronic skin lesions and apparently minor or clean wounds. [132] In 10 to 20% of cases, a precedent wound cannot be identified. Patients' recall of past immunizations is imperfect, and immunity may rarely be inadequate after a complete series of tetanus toxoid. [133] Furthermore, there is no precise consensus on the definition of a "tetanus-prone wound," yet treatment decisions are based on the differentiation between clean and contaminated wounds. Some investigators warn of overtreatment, and others maintain that the risk of therapy is minimal compared with the danger of tetanus.  After comparing those risks and benefits, most clinicians would agree that a certain amount of overtreatment is acceptable.

Tetanus-prone wounds include injuries >6 hours old; wounds contaminated by feces, saliva, purulent exudate, or soil; wounds with retained foreign bodies or containing devitalized or avascular tissue; established wound infections; penetrating abdominal wounds involving bowel; deep puncture wounds; and wounds caused by crush, burns, or frostbite (Fig. 36-21) . When patients are questioned about their tetanus immunization status, they should be asked if they completed the primary immunization series, and if not, how many doses have been given.

Patients who have not completed a full primary series of injections may require both tetanus toxoid and passive immunization with tetanus immune globulin. The preferred preparation for active tetanus immunization in patients 7 years of age and older is 0.5 mL of tetanus toxoid (plus the lower, adult dose of diphtheria toxoid); the dose of tetanus immune globulin is 250 to 500 units given intramuscularly. [130] Mild local

reactions consisting of erythema and induration are common after tetanus toxoid injections. Some patients with high antibody levels develop a hypersensitivity reaction of tenderness, erythema, and swelling, or serum sickness. Generalized urticarial reactions and peripheral neuropathy have also been reported. A significant percentage of elderly patients fail to develop protective antitoxin antibody titers after 14 days when given tetanus toxoid boosters.

Because protective levels of tetanus antibodies tend to parallel levels of antibodies to diphtheria, it has been recommended that both immunizations be given simultaneously. Both tetanus and diphtheria immunization have been implicated as a cause of adverse reactions. Tetanus and diphtheria toxoid are products of human antisera, and serious reactions are rare. [138] The most common reaction is a painful, indurated, tender eruption at the injection site, occasionally accompanied by a fever and mild systemic symptoms. This is a hypersensitivity reaction (arthus-type reaction), not an infection or immunoglobulin E (IgE)-mediated allergy. As such, this reaction does not require drainage or antibiotics, nor does it represent an absolute contraindication to further immunizations. Local reactions are more common in patients who have been given multiple immunizations, so it is unwise to give excessive immunizations "just to be safe." In tetanus-prone injuries, "hyperreactors" can be given tetanus immune globulin. A minor febrile illness, such as an upper respiratory infection, is not a reason to delay immunization. The only absolute contraindication to tetanus toxoid is a history of anaphylaxis or a neurologic event. In such cases, tetanus immune globulin can be safely given. Pregnancy is not a contraindication to either toxoid or immune globulin, although some suggest that the toxoid be used with caution during the first trimester. Given the excellent amnestic response to the toxoid, it is likely that the primary immunization series, coupled with intermittent boosters, conveys immunity for most of one's life.

When a wound results from the bite or scratch of a wild or domestic animal, prophylaxis against rabies also must be considered (Tables 36-3 and 36-4) . Further discussion of the prevention of rabies is provided elsewhere. [139]

## PATIENT INSTRUCTIONS

Successful wound healing is partly dependent on the care given to the wound once the patient leaves the emergency center. Patient satisfaction depends not only on the cosmetic result, but also on the expectation of that result. [6] Therefore, the patient should receive thorough and understandable instructions.

The patient should be informed that no matter how skillful the repair, any wound of significance produces a scar. Most scars deepen in color and become more prominent before they mature and fade. The final appearance of the scar cannot be judged before 6 to 12 months after the repair. [83]

Patients may experience dysesthesias in or around a scar, particularly about the midface. Gentle rubbing or pressing on the skin may relieve the symptoms. If wounds extending to SQ levels lacerate cutaneous nerves, patients may be bothered by hypoesthesia distal to the wound. Dysesthesia and anesthesia usually resolve in 6

months to 1 year. [6]

Because the wound edges are rapidly sealed by coagulum and bridged by epithelial cells within 48 hours, the wound is essentially impermeable to bacteria after 2 days. [13] [140] The patient should be instructed to keep the wound protected by keeping the dressing clean and dry for 24 to 48 hours. In this initial period the dressing should be changed only if it becomes externally soiled or soaked by exudate from the wound. If possible, the injured part should be kept elevated. There is a tendency on the part of most patients to avoid getting sutures wet. *There is no proven harm in exposing sutured wounds to soap and tap water for short periods*, and many physicians routinely allow patients to bathe with sutures in place. Some advise patients to wash wounds daily to remove dried blood and exudate, especially on areas such as the face or the scalp.

After 48 hours, the patient may remove the dressing in uncomplicated wounds and check for evidence of infection: redness, warmth, increasing pain, swelling, purulent drainage, or the "red streaks" of lymphangitis. Not all patients are able to identify these signs of infection; it is prudent to have patients with complicated or infection-prone wounds examined in 2 days by a physician or nurse. [141] Interestingly, patients may be more likely to fail to recognize a bona fide infection than to overdiagnose an infection when it is absent. [140] If there is no sign of infection after 48 hours, the patient can care for the wound until it is time for removal of the sutures. A daily gentle washing with mild soap and water to remove dried blood and exudate is beneficial. [139] [141] Undiluted hydrogen peroxide may destroy granulation tissue and newly formed epithelium, and it should not be used as

| **TABLE 36-3** -- Rabies Postexposure Prophylaxis Guide--July 1984 [139] *From Leads from the Mortality and Morbidity Weekly Report. JAMA 252:883-887, 1984.* | | |
|---|---|---|
| **The following recommendations are only a guide. In applying them, take into account the animal species involved, the circumstances of the bite or other exposure, the vaccination status of the animal, and presence of rabies in the region. Local or state public health officials should be consulted if questions arise about the need for rabies prophylaxis.** | | |
| **Animal Species** | **Condition of Animal at Time of Attack** | **Treatment of Exposed Person A** |
| *Domestic* | | |

| Dog and cat | Healthy and available for 10 days of observation | None, unless animal develops rabies [D] |
|---|---|---|
| | Rabid or suspected rabid | HRIG [I] and HDCV |
| | Unknown (escaped) | Consult public health officials. If treatment is indicated, give HRIG [I] and HDCV |
| *Wild* | | |
| Skunk, bat, fox, coyote, raccoon, bobcat, and other carnivores | Regard as rabid unless proven negative by laboratory tests [T] | HRIG [I] and HDCV |
| *Other* | | |
| Livestock, rodents, and lagomorphs (rabbits and hares) | Consider individually. Local and state public health officials should be consulted on questions about the need for rabies prophylaxis. Bites of squirrels, hamsters, guinea pigs, gerbils, chipmunks, rats, mice, other rodents, rabbits, and hares almost never call for antirabies prophylaxis. | |

*All bites and wounds should immediately be thoroughly cleansed with soap and water.* If antirabies treatment is indicated, both human rabies immune globulin (HRIG) and human diploid cell rabies vaccine (HDCV) should be given as soon as possible, *regardless* of the interval from exposure. Local reactions to vaccines are common and do not contraindicate continuing treatment. Discontinue vaccine if fluorescent antibody tests of the animal are negative.

During the usual holding period of 10 days, begin treatment with HRIG and HDCV at first sign of rabies in a dog or cat that has bitten someone. The symptomatic animal should be killed immediately and tested.

If HRIG is not available, use antirabies serum, equine (ARS). Do not use more than the recommended dosage.

The animal should be killed and tested as soon as possible. Holding for observation is not recommended.

a cleaning agent on the scar itself. [42] Generally, a wound should be protected with a dressing during the first week, and the dressing should be changed daily. If the wound is unlikely to be contaminated or traumatized, it may be left uncovered. Although it is

generally recommended that uncovered scalp wounds can be washed after 1 to 2 days, a gentle rinse with water in the first few hours after wound closure is unlikely to be harmful. Vigorous scrubbing of wounds should be discouraged.

Patients should be informed that sutures themselves do not cause pain. A painful wound is often a sign of infection or suture reaction, and pain should prompt a wound check.

| TABLE 36-4 -- Rabies Postexposure Prophylaxis Schedule, United States *From the Recommendations of the Immunization Practices Advisory Committee, MMWR 40(RR-3):1, 1991.* | | |
|---|---|---|
| **Vaccination Status** | **Treatment** | **Regimen** [*] |
| Not previously vaccinated | Local wound cleansing | All postexposure treatment should begin wih immediate thorough cleansing of all wounds with soap and water |
| | HRIG | 20 IU/kg of body weight; if anatomically feasible, up to half the dose should be infiltrated around wounds and rest administered IM in gluteal area; HRIG should not be administered in same syringe or into same anatomic site as vaccine; because HRIG may partially suppress active production of antibody, no more than recommended dose should be given |
| | Vaccine | HDCV or RVA, 1 mL, IM (deltoid area ), one each on days 0, 3, 7, 14, and 28 |

| Previously vaccinated | Local wound cleansing | All postexposure treatment should begin with immediate thorough cleansing of all wounds with soap and water |
| --- | --- | --- |
| | HRIG | HRIG should not be administered |
| | Vaccine | HDCV or RVA, 1 mL, IM (deltoid area ), one each on days 0 and 3 |

*These regimens are applicable for all age groups, including children.
The deltoid area is the only acceptable site of vaccination for adults and older childen. For younger children, the outer aspect of the thigh may be used. Vaccine should never be administered in the gluteal area.
Any person with a history of preexposure vaccination with HDCV or RVA, prior postexposure prophylaxis with HDCV or RVA, or previous vaccination with any other type of rabies vaccine and a documented history of antibody response to the prior vaccination. HDCV, human diploid cell rabies vaccine; HRIG, human rabies immune globulin; RVA, rabies vaccine, adsorbed.

Some wounds heal with wide, unattractive scars despite the physician's best efforts. These include wounds that cross perpendicular to joints, wrinkle lines, or lines of minimum tension (Kraissel's lines); wounds that retract >5 mm; and wounds that are over convexities or in certain anatomic locations (e.g., back, shoulders) where hypertrophic scars are common. A wound crossing a concave surface may result in a bowstring deformity; one crossing a convexity may leave a scar depression. To avoid these complications, a Z-plasty procedure can be done at the time of initial wound management, or the scar can be revised later. The patient should be told to expect suboptimal outcomes in these situations. [30]

If an injured extremity or finger is protected by a splint, it should be left undisturbed until the sutures are removed. Patients with intraoral lacerations should be instructed to use warm salt water mouth rinses at least three times a day.

Patients may ask about the efficacy of various creams and lotions (e.g., vitamin E, cocoa butter) in limiting scar formation. At this time there are no data to evaluate the use of these substances. Patients should be told to avoid aspirin, as it has been shown to decrease the development of tensile strength and increase the likelihood of hematoma formation.

## SECONDARY WOUND CARE

### Reexamination

Patients with simple sutured wounds may be released with appropriate instructions for home care and told to return for suture removal at an appropriate time. Selected wounds should be examined in 2 to 3 days for signs of infection. All wounds should be inspected if the patient experiences increasing discomfort or develops a fever. [143] Bite wounds and other infection-prone wounds should be inspected in 2 days. Wounds being considered for delayed primary closure are evaluated in 4 to 5 days. [78]

Wounds in which extensive dissection of SQ tissue has been performed may develop a low-grade, localized cellulitis. It is rarely necessary to open these wounds. The removal of 1 or 2 stitches may relieve some of the tension caused by mild swelling. With daily cleansing using water and a mild soap and with application of warm compresses, this type of wound reaction should subside within 24 to 48 hours. [143]

*A wound that has become infected should be evaluated for the presence of a retained foreign body.* Also, in most sutured wounds that become infected, the sutures must be removed to allow drainage. If a wound exhibits a minor infection, a few sutures, or all of them, may be removed, but grossly infected wounds should be packed open to allow for further drainage. The presence of sutures in a contaminated wound considerably limits the activity of various antibiotics. [144] Infection around a suture can lead to the formation of a stitch mark. [64] Infected wounds should be treated with daily cleansing, warm compresses, and antibiotics. Wounds that have been opened should be left to heal by secondary intention, which involves wound contraction, granulation tissue formation, and epithelialization.

**Suture Removal**

Because wounds do not heal at a standard rate, no strict guidelines can be set for time of suture removal. The optimal time for suture removal varies with the location of the wound, the rate of wound healing, and the amount of tension on the wound. Certain areas of the body such as the back of the hand heal slowly, whereas facial or scalp wounds heal rapidly. Speed of wound healing is affected by systemic factors such as malnutrition, neoplasia, or immunosuppression. At the time that suture removal is being considered, 1 or 2 sutures may be cut to determine whether the skin edges are sufficiently adherent to allow removal of all the sutures. [3] Removing sutures too early invites wound dehiscence and widening of the scar, whereas leaving sutures in longer than necessary may result in epithelial tracts, infection, and unsightly scarring. [145]

Small stitch abscesses are common in wounds in which sutures have been left in place for longer than 7 to 10 days. Localized stitch abscesses generally resolve following removal of the sutures and application of warm compresses. There is usually no need for antibiotic therapy with localized stitch abscesses.

Percutaneous sutures stimulate an inflammatory reaction along the suture track. Factors that determine the severity of stitch marks include the length of time skin stitches are left in place, skin tension, the relationship of the suture to the wound edge, the region of the body, infection, and tendency for keloid formation. [64] [146] The skin of the eyelids, palms, and soles and the mucous membranes seldom show stitch marks. In contrast, oily skin

and the skin of the back, the sternal area, the upper arms, the lower extremities, the dorsum of the nose, and the forehead are likely to develop the permanent imprints of suture material on the skin surface. [64]

If sutures are removed within 7 days, generally no discernible needle puncture or stitch mark will persist. [146] However, at 6 days, the wound is held together by a small amount of fibrin and cells and has minimal strength (see Fig. 36-1) . [82] The tensile strength of most wounds at this time is adequate to hold the wound edges together, but only if there are no appreciable dynamic or static skin forces pulling the wound apart. [3] Minimal trauma to an unsupported wound at this point could cause dehiscence. The physician should decide on the proper time of suture removal after weighing these various factors. If early suture removal is necessary, wound repair can be maintained with strips of surgical skin tape. The key to wound tensile strength after suture removal is an adequate deep tissue layer closure.

There are some general guidelines for suture removal. Sutures on the face should be removed on the fifth day following the injury, or alternate sutures should be removed on the third day and the remainder on the fifth day. On the extremities and the anterior aspect of the trunk, sutures should be left in place for approximately 7 days to prevent wound disruption. Sutures on the scalp, back, feet, and hands and over the joints must remain in place for 10 to 14 days, even though permanent stitch marks may result. [64] Some physicians recommend the removal of sutures in eyelid lacerations in 48 to 72 hours to avoid epithelialization along the suture tract, with subsequent cyst formation. [147]

Removing sutures is relatively simple. The wound should be cleansed, and any remaining crust overlying the wound surface or surrounding the sutures should be removed. The skin is wiped with an alcohol swab. Each stitch is cut with a scissors or the tip of a scalpel blade (No. 11 Bard-Parker) at a point close to the skin surface on one side.

**Figure 36-22** (Figure Not Available) Technique for suture removal. Pull should be toward the wound line *(A)* rather than away from it *(B),* which causes the wound to tear apart. *(Modified from Stuzin J, Engrav LH, Buehler PK: Emergency treatment of facial lacerations. Postgrad Med 71(3):81, 1982.)*

The suture is grasped on the opposite side with forceps and is pulled across the wound (Fig. 36-22) (Figure Not Available) . The amount of exposed suture that is dragged through the suture tract is thereby minimized. It is difficult to remove sutures with very short ends. At the time of suture placement, the length of the suture ends should generally equal the distance between sutures to permit easy grasping of the suture during subsequent removal yet avoiding entanglement during the knotting of adjacent sutures.

Once the skin sutures are removed, the width of the scar increases gradually over the next 3 to 5 weeks unless it is supported. Support is provided by previously placed SQ stitches that brought the skin edges into apposition, by a previously placed subcuticular stitch, or by the application of skin tape (Fig. 36-23) . A nonabsorbable subcuticular suture can be left in place for 2 to 3 weeks to provide continued support for the wound. Although complications such as closed epithelial sinuses, cysts, or internal tracts can

occur from prolonged use of this stitch, they are unusual and can be avoided by the placement of a buried subcuticular stitch using an absorbable suture. [13]

If a subcuticular stitch with reliefs has been used, the suture is cut at the midpoint of the relief. Half of the suture is removed at the original point of entry into the skin and the other half through the original exit point (Fig. 36-24) (Figure Not Available) . [148] If a nonabsorbable subcuticular suture cannot be removed or a portion of it ruptures during removal, the protruding end should be grasped with a hemostat, pulled taut, and cut with scissors as close to the skin as possible so that the end of the suture retracts under the skin.

If time and effort have been invested in a cosmetic closure of the face, the repair should be protected with skin tape after the skin sutures have been removed. Wound contraction and scar widening continue for 42 days after the injury. [82] Because the desired result is a scar of minimal width, the tape should be used for 5 weeks following suture removal. With exposure to sunlight, scars in their first 4 months redden to a greater extent than surrounding skin. In exposed cosmetic areas and when prolonged exposure to the sun is anticipated, this should be prevented with the use of a sunscreen containing para-amino benzoic acid (PABA).

## COMPLICATIONS

Infection is probably the most common cause of dehiscence. If the patient is careless or unlucky, reinjury can reopen a wound despite the protection of a thick dressing. If the suture size is too small, the stitch may break. A stitch that is too fine or tied too tightly may cut through friable tissue and pull out. Knots that have not been tied carefully may unravel. The suture material may be extruded or absorbed too rapidly. Finally, if a stitch is removed too early (i.e., before tissues regain adequate tensile strength), the wound

**Figure 36-24** (Figure Not Available) At the time of suture removal, the suture is cut at the midpoint of the relief *(A).* The proximal portion is removed at the point of original entry into the skin *(B),* and the distal portion is removed through the original exit point *(C).* (From Grimes DW, Garner RW: "Reliefs" in intracuticular sutures. Surg Rounds 1:48, 1978. Reproduced by permission.)

loses needed support and falls open. If the wound edges show signs of separating at the time of suture removal, alternate stitches can be left in place and the entire length of the wound supported by strips of adhesive tape.

There are several reasons why wounds fail to heal; some are related to decisions made at the time of wound closure, and others are consequences of later events. Some of the impediments to healing include ischemia or necrosis of tissue, hematoma formation, prolonged inflammation caused by foreign material, excessive tension on skin edges, and immunocompromising systemic factors. In attempting to repair wounds, physicians sometimes inadvertently retard the healing process (e.g., with premature closure of contaminated wounds). With the development of new methods and solutions for cleansing wounds and the discovery of the optimal concentrations of solutions currently in use, tissue-toxic antiseptic solutions can be abandoned. Better suture materials are replacing the reactive sutures that often served as foreign bodies rather than tissue

supports. Improved materials used for dressing wounds enhance wound healing.

One of the primary causes of delayed healing is wound infection. Wound cleaning and debridement, atraumatic and aseptic handling of tissues, and the use of protective dressings minimize this complication. Inversion of the edges of a wound during closure produces a more noticeable scar, whereas skillful technique can convert a jagged, contaminated wound into a fine, inapparent scar. However, the patient's actions also affect wound healing. Delay in seeking treatment for an injury may significantly affect the ultimate outcome of the wound. Furthermore, in the first few days following an injury, the patient must take responsibility for protecting the wound from contamination, further trauma, and swelling.

The final appearance of a scar is determined by several factors. Infection, tissue necrosis, and keloid formation widen a scar. Wounds located in sebaceous skin or oriented 90° to dynamic or static skin tension lines result in wide scars.

## CONCLUSION

The objective of traumatic wound management is the restoration of tissue continuity and strength in the least possible time, with maximal preservation of tissues and minimal scar formation, deformity, or loss of function.

It is important that physicians follow the basic principles of wound care when cleaning, debriding, closing, and protecting wounds and continue to refine their management of wounds as further improvements in techniques and materials become available.

# Chapter 37 - Methods of Wound Closure

**Richard L. Lammers, Alexander T. Trott**

Once the decision to close a wound has been made, the physician must select the closure technique best suited for the location and configuration of the wound. Available techniques include hair tying (in the scalp) and use of tape, tissue adhesive, metal staples, and sutures. All traumatic wounds should be cleaned, and wounds containing devitalized tissue should be debrided prior to closure (see Chapter 36) .

## HAIR TYING OF SCALP WOUNDS

Scalp wounds that gape have traditionally been closed with suture material or skin staples. One "folk method" of scalp wound closure that has received limited discussion [1] and study [2] is the technique of tying together "roped" strands of hair from opposite sides of the wound.

The advantages of this technique are that no surgical instruments are required; no foreign material is placed in the wound; and it is relatively painless, because a local anesthetic injection is not needed. This technique may be of particular value in wilderness settings when wound repair equipment is unavailable and the wound is relatively clean. In certain superficial scalp wounds in children, this technique offers a particularly humane method of wound closure.

### Indications and Contraindications

Closure by hair tying can be performed on small scalp wounds (i.e., 1 to 2 cm in length). Davies suggests the following 6 criteria for consideration of this technique [2] :

1. The patient's hair must be of adequate length to form "roped" strands that can be tied across the wound.
2. The wound edges should not be contused.
3. There should be no gross wound contamination.
4. There must be good wound hemostasis.
5. The galea (occipitofrontalis aponeurosis) must not be divided.
6. There must be no underlying skull fracture.When these conditions cannot be met, the technique should not be undertaken. If local anesthesia must be used to permit evaluation of the deep structures of the wound, it may be best to simply repair the wound with sutures or staples.

### Procedure

When possible, the area surrounding the wound should be cleansed with mild disinfectant, avoiding contact with the unanesthetized wound. The wound should be irrigated with normal saline. The wound should be gently explored using a gloved hand or cotton-tipped applicator to verify that the galea is not compromised and that no

foreign material remains in the wound.

Hair on each side of the laceration is then twisted to form "ropes" of hair (Fig. 37-1 *A*). These "roped" strands are tied across the wound in a surgical knot, with several additional throws (Fig. 37-1 *B*) to tightly appose the skin edges. Davies recommends spraying the knot with a plastic sealant to avoid loosening of the knot. [2]

Postclosure wound care is similar to that for routine scalp closure. The patient may gently shampoo the hair, but *vigorous* hair massage or combing in the area should be avoided. The knot is allowed to grow away from the wound edge and can be cut free in 2 to 4 weeks.

### Complications

In 1 series of 25 children under 8 years of age whose scalp wounds were closed by hair tying, 48-hour follow-up showed no evidence of wound infection and 2 cases of mild (2 to 4 mm) wound separation. [2] The investigators noted that some of the children complained of the sensation that their hair was "being pulled" during wound closure, but all cooperated without restraints or anesthesia. The most common complaint noted at follow-up was that the hair-tie knot was untidy.

### Conclusion

Closure of scalp wounds by hair tying offers an attractive alternative for closure of small, superficial scalp wounds in children and for clean scalp wound repair in wilderness settings.

## WOUND TAPE

The use of surgical tape strips to close simple wounds has become routine in recent years. Tape strips can be applied by health care personnel in many settings, including emergency departments, operating rooms, clinics, and first-aid stations. Advantages of tape strips include ease of application, reduced need for local anesthesia, more evenly distributed wound tension, no residual suture marks, minimal skin reaction, no need for suture removal, superiority for some grafts and flaps, and suitability for use under plaster casts. One of the main advantages of wound tapes is their greater resistance to wound infection compared with standard sutures and wound staples.

### Background and Tape Comparisons

Tape closure of wounds has been reported since 1600 B.C. [7] It was not until the late 1950s, however, with the introduction of woven tapes and nonsensitizing adhesive, that tapes gained widespread acceptance in the United States. [8] Since then, there have been rapid advances in the manufacture of tapes with increased strength, improved adhesiveness, and presterilized packaging.

Currently there are several brands of tapes with differing porosity, flexibility, strength, and configuration. Steri-Strips (3M Corporation, St. Paul, Minn) are microporous tapes with ribbed backing. They are porous to air and water, and the ribbed backing provides extra strength. Cover-Strips (Beiersdorf, South Norwalk, Conn) are woven in texture and have a high degree of porosity. They allow not only air and water, but also wound exudates to pass through the tape. Shur-Strip (Deknatel, Inc., Floral Park, NY) is a nonwoven microporous tape. Clearon (Ethicon, Inc., Somerville, NJ) is a synthetic plastic tape whose backing contains longitudinal parallel serrations to permit gas and fluid permeability. An iodoform-impregnated Steri-Strip (3M Corporation) is intended to further retard infection without sensitization to iodine. [3] Other tape products include Curi-Strip (Kendall, Boston), Nichi-Strip (Nichiban Co., Ltd, Tokyo), Cicagraf (Smith & Nephew, London), and Suture Strip (Genetic Laboratories, St. Paul).

Scientific studies of wound closure tapes have been limited, and because of different investigators' choices of products and methods, it is not always easy to compare results. Koehn showed that the Steri-Strip tapes maintained adhesiveness about 50% longer than Clearon tape. [9] Rodeheaver and coworkers compared Shur-Strip, Steri-Strip, and Clearon tape in terms of breaking strength, elongation, shear adhesion, and air porosity. [10] The tapes were tested in both dry and wet conditions. The Steri-Strip tape was found to have about twice the breaking strength of the other two tapes in both dry and wet conditions; there was minimal loss of strength in all tapes when wetted. The Shur-Strip tapes showed approximately two to three times the elongation of the other tapes at the breaking point, whether dry or wet. Shear adhesion (amount of force required to dislodge the tape when a load is applied in the place of contact (angle = 0°) was slightly better for the Shur-Strip tape than for the Steri-Strip tape and approximately 50% better than for the Clearon tape. Of these three wound tapes, the investigators considered Shur-Strips to be superior for wound closure.

One comprehensive study of wound tapes compared Curi-Strip, Steri-Strip, Nichi-Strip, Cicagraf, Suture Strip, and Suture Strip Plus. [11] All tapes were 12 mm wide except for Nichi-Strip, which was 15 mm. Each tape was compared for breaking strength, elongation under stress, air porosity, and adhesiveness. Curi-Strip, Cicagraf, and Steri-Strip exhibited equivalent dry breaking strengths. However, when wet (a condition that can occur in the clinical setting), Cicagraf outperformed all tapes. All of the tested tapes had similar elongation-under-stress profiles with the exception of Suture Strip Plus. This tape did not resist elongation under low or high forces. Excessive elongation may allow wound dehiscence. Nichi-Strip was the most porous to air, and Cicagraf was almost vapor impermeable. Nichi-Strip and Curi-Strip had the best adherence to untreated skin. When the skin was treated with tincture of benzoin, however, Steri-Strip dramatically outperformed all other products. When all of the study parameters were considered, Nichi-Strip, Curi-Strip, and Steri-Strip achieved the highest overall performance rankings.

**Indications**

The primary indication for tape closure is a superficial straight laceration under little tension. If necessary, tension can be reduced by undermining or placing deep closures.

Areas particularly suited for tape closure are the forehead, chin, malar eminence, thorax, and nonjoint areas of the extremities. Tape also may be preferred for wounds in anxious children when suture placement is not essential. In young children who are likely to remove tapes, tape closures must be protected with an overlying bandage. However, adhesive bandages (e.g., Band-Aids) should be avoided (see below).

In experimental wounds inoculated with *Staphylococcus aureus*, tape-closed wounds resisted infection better than wounds closed with nylon sutures. [6] Therefore, tape closures may be considered on wounds with potential for infection, although infection rates are generally comparable to those of sutured wounds. Tape closures work well under plaster casts when superficial suture removal would be delayed. Tape closures effectively hold flaps and grafts in place, particularly over fingers, the flat areas of the extremities, and the trunk. [3] [4] Wounds on the pretibial area are difficult to close. This area is particularly problematic in the elderly because of tissue

atrophy. One report found that wound tapes outperformed suture closure of the pretibial area with regard to time to healing and complications (Fig. 37-2 *A* and *B*). [12] Tape closures can be applied to wounds following early suture removal to maintain wound edge approximation while reducing the chance of permanent suture mark scarring. Finally, because of the minimal skin tension created by tapes, they can be used on skin that has been compromised by vascular insufficiency or altered by prolonged use of steroids.

## Contraindications

There also are disadvantages to tape closures. Tape does not work well on wounds that are under significant tension or on wounds that are irregular, on concave surfaces, or in areas of marked tissue laxity. In many cases tape does not provide satisfactory wound edge apposition without concurrent underlying deep closures. Tape does not stick well to naturally moist areas, such as in the axilla, the palms of the hands, the soles of the feet, and the perineum. Tape also has difficulty adhering to wounds that will have copious exudates.  Tape strips also can be prematurely removed by young children.

Tape closures are contraindicated in wounds that are irregular or under tension and in those that cannot be appropriately dried of blood or secretions. They are of little value on lax and intertriginous skin and in the scalp and beard areas.

Tapes should never be placed circumferentially around a digit, because they have insufficient ability to stretch or lengthen. If placed circumferentially, the natural wound edema of an injured digit can make the tape act like a constricting band, which can lead to ischemia and possible necrosis of the digit. Semicircular or spiral placement techniques should be used if digits are to be taped.

## Equipment

For a simple tape closure, the required equipment includes forceps and tape of the proper size. Most taping can be done in the emergency department with ¼-inch × 3-inch

strips. In wounds larger than 4 cm, however, ½-inch-wide strips might be desirable. Most companies manufacture strips up to 1 inch wide and up to 4 inches long.

## Procedure

Application of the tape must be preceded by proper wound preparation, irrigation, debridement, and hemostasis. Fine hair may be shaved, and the area of the tape application *is thoroughly dried to ensure proper adhesion*. Attempting to apply tapes to a wet area or over a wound that is slowly oozing blood will usually result in failure of the tapes to stick to the skin. On fingers, tapes can be applied to a wound that is kept dry by a tourniquet temporarily placed at the base of the finger.

Tincture of benzoin and Mastisol are liquid adhesives that can be applied initially to increase tape adhesion. [6] The physician should use sterile technique at all times. Wound tapes do not adhere unduly to surgical gloves, and sterile glove use allows the operator to maintain proper sterile technique. All tapes come in presterilized packages and can be opened directly onto the operating field.

The technique of applying tapes is shown in [Figures 37-3](#) *A- J.* After the wound has been dried and a liquid adhesive has been applied and has dried, the tapes, with backing attached, are cut to the desired length. Tapes should be long enough to allow for approximately 2 to 3 cm of overlap on each side of the wound. After the tape is cut to length, the end tab is removed. The tape is gently removed from the backing with forceps by pulling straight back. Do not pull to the side, because the tape will curl and be difficult to apply to the wound. One half of the tape is securely placed at the midportion of the wound. The opposite wound edge is gently but firmly apposed to its counterpart. The second half of the tape is then applied. The wound edges should be as close together as possible and at equal height to prevent the development of a linear, pitting scar. Additional tapes are applied by bisecting the remainder of the wound. A sufficient number of tape strips should be placed so that the wound is completely apposed without totally occluding the wound edges. Finally, additional cross tapes are placed to add support and prevent blistering caused by unsupported tape ends. [5]

Taped wounds are left open, without occlusive dressings. Adhesive bandages (e.g., Band-Aids) and other dressings promote excessive moisture, which can lead to premature tape separation from the wound. The bandage also may adhere to the closure tapes, causing separation of the closure tape from the skin at the time of the removal. Tapes may remain in place for approximately 2 weeks and in some cases longer. The duration of application is a decision that varies with the requirements of each wound. The patient can be allowed to clean the taped laceration gently with a moist, soft cloth after 24 to 48 hours. However, if excessive wetting or mechanical force is used, premature separation may result. Patients may be instructed to gently trim curled edges of the closure tape with fine scissors to avoid premature removal of the tape.

## Complications

Complications are uncommon with tape closure. The infection rate is approximately 5%

in clean wounds closed with tape. [5] This compares favorably with rates for other standard closures. Premature tape separation occurs in approximately 3% of cases. [10] Other complications include (1) skin blistering, which occurs if the tape is not properly anchored with the cross-stay strip or the tape is stretched excessively across the wound; and (2) wound hematoma, which results if hemostasis is inadequate.

When tincture of benzoin is used, it should be applied carefully to the surrounding uninjured skin. If spillage occurs into the wound, the wound is at higher risk for infection. [13] Benzoin vapors cause pain when applied near an open wound that has not been anesthetized. Benzoin can also injure the mucous membranes of the eye.

### Summary

Most investigators believe that the results of proper tape closure are as successful as those of suture closure. [5] [7] However, some investigators believe that tape closure leads to inferior cosmetic results. [14] In the aggregate, modern tape products and techniques serve a valuable role in minor wound management of selected patients in the emergency department. Generally, closure tapes are underused, and many wounds that are currently sutured in cosmetically unimportant areas could be adequately closed with tapes. As a general guide, tapes should be considered in those cases where sutures are not clearly required, but the wound is too wide for a simple dressing.

## TISSUE ADHESIVE

Adhesive tape can only be used on superficial wounds on relatively flat, hairless body surfaces. The tissue adhesive *N*-2-butylcyanoacrylate (Histoacryl glue) is a bonding agent that can be used on superficial wounds, even in hair-bearing areas. Tissue adhesive (also called *tissue glue*) polymerizes when it comes in contact with water. This substance is biodegradable but remains in the wound until well after healing. [15]

### Procedure

Tissue adhesive can be used to approximate wounds not requiring deep-layer closure. In preparation for closure, the wound should be anesthetized, cleaned, and debrided when indicated. Bleeding must be controlled.

A small volume of the adhesive solution is drawn into a syringe and 25-ga needle. Alternatively, the needle can be attached to the cut end of the cylindrical plastic container. As the wound edges are held together, droplets of tissue adhesive are placed over the laceration along the length of the wound. Alternatively, one can place the glue in strips perpendicular to the laceration (analogous to placement of closure tapes). The purple color of the solution facilitates placement of the droplets. The wound edges should be supported with edges approximated for at least 1 minute while the glue dries. The closure can be reinforced and protected with a bandage such as Elastoplast.

The primary advantage of tissue adhesive is the speed of closure. Wounds can be closed in as little as one sixth of the time required for repair with sutures. Although 1

animal study found that wounds closed with tissue adhesive had less tensile strength than sutured wounds at 4 days, [16] other studies testing wounds 1 week after closure found that tensile strength and overall degree of inflammation in wounds closed with adhesive were equivalent to those closed with sutures. [15] [17] Cosmetic results are similar to those obtained with suture repair. [18] [19] Although the glue should not be placed within the wound cavity, tissue adhesive does not cause a significant foreign body reaction. [17]

### Complications

Percutaneous sutures provide a more secure immediate closure than tissue adhesive. [15] Tissue adhesive should not be used near the eyes, over or near joints, or on wounds under significant static tension. If hemostasis is inadequate or an excessive amount of adhesive is applied too quickly, the patient can experience a burning sensation from the heat of polymerization.

### Summary

Tissue adhesive is currently being used in many countries, but its use remains investigational in the United States.

## WOUND STAPLES

### Background

Wound stapling devices date back to the early part of this century. Several Russian, Hungarian, and Japanese investigators pioneered various instruments, but it was not until the early 1960s that significant interest in the use of these devices developed in the United States. [20] [21] Since that time there has been a steady improvement in technology, including the introduction of automatic and disposable devices, precocking mechanisms, and optimal staple configurations.

Automatic stapling devices have become commonplace for closure of surgical incisions and are finding acceptance among clinicians for closure of traumatic wounds. Clinical studies of patients with stapled surgical incisions have consistently revealed that there is no significant difference between stapling and suturing when infection rates, healing outcome, and patient acceptance are compared. Four important studies have demonstrated that selected traumatic wounds in both adult and pediatric patients can be closed successfully with staples in the emergency department setting. Wound stapling and nylon suture closure of skin compared favorably in wound tensile strength, complication rates, patient tolerance, efficiency of closure, scar width, color, general appearance, suture or staple marks, infection rates, and cost. However, in 1 study more patients in the staple group reported discomfort with removal. [29] In animal models, staples cause less wound inflammation, preserve wound defense mechanisms, and offer more resistance to infection in contaminated wounds.

The most significant advantage of wound stapling over suturing is speed of closure. On

average, stapling is 3 to 4 times faster than suturing traumatic wounds. [27] [29] The time for actual staple application is 30 seconds or less for a laceration 3 to 5 cm in length. Cost has been cited as a disadvantage of staple closure, particularly when large, multistaple (25 to 35) surgical units are the only product available. [30] However, with the introduction of smaller devices more appropriate for the average laceration, the cost of stapling devices has been reduced significantly. [30] When physician time and cost of instruments are considered, the cost difference is minimal [27] or favors stapling. [37]

## Indications and Contraindications

Currently the indications for stapling are limited to relatively linear lacerations with straight, sharp edges located on an extremity, the trunk, or the scalp. Staples may be especially useful for superficial scalp lacerations in the agitated or intoxicated patient. Because of their superficial placement in the adult scalp (usually above the galea), staples are *not* ideal for *deep* scalp lacerations. Staples may not provide the same hemostasis that is possible with deep sutures. Also, they should not be placed in scalp wounds if computed tomography head scans are to be performed because staples produce scan artifacts. Similarly, staples should not be used if the

patient is expected to undergo magnetic resonance imaging, because the powerful magnetic fields may avulse the staples from the skin surface. As they are currently configured and manufactured, staples *should not be used on the face, neck, hands, or feet*.

## Equipment

Standard wound care should precede wound closure (see Chapter 36) . In many cases, when debridement and dermal (deep) closures are unnecessary, only tissue forceps are needed to assist in everting wounds.

Many stapling devices are commercially available. The most versatile and least expensive stapler is the Precise (3M Corporation). Different units can be purchased that hold between 5 and 25 staples. The 10-staple unit will suffice for most lacerations. Other devices include the Proximate 11 (Ethicon, Inc.), Cricket (US Surgical, Irvine, Calif), and Appose (Davis & Geck, Columbus, Ohio). These staplers have

**Figure 37-4** (Figure Not Available) The skin edges must be approximated and everted by hand or with forceps before they are secured with staples. Failure to evert the wound edges is a common error that may cause an unacceptable result. *(Adapted with permission from Edlich RF: A Manual for Wound Closure. St. Paul, Minn, 3M Medical-Surgical Products, 1979. Reproduced by permission.)*

a minimum of 15 staples and are 3 to 5 times more expensive than the Precise stapler.

## Procedure

The wound is prepared in the manner described in Chapter 36 . Whenever necessary, deep, absorbable sutures are used to close deep fascia and to reduce tension in the

superficial fascia and dermal layers. Before stapling, the wound edges should be everted, preferably by a second operator. The assistant precedes the operator along the wound and everts the wound edges with forceps or pinches the skin with the thumb and forefinger. This technique allows the staple to be precisely placed. Once the edges are held in eversion, the staple points are gently placed across the wound (Fig. 37-4) (Figure Not Available) . By squeezing the stapler handle or trigger, the staple is advanced automatically into the wound and bent to the proper configuration (Figs. 37-5 (Figure Not Available) and 37-6) (Figure Not Available) . One must take care not to press too hard on the skin surface in order to prevent placing the staple too deeply and causing ischemia within the staple loop. When properly placed, the *crossbar of the staple is elevated a few millimeters above the skin surface* (Fig. 37-7) (Figure Not Available) . Enough staples should be placed to provide proper apposition of the edges of the wound along its entire length.

After the wound is stapled, an antibiotic ointment may be applied to minimize dressing adherence, and a sterile dressing is applied. If necessary, the patient can remove the dressing and gently clean the wound in 24 to 48 hours. The interval between staple application and removal is the same as that for standard suture placement and removal.

Removal of staples requires a special instrument that is made available by each manufacturer of stapling devices. The lower jaw of the staple remover is placed under the crossbar (Fig. 37-8) (Figure Not Available) . One brings down the upper jaw by squeezing the handle (Fig. 37-9) (Figure Not Available) . This action compresses the crossbar, thereby releasing the staple points for easy removal. If the patient is referred for office removal of staples, it may be advisable to provide the patient with the staple removal device on emergency department release, because many physicians do not routinely stock the instrument. Staples should be removed under the same timetable as sutures.

**Figure 37-9** (Figure Not Available) By squeezing the handle gently, the upper jaw compresses the staple and allows it to exit the skin. *(Adapted from Edlich RF: A Manual for Wound Closure. St. Paul, Minn, 3M Medical-Surgical Products, 1979. Reproduced by permission.)*

## Complications

Complications can occur with staple-closed wounds, although the incidence is low and equivalent to that for sutured wounds. In 2 studies of traumatic wounds closed with staples, the infection rates were reported to be 0% and 5%. [29] [30] Staple acceptance and comfort have been reported to be equal to those of sutures, but in 1 study, removal of staples was somewhat more uncomfortable than removal of sutures. [29] Wound dehiscence has been reported, but the incidence is not considered significant. [29] A common error is failure to *evert* the skin edges before stapling (Fig. 37-10 *A* and *B*). Eversion avoids the natural tendency of the device to invert the closure. Eversion may be accomplished with forceps or by pinching the skin with the thumb and index finger, a procedure that requires some practice. Staples do cause marks in the skin similar to sutures. In patients who tend to scar more easily, the resulting scar from the staples may be more pronounced than that produced by sutures, especially if the staples are

left in place for prolonged periods.

## Conclusion

Overall results are favorable when staples are used for surgical incisions and traumatic lacerations of the scalp, trunk, and extremities. Wound stapling does not differ significantly from suturing in infection rates, wound healing, and patient acceptance. Stapling is clearly superior in reducing time to closure. With the introduction of new devices, the cost of wound stapling is comparable to that of suturing. Because of the increased availability and versatility of stapling instruments, they are being used more frequently in emergency department wound management.

# SUTURES

In most situations, suturing is the closure method of choice. Currently in the United States, most traumatic wounds are closed with sutures.

## Equipment

### Instruments

In addition to the instruments used for debridement, a needle holder and suture scissors are required for suturing. The size of the needle holder should match the size of the needle selected for suturing--that is, the needle holder should be large enough to hold the needle securely as it is passed through tissue, yet not so large that the needle is crushed or bent by the instrument. The mechanical performance of disposable needle holders distributed by different surgical instrument companies varies considerably. [38] Instruments used to debride a grossly contaminated wound should be discarded and fresh instruments obtained for the closure of the wound. Instruments covered with coagulated blood can be cleansed with hydrogen peroxide, rinsed with sterile saline or water, and then used for suturing.

### Suture Materials

A wide variety of suture materials are available. For most wounds that require closure of more than 1 layer of tissue, the physician must choose sutures from 2 general categories: an absorbable suture for the subcutaneous (SQ) layer and a nonabsorbable suture for skin closure.

Sutures can be described in terms of four characteristics:

1. Composition (i.e., chemical and physical properties)
2. Handling characteristics and mechanical performance
3. Absorption and reactivity
4. Size and retention of tensile strength

**Composition.**

Sutures are made from natural fibers (cotton, silk), from sheep submucosa or beef serosa (plain gut, chromic gut), or from synthetic materials such as nylon (Dermalon, Ethilon, Nurulon, Surgilon), Dacron (Ethiflex, Mersilene), polyester (Ti-Cron), polyethylene (Ethibond), polypropylene (Prolene, Surgilene), polyglycolic acid (Dexon), and polyglactin (Vicryl, coated Vicryl). Stainless steel sutures are rarely, if ever, useful in wound closure in the emergency department setting because of handling difficulty and fragmentation. Some sutures are made of a single filament (monofilament); others consist of multiple fibers braided together (Table 37-1) . [39]

**Handling and performance.**

Desirable handling characteristics

| TABLE 37-1 -- Examples of Suture Materials | |
|---|---|
| **Filament Type Absorbable Sutures** | **Nonabsorbable Sutures** |
| Monofilament | |
| Plain gut | Dermalon * |
| Chromic gut | Ethilon * |
| PDS | Prolene |
| | Silk |
| | Steel |

|  | Surgilene |
|  | Tevdek |
| Multifilament |  |
| Dexon ¶ | Ethibond § |
| Vicryl * | Mersilene |
| Coated Vicryl * | Nurulon * |
|  | Surgilon * |
|  | TiCron |

in a suture include smooth passage through tissues, ease in knot tying, and stability of the knot once tied. Smooth sutures pull through tissues easily, but knots slip more readily. Conversely, sutures with a high coefficient of friction have better knot-holding capacity but are difficult to slide through tissues. Smooth sutures will loosen after the first throw of a knot is made, and a second throw is needed to secure the first in place. However, the physician may want to tighten a knot further after the first throw is made. This is difficult with rougher types of sutures.

Multifilament sutures have the best handling characteristics of all sutures, whereas steel sutures have the worst. In terms of performance and handling, significant improvements have been made in the newer absorbable sutures. Gut sutures have many shortcomings, including relatively low and variable strength, a tendency to fray when handled, and stiffness despite being packaged in a softening fluid. [40] [41] Multifilament synthetic absorbable sutures are soft and easy to tie and have few problems with knot slippage. Polyglactin 910 (coated Vicryl) sutures have been improved with the application of an absorbable lubricant coating. The "frictional drag" of these coated sutures as they are pulled through tissues is less than that of uncoated multifilament

materials, and the resetting of knots following the initial throw is much easier. This characteristic allows retightening of a ligature without knotting or breakage and with smooth, even adjustment of suture line tension in running subcuticular stitches. [42] Synthetic monofilament sutures have the troublesome property of "memory"--a tendency of the filament to spring back to its original shape, which causes the knot to slip and unravel. Some nonabsorbable monofilament sutures are coated with polytetrafluorethylene (Teflon) or silicone to reduce their friction. This coating improves the handling characteristics of these monofilaments but results in poorer knot security. [41]

Three square knots will secure a stitch made with silk or other braided, nonabsorbable materials, and 4 knots are sufficient for synthetic, absorbable and nonabsorbable monofilament sutures. [43] Five knots are needed for the Teflon-coated synthetic Tevdek. [44] With the use of coated synthetic suture materials, attention to basic principles of knot tying is even more important. An excessive number of throws in a knot weakens the suture at the knot. If the physician uses square knots (or a surgeon's knot on the initial throw, followed by square knots) that lie down flat and are tied securely, knots will rarely unravel. [45]

**Absorption and reactivity.**

Sutures that are rapidly degraded in tissues are termed *absorbable*; those that maintain their tensile strength for longer than 60 days are considered *nonabsorbable* (see Table 37-1) . Plain gut may be digested by white blood cell lysozymes in 10 to 40 days; chromic gut will last 15 to 60 days. Remnants of both types of sutures, however, have been seen in wounds more than 2 years after their placement.  A newer type of catgut (Ethicon) is rapidly absorbed within 10 to 14 days but with less inflammation than that caused by chromic catgut. [47] Vicryl is absorbed from the wound site within 60 to 90 days, [40] [43] and Dexon, within 120 to 210 days. [48] [49] When placed in the oral cavity, plain gut disappears after 3 to 5 days, chromic gut after 7 to 10 days, and polyglycolic acid after 16 to 20 days. [50] In contrast, SQ silk may not be completely absorbed for as long as 2 years. [43] The rate of absorption of synthetic absorbable sutures is independent of suture size. [48]

Sutures may lose strength and function before they are completely absorbed in tissues. Braided synthetic absorbable sutures lose nearly all of their strength after about 21 days. In contrast, monofilament absorbable sutures (modified polyglycolic acid [Maxon, Davis & Geck] and polydioxanone [PDS, Ethicon]) retain 60% of their strength after 28 days. [51] [52] Gut sutures treated with chromium salts (chromic gut) have a prolonged tensile strength; however, all gut sutures retain tensile strength erratically. [40] [43] Of the absorbable types of sutures, a wet and knotted polyglycolic acid suture is stronger than a plain or chromic gut suture subjected to the same conditions. [41] [53]

Polypropylene remains unchanged in tissue for longer than 2 years after implantation. [54] In comparison testing, Hermann found that sutures made of natural fibers such as silk, cotton, and gut were the weakest; sutures made of Dacron, nylon, polyethylene, and polypropylene were intermediate in tensile strength; and metallic sutures were the strongest. [41] Kaplan and Hentz used the comparison of suture strength versus wound

strength as a measure of the usefulness of a suture. They stated that catgut is stronger than the soft tissue of a wound for no more than 7 days; chromic catgut, Dexon, and Vicryl are stronger for 10 to 21 days; and nylon, wire, and silk are stronger for 20 to 30 days. [55]

All sutures placed within tissue will damage host defenses and provoke inflammation. Even the least reactive suture impairs the ability of the wound to resist infection. [54] The magnitude of the reaction provoked by a suture is related to the quantity of suture material (diameter times total length) placed in the tissue and to the chemical composition of the suture. Among absorbable sutures, polyglycolic acid and polyglactin sutures are least reactive, followed by chromic gut. Nonabsorbable polypropylene is less reactive than nylon or Dacron. [41] [56] [57] Significant tissue reaction is associated with catgut, silk, and cotton sutures; highly reactive materials should be avoided in contaminated wounds. Adams found absorbable polyglycolic acid sutures to be less reactive than those of nonabsorbable silk. [58]

The chemical composition of sutures is an important determinant of early infection. The infection rate in experimental wounds when polyglycolic acid sutures are used is less than the rate when gut sutures are used. It is surprising that plain gut sutures elicit infection less often in contaminated wounds than do chromic gut sutures. [54] Lubricant coatings on sutures do not alter suture reactivity, absorption characteristics, breaking strength, or the risk of infection. [42] [54] Multifilament sutures provoke more inflammation and are more likely to produce infection than monofilament sutures if left in place for prolonged periods. [59] [60] Monofilament sutures elicit less tissue reaction than do multifilament sutures, and multifilament materials tend to wick up fluid by capillary action. Bacteria that adhere to and colonize sutures can envelop themselves in a glycocalix that protects them from host defenses, [61] or they can "hide" in the interstices of a multifilament suture and, as a result, be inaccessible to leukocytes. [59] Polydioxanone (PDS) provides the advantages of a monofilament suture in an absorbable form, making it a good choice as a subcuticular stitch. Polypropylene sutures have a low coefficient of friction, and subcuticular stitches with this material are easy to pull out. [62]

**Size and strength.**

Size of suture material (thread diameter) is a measure of the tensile strength of the suture; threads of greater diameter are stronger. The strength of the suture is proportional to the square of the diameter of the thread. Therefore, a 4-0 size suture of any type is larger and stronger than a 6-0 suture. The correct suture size for approximation of a layer of tissue depends on the tensile strength of that tissue. The tensile strength of the suture material should be only slightly greater than that of the tissue, because the magnitude of damage to local tissue defenses is proportional to the amount of suture material placed in the wound. [43] [63]

Synthetic absorbable sutures have made the older, natural suture materials obsolete. Polyglycolic acid (Dexon) and polyglactin 910 (coated Vicryl) have improved handling characteristics, knot security, and tensile strength. Their absorption rates are predictable, and tissue reactivity is minimal. [64] [65] The distinct advantages of synthetic

nonabsorbable sutures over silk sutures are their greater tensile strength, low coefficient of friction, and minimal tissue reactivity. [54] [64] They are extensible, elongating without breaking as the edges of the wound swell in the early postoperative period. [63] [64] In contrast with silk sutures, synthetics can be easily and painlessly removed once the wound has healed. The monofilament synthetic suture Novofil has elasticity that allows a stitch to enlarge with wound edema and to return to its original length once the edema subsides. Stiffer materials lacerate the encircled tissue as the wound swells. [66]

The suture materials most useful to emergency physicians for wound closure are Dexon or coated Vicryl for SQ layers and synthetic nonabsorbable sutures (e.g., nylon or polypropylene) for skin closure. Fascia can be sutured with either absorbable or nonabsorbable materials. In most situations, 3-0 or 4-0 sutures are used in the repair of fascia, 4-0 or 5-0 absorbable sutures in SQ closure, and 4-0 or 5-0 nonabsorbable sutures in skin closure. Lips, eyelids, and the skin layer of facial wounds are repaired with 6-0 sutures, whereas 3-0 or 4-0 sutures are used when the skin edges are

**Figure 37-11** (Figure Not Available) The eyeless, or "swaged," needle. *(From Suture Use Manual: Use and Handling of Sutures and Needles. Somerville, NJ, Ethicon, Inc, 1977, p 29. Reproduced by permission.)*

subjected to considerable dynamic stresses (e.g., wounds overlying joint surfaces) or static stresses (e.g., scalp).

**Needles**

The eyeless, or "swaged," needle is used for wound closure in most emergency centers (Fig. 37-11) (Figure Not Available) . The traditional closed-eye needle requires additional handling to enable one to thread the needle with the suture, and its increased width causes more damage when passing through tissue than does a swaged needle.

Selection of the appropriate needle size and curvature is based on the dimensions of the wound and the characteristics of the tissues to be sutured. The needle should be large enough to pass through tissue to the desired depth and then to exit the tissue or the skin surface far enough that the needle holder can be repositioned on the distal end of the needle at a safe distance from the needle point (Fig. 37-12) . While it is inviting to use the fingers to grasp the needle tip to pull the needle through the skin, this practice is an invitation for a needle stick. The clinician should either reposition the needle holder or use forceps to disengage the needle from the laceration.

In wound repair, needles must penetrate tough, fibrous tissues--skin, SQ tissue, and fascia--yet should slice through these tissues with minimal resistance or trauma and without bending. The type of needle best suited for closure of SQ tissue is a *conventional cutting needle* in a three-eighths or one-half circle (Fig. 37-13) . The use of double curvature

**Figure 37-13** One-half and three-eighths circle needles, used for most traumatic wound closures.

needles (coated Vicryl with PS-4-C cutting needles, Ethicon) may enhance the physician's ability to maneuver the needle in narrow, deep wounds. For percutaneous closure, a conventional cutting-edge needle may permit more precise needle placement and require less penetration force (Fig. 37-14) (Figure Not Available) . [67] [68]

## Suturing Techniques

### Skin Preparation

Before closing the wound, the skin surrounding it is prepared with a povidone-iodine solution and covered with sterile drapes. Some surgeons do not drape the face but prefer to leave facial structures and landmarks adjacent to the wound uncovered and within view. A clear plastic drape (Steri-Drape, 3M Corporation) can be used to provide a sterile field and a limited view of the area surrounding the wound. If no drapes are used on the face, the skin surrounding the wound should be widely cleansed and prepared. Wrapping the hair in a sheet prevents stray hair from falling into the operating field (Fig. 37-15) (Figure Not Available) . Some emergency departments keep a supply of oversized scrub hats to use as an alternative to wrapping.

### Closure Principles

Three principles apply to the suturing of lacerations in any location: (1) minimize trauma to tissues, (2) relieve tension exerted on the wound edges by undermining and layered wound closure, and (3) accurately realign landmarks and skin edges by layered closure and precise suture placement.

**Minimizing tissue trauma.**

The importance of careful handling of tissue has been emphasized since the early days of surgery. Skin and SQ tissue that has been stretched, twisted, or crushed by an instrument or strangled by a suture that is tied too tightly may undergo necrosis, and increased scarring and infection may result. When the edges of a wound must be manipulated, the SQ tissues should be lifted gently with a toothed forceps or skin hook, avoiding the skin surface.

When choosing suture sizes, the physician should select the smallest size that will hold the tissues in place. Skin

**Figure 37-15** (Figure Not Available) *A-D,* Technique for wrapping the scalp to keep stray hair from falling into the operating field. A scrub hat is an acceptable alternative.

stitches should incorporate no more tissue than is needed to coapt the wound edges with little or no tension. Knots should be tied securely enough to approximate the wound edges but without blanching or indenting the skin surface. [69]

**Relieving tension.**

Many forces can produce tension on the suture line of a reapproximated wound. Static skin forces that stretch the skin over bones cause the edges of a fresh wound to gape and also continuously pull on the edges of the wound once it has been closed. Traumatic loss of tissue or wide excision of a wound may have the same effect. The best cosmetic result occurs when the long axis of a wound happens to be parallel to the direction of maximal skin tension; this alignment brings the edges of the wound together. [66]

Muscles pulling at right angles to the axis of the wound impose dynamic stresses. Swelling following an injury creates additional tension within the circle of each suture. [69] Skin suture marks result not only from tying sutures too tightly, but also from failing to eliminate underlying forces distorting the wound. Tension can be reduced during wound closure in two ways: undermining of the wound edges and layered closure.

**Undermining.**

The force required to reapproximate the wound edges correlates with the subsequent width of the scar. [70] Wounds subject to significant static tension require the undermining of at least 1 tissue plane on both sides of the wound to achieve a tension-free closure. Undermining involves the creation of a flap of tissue freed from its base at a distance from the wound edge that is approximately equal to the width of the gap that the laceration presents at its widest point (Fig. 37-16) . The depth of the incision can be modified, depending on the orientation of the laceration to skin tension lines and the laxity of skin in the area. A No. 15 scalpel blade held parallel to the skin surface is used to incise the adipose layer or the dermal layer of the wound. The clinician also can accomplish this technique by spreading scissors in the appropriate tissue plane. Undermining allows the skin edges to be lifted and brought together with gentle traction. [71] Because undermining may harm the underlying blood supply, this technique should be reserved for relatively uncontaminated wounds. [66] Other potential complications of this procedure include injury to cutaneous nerves and creation of a hematoma under the flap. [55]

**Layered closure.**

The structure of skin and soft tissue varies with the location on the body (Fig. 37-17 *A-D*). The majority of wounds handled in an emergency department require approximation of no more than 3 layers: fascia (and associated muscle), SQ tissue, and skin surface (papillary layer of dermis and epidermis). [72]

Closure of individual layers obliterates "dead space" within the wound that would otherwise fill with blood or exudate. The presence of dead space enhances the development of infection; however, it is not necessary to close the adipose layer of soft tissue with a separate stitch. A "fat stitch" is not necessary, because little support is provided by closure of the adipose layer, and the additional suture material that is

required may enhance the possibility of infection. [6] [73]

Separate approximation of muscle and SQ layers hastens the healing and return of function to the muscle. However, *one should suture fascia, not muscle*. Muscle tissue itself is too friable to hold a suture. Layered closure is particularly important in the management of facial wounds; this technique prevents scarring of muscle to the SQ tissue and consequent deformation of the surface of the wound with contraction of the muscle. If a deep, gaping wound is closed without approximation of underlying SQ tissue, a disfiguring depression may develop at the site of the wound. Finally, layered closure provides support to the wound and considerably reduces tension at the skin surface.

There are exceptions to the general rule of multilayered closure. Scalp wounds are generally closed in a single layer. For lacerations penetrating the dermis in fingers, hands, toes, and feet, the amount of SQ tissue is too small to warrant layered closure; in fact, SQ stitches may leave tender nodules in these sensitive locations. In the sebaceous skin of the nasal tip, SQ sutures should be avoided, because they provoke inflammation and increase the risk of infection. Layered closure is not recommended in wounds without tension, those with poor vascularity, and those with moderate infection potential. With single-layer closure, the surface stitch should be placed more deeply. [55]

**Suture Placement**

Before suturing, the physician should ensure adequate exposure and illumination of the wound. The physician should assume a comfortable standing or sitting position, with the patient placed at an appropriate height. The best position for the physician is at one end of the long axis of the wound.

**SQ layer closure.**

Once fascial structures have been reapproximated, the SQ layer is sutured. Although histologically the fatty and fibrous SQ tissue (hypodermis) is an extension of (and is continuous with) the reticular layer of the dermis, [74] suturing of these layers is traditionally referred to as an "SQ closure." One approach is to close this layer in segments, placing the first stitch in the middle of the wound and bisecting each subsequent segment until the closure of the

**Figure 37-18** Technique of handling the needle holder. An alternate method is shown in Figure 37-26 (Figure Not Available) .

layer has been completed. [39] This technique is useful in the closure of wounds that are long or sinuous and is particularly effective in wounds with one elliptic and one linear side. The needle is grasped by the needle holder close to the suture end. Greater speed in suturing is possible if the fingers are placed on the midshaft of the needle holder rather than in the rings of the instrument (Fig. 37-18) .

The suture enters the SQ layer at the bottom of the

**Figure 37-19** *A* and *B,* Inverted SQ stitches.

wound (Fig. 37-19 *A*) or, if the wound has been undermined, at the base of the flap (Fig. 37-19 *B*) and exits in the dermis. Once the suture has been placed on one side of the wound, it can be pulled across the wound to the opposite side (or the wound edges pushed together) to determine the matching point on the opposite side. It is at this matching point along the opposite side of the wound that the needle is inserted. The needle should enter the dermis at the same depth as it exited from the opposite side, pass through the tissue, and exit at the bottom of the wound (or the base of the flap). The edges of the wound can be closely apposed by pulling the 2 tails of the suture in the same direction along the axis of the wound (Fig. 37-20) . Some physicians place their SQ suture obliquely rather than vertically to facilitate knot tying. When the knot in this SQ stitch is tied, it will remain inverted, or "buried," at the bottom of the wound. Burying the knot of the SQ stitch avoids a painful, palpable nodule beneath the epidermis and keeps the bulk of this foreign material away from the skin surface. The techniques of tying knots by hand and by instrument are well described and illustrated in wound care texts. [75] [76] Once the knot has been secured, the tails of the suture should be pulled taut for cutting. The scissors are held with the index finger on the junction of the two blades. The blade of the scissors is slid down the tail of the suture until the knot is reached. With the cutting edge of the blade tilted away from the knot, the tails are cut. This technique prevents the scissors from cutting the knot itself and leaves a tail of 3 mm, which protects the knot from unraveling (Fig. 37-21) (Figure Not Available) . [77] The entire SQ layer is sutured in this manner.

After the SQ layer has been closed, the distance between the skin edges indicates the approximate width of the scar in its final form. If this width is acceptable, percutaneous sutures can be inserted. [78] Despite undermining and placement of a sufficient number of SQ sutures, on rare occasions a large gap between the wound edges may persist. In such cases a horizontal dermal stitch may be used to bridge this gap (Fig. 37-22) .

**Skin closure.**

The epidermis and the superficial layer of dermis are sutured with nonabsorbable synthetic sutures. The choice of suture size, the number of sutures used, and the depth of suture placement depend on the amount of skin tension remaining after SQ closure. If the edges of the wound are apposed following closure of deeper layers, small 5-0 or 6-0 sutures can be used simply to match the epithelium of each side. If the wound edges remain retracted or if SQ stitches were not used, a larger size suture may be required. Skin closure may be accomplished with sutures placed in segments (Fig. 37-23) or from end to end. Either technique is acceptable.

Unless the wound edges are uneven, sutures should be placed in a mirror-image fashion such that *the depth and width are the same on both sides of the wound.*[43] In

general, the distance between each suture should be approximately equal to the distance from the exit of the stitch to the wound edge. [39] [75] Grabb suggests that "the number of sutures used in closing any wound will vary with the case, location of the repair, and degree of accuracy required by the physician and patient. In an area such as the face, sutures would probably be placed between 1 and 3 mm apart and 1 to 2 mm from the wound edge." [72]

Sutures act as foreign bodies in a wound, and any stitch may damage a blood vessel or strangulate tissue. Therefore, the physician should strive to use the smallest size and the least number of sutures that will adequately close the wound (Fig. 37-24) . [79] Wounds with greater tension should have skin stitches placed closer to each other and closer to the wound edge; layered closure is important in such wounds. If sutures are tied too tightly around wound edges or if individual stitches are under excessive tension, blood supply to the wound may be impeded, increasing the chance of infection, and suture marks may form even after 24 hours. [43] [80]

When suturing the skin, right-handed operators should pass the needle from the right side of the wound to the left. The needle should enter the skin at an oblique angle to produce an everting, bottle-shaped stitch that is *deeper than it is wide* (Fig. 37-25) (Figure Not Available) . If the skin stitch is intended to produce some eversion of the wound edges, the stitch must include a sufficient amount of SQ tissue. However, encompassing too much tissue with a small needle is a common error. Forcefully pushing or twisting the needle in an effort to bring the point out of the tissue may bend or break the body of the needle. Using a needle of improper size will defeat the best suturing technique. The needle should be driven through tissue by flexing the wrist and supinating the forearm; the course taken by the needle should result in a curve identical to the curvature of the needle itself (Fig. 37-26) (Figure Not Available) . The angle of exit for the needle should be the same as its angle of entrance so that an identical volume of tissue is contained within the stitch on each side of the wound.

Once the needle exits the skin on the opposite side of the wound, it is regrasped by the needle holder and is advanced through the tissue; care should be taken to avoid crushing the point of the needle with the instrument. Forceps are designed for handling tissue and thus should not be used to grasp the needle. The forceps can stabilize the needle by holding the needle within the tissue through which the needle has just passed. Excess thread can be kept clear of the area being sutured by an assistant, or the excess can be looped around the operator's fingers. If the point of the needle becomes dulled before all of the attached thread has been used, the suture should be discarded.  If these techniques are applied to most wounds, the edges of the wound will be matched precisely in all three dimensions.

**Eversion techniques.**

If the edges of a wound invert or if one edge rolls under the opposite side, a poorly formed, deep, noticeable scar will result. Excessive eversion that exposes the dermis of both sides also will result in a larger scar than if the skin edges are perfectly apposed, but inversion produces a more visible scar than does eversion. Because most scars undergo some flattening with contraction, optimal results are achieved when the

epidermis is slightly everted without excessive suture tension (Fig. 37-27) (Figure Not Available) . Wounds over mobile surfaces, such as the extensor surfaces of joints, should be everted; in time, the scar will be flattened by the dynamic forces acting in the area.

A number of techniques can be used to avoid inversion of the edges of the wound. If the clinician angles the needle away from the laceration, percutaneous stitches can be placed so that their depth is greater than their width. [71] Converse described this method as follows: "The needle penetrates the skin close to the incision line, diverging from the edge of the wound in order to encircle a larger amount of tissue in the lower depths of the skin than at the periphery." [81] The edge of the wound can be lifted and everted with a skin hook or fine-tooth forceps before insertion of the needle on each side (Fig. 37-28) . Eversion can also be

**Figure 37-26** (Figure Not Available) Motion of the needle holder. *(From Anderson CB: Basic surgical techniques. In Klippel AP, Anderson CB: Manual of Outpatient and Emergency Surgical Techniques. Boston, Little, Brown, 1979. Reproduced by permission.)*

obtained simply by slight retraction of the wound with the thumb (Fig. 37-29) (Figure Not Available) . This technique puts the operator at risk for a needle stick; eversion may be done more safely by applying slight pressure on the wound edge with a closed forceps. Each of these methods also serves to steady the skin against the force of the needle. [75] [81] Vertical mattress sutures are particularly effective in everting the wound edges and can be used exclusively or alternated with simple interrupted sutures (Fig. 37-30) (Figure Not Available) . [81] In wounds that have been undermined, an SQ stitch placed at the base of the flap on each side can in itself evert the wound (Fig. 37-31) (Figure Not Available) .

**Interrupted stitch.**

The simple interrupted stitch is the most frequently used technique in the closure of skin. It consists of separate loops of suture individually tied. Although the tying and cutting of each stitch are time consuming, the advantage of this method is that if one stitch in the closure fails, the remaining stitches continue to hold the wound together (Fig. 37-32) (Figure Not Available) .

**Continuous stitch.**

In a continuous, or "running," stitch, the loops are the exposed portions of a helical coil that is tied at each end of the wound. A continuous suture line can be placed more rapidly than a series of interrupted stitches. The continuous stitch has the additional advantages of strength (with tension being evenly distributed along its entire length), fewer knots (which are the weak points of stitches), and more effective hemostasis. The continuous technique is useful as an epithelial stitch in cosmetic closures; however, if the underlying SQ layer is not stabilized in a separate closure, the continuous surface stitch tends to invert the wound edges.

The continuous suture technique has other disadvantages. This technique cannot be

used to close wounds overlying joints. If a loop breaks at 1 point, the entire stitch may unravel. Likewise, if infection develops and the incision must be opened at 1 point, cutting a single loop may allow the entire wound to fall open. There is also the theoretical problem of impeded blood supply to the wound edges, particularly if the suture is interlocked. [43] Speer found that wounds closed with an interrupted stitch had 30% to 50% greater tensile strength, less edema and induration, and less impairment in the microcirculation at the wound margin than did wounds closed with a continuous stitch. [82] The simple continuous stitch has a tendency to produce suture marks if used in large wound closures and if left in place for more than 5 days. [69] However, if all tension on the wound can be removed by SQ sutures, stitch marks are seldom a problem.

Among the variations of the continuous technique, the simple continuous stitch is the most useful to emergency physicians (Fig. 37-33) (Figure Not Available) . An interrupted stitch is placed at one end of the wound, and only the free tail of the suture is cut. As suturing proceeds, the stitch encircles tissue in a spiral pattern. After each passage of the needle, the loop is tightened slightly, and the thread is held taut in the physician's nondominant hand. The needle should travel perpendicularly across the wound on each pass. The last loop is placed just beyond the end of the wound, and the suture is tied, with the last loop used as a "tail" in the process of tying the knot (Fig. 37-34) . A locking loop may be used in continuous suturing to prevent slippage of loops as the suturing proceeds (Fig. 37-35) (Figure Not Available) . The interlocking technique allows the use of the continuous stitch along an irregular laceration.  A continuous stitch is an effective method for closing relatively clean wounds that are under little or no tension and are on flat, immobile skin surfaces in patients who have no medical conditions that would impair healing.

**Continuous subcuticular stitch.**

Nonabsorbable sutures used in percutaneous skin closure outlast their usefulness and must be removed. On occasion, wounds require an extended period of support, longer than that provided by surface stitches. Some patients with wounds that require skin closure are unlikely or unwilling to return for suture removal. Some sutured wounds are covered by plaster casts. On occasion, the patient (child or adult) is likely to be as

**Figure 37-35** (Figure Not Available) Continuous interlocking stitch. *(Modified from Suture Use Manual: Use and Handling of Sutures and Needles. Somerville, NJ, Ethicon, Inc, 1977. Reproduced by permission.)*

frightened and uncooperative for suture removal as for suture placement. The continuous subcuticular (or "dermal") suture technique is ideal for these situations; the wound can be closed with an absorbable subcuticular stitch, obviating the need for later suture removal. In patients prone to keloid formation, the subcuticular technique can be used in lieu of percutaneous stitches, and disfiguring stitch marks can thereby be avoided. (Because children's skin is under greater tension than that of adults, percutaneous sutures are more likely to produce stitch marks in children.) Because stitch marks are avoided, a nonabsorbable subcuticular suture can be left in place for a

longer period than a percutaneous suture. [81]

Although this technique is commonly used in cosmetic closures, some researchers believe that closure of the subcuticular layer alone does not alter the scar width. [83] This technique does not allow for perfect approximation of the vertical heights of the 2 edges of a wound, [84] and in cosmetic closures it is often followed by a percutaneous stitch. Although theoretically the large amount of suture material left in the wound might increase the risk of infection, some investigators report a lower infection rate with the subcuticular technique. [83] [85] Buried, absorbable subcuticular stitches do not appear to provoke more inflammation than percutaneous running stitches with monofilament nylon. [86]

The subcuticular stitch requires a 4-0 or 5-0 suture that is made of either absorbable material or nonabsorbable synthetic monofilament. An absorbable suture can be "buried" within the wound, whereas a nonabsorbable suture is used for a "pullout" stitch. The absorbable synthetic monofilament suture polydioxanone (PDS, Ethicon) is designed for subcuticular closure. It passes through tissues as easily as nonabsorbable monofilament sutures and is absorbed if left in the wound.

Before the subcuticular stitch is placed, the SQ layer should be approximated with interrupted sutures to minimize tension on the wound. The pullout subcuticular stitch is started at the skin surface approximately 1 to 2 cm away from 1 end of the wound. The needle enters and exits the dermis at the apices of the wound (Fig. 37-36) (Figure Not Available) . Bites through tissue are taken in a horizontal direction, with the needle penetrating the dermis 1 to 2 mm from the skin surface. These intradermal bites should be small, of equal proportion, and at the same level on each side of the wound. [64] [81] Accidental interlocking of the stitch should be avoided. Each successive bite should be placed 1 to 2 mm *behind* the exit point on the opposite side of the wound so that when the wound is closed, the entrance and exit points on either side are not directly apposed (see Fig. 37-36) (Figure Not Available) . Small bites should be taken to avoid puckering of the skin surface. Some physicians prefer to place a fine (6-0) running skin suture in addition to the subcuticular suture for meticulous skin approximation. The skin suture is removed in 3 to 4 days to avoid suture marks.

If the subcuticular stitch is used on lengthy lacerations, it is difficult to remove the suture. The placement of "reliefs" consisting of periodic loops through the skin during the length of the stitch facilitates later removal (Fig. 37-37) (Figure Not Available) . Reliefs should be placed every 4 to 5 cm. The suture is crossed to the opposite side, and the needle is passed from SQ tissue to the skin surface. The suture is carried over the surface for approximately 2 cm before reentering the skin and SQ tissue. The subcuticular stitch is then continued at

approximately the point at which the next bite would have been placed had the relief not been used.

At the completion of the stitch, the needle is placed through the apex to exit the skin 1 to 2 cm away from the end of the wound. One should tighten the stitch by pulling each end taut. If reliefs have been used, one can take up any slack in the stitch by pulling on the

reliefs. The clinician can secure the 2 ends of the stitch by taping them to the skin surface with wound closure tape, by placing a cluster of knots on each tail close to the skin surface, or by tying the 2 ends of the suture to each other over a dressing. Laxity of the subcuticular stitch is often noted with a decrease in tissue swelling 48 hours after wound closure. Some physicians tighten the stitch when they reexamine the wound after 48 hours.

Subcuticular closure using absorbable sutures that do not penetrate the skin is possible. The closure is begun with a dermal or SQ suture placed at one end of the wound and secured with a knot. After placement of the continuous subcuticular stitch from apex to apex, the suture is pulled taut, and a knot is tied using a tail and a loop of suture (Fig. 37-38) (Figure Not Available) . The final knot can be buried by inserting the needle into deeper tissue; the needle exits several millimeters from the wound edge. If one pulls on the needle end, the knot disappears into the wound. [64] The obvious advantage of this technique is that there are no suture marks in the skin. Another method that avoids penetrating the skin is the interrupted subcuticular stitch (Fig. 37-39) . [81] Wounds with strong static skin tension may benefit from a few interrupted dermal stitches placed horizontal to the skin surface instead of a continuous subcuticular stitch.

Nonabsorbable sutures can be left in place for 2 to 3 weeks, thus providing a longer period of support than percutaneous sutures, without the problem of stitch marks. [69] If skin sutures are used in conjunction with the subcuticular stitch, they are removed in 3 to 4 days. A subcuticular closure in itself is stronger than a tape closure. If the subcuticular technique is used exclusively to approximate the skin surface, it is advisable to apply skin tape to correct surface unevenness and to provide a more accurate apposition of the epidermis.

**Mattress stitch.**

The various types of mattress stitches are all interrupted stitches. The *vertical mattress stitch* is an effective method of everting skin edges (Fig. 37-40) (Figure Not Available) . The verticle mattress stich may be used to take a deep bite of skin in lieu of a layered closure in areas where excessive tension does not result. If the superficial loop is placed first, the tails can be pulled upward while the deep loop is placed, ensuring wound eversion in less time than with the traditional technique. [87] Unfortunately, this stitch causes more ischemia and necrosis inside its loop than either simple or continuous stitches. [88] The *horizontal mattress stitch* approximates skin edges closely while providing some degree of eversion (Fig. 37-41) . [69] The horizontal mattress suture may be ideal for areas where eversion is desirable but there is little SQ tissue. The *half-buried horizontal mattress stitch,* also called a *mattress stitch with a dermal component,* combines an interrupted skin stitch with a buried intradermal stitch (Fig. 37-42) (Figure Not Available) . It is effective in joining the edges of a skin flap to the edges of the "recipient site"; the dermal component is placed through the dermis of the flap. [81] The half-buried horizontal mattress stitch is also useful at the scalp-forehead junction when there is tension on the wound edges. This technique halves the number of suture marks in the skin and avoids necrosis of the edge of a skin flap.

The half-buried horizontal mattress stitch is particularly

useful in suturing the easily damaged apex of a V-shaped flap (Fig. 37-43) . In the execution of the "corner stitch," the suture needle penetrates the skin at a point beyond the apex of the wound and exits through the dermis. The corner of the flap is elevated, and the suture is passed through the dermis of the flap. The needle is then placed in the dermis of the base of the wound and returned to the surface of the skin. All dermal bites should be placed at the same level. The suture is tied with sufficient tension to pull the flap snugly into the corner without blanching the flap. [69] [89] If the tip of a large flap with questionable viability may be further jeopardized by postoperative swelling, a cotton stent can be placed underneath the knot of the corner stitch. The cotton absorbs the tension produced by swelling.

**Figure-of-eight stitch.**

The figure-of-eight stitch is useful in wounds with friable tissue, on the eyelids where the skin is too thin for buried sutures, or in areas in which buried sutures are undesirable (Fig. 37-44) (Figure Not Available) . [90] This stitch reduces the amount of tension placed on the tissue by the suture, allowing the stitch to hold in place when a simple stitch would tear through the tissue. One disadvantage of this technique is that more suture material is left in the wound. A vertical variation of the figure-of-eight stitch is sometimes used to approximate close, parallel lacerations (Fig. 37-45) (Figure Not Available) . [91] Another technique involves a vertical mattress stitch. The central "island" of tissue is secured by passing the superficial portion of the stitch through the island at the subcuticular level (Fig. 37-46) (Figure Not Available) . [92] If the viability of the central island is questionable and the surrounding tissue is loose, it can be excised.

**Correction of dog-ears.**

When wound edges are not precisely aligned horizontally, there will be an excess of tissue on 1 or both ends. This small flap of excess skin that bunches up at the end of a sutured wound is commonly

called a *dog-ear.* This effect also occurs when one side of the wound is more elliptical than the opposite side or when an excision of a wound is not sufficiently elliptical--that is, when it is either too straight or too nearly circular. [39] [81]

If a dog-ear is present, it can be eliminated on one side of the wound in the following manner: The flap of excess skin is elevated with a skin hook, and an incision is carried at an oblique angle from the apex of the wound toward the side with the excess skin. The flap is then undermined and laid flat. The resulting triangle of skin is trimmed, and the closure is completed (Fig. 37-47 (Figure Not Available) *A*). [78] [89] An alternative method consists of carrying the incision directly from the apex, in line with the wound. The flap of excess tissue is pulled over the incision while skin hooks are used to retract the extended apex of the wound. Excess tissue is excised, and the remainder of the wound is sutured. [81] If dog-ears are present on *both* sides of 1 end of the wound, the bulge of excess tissue can be excised in an elliptical fashion, and the wound can be closed (Fig. 37-47 (Figure Not Available) *B*). [89]

**V-Y advancement flap.**

If a corner stitch produces excessive tension on the tip of the flap, a V-Y closure can be used to approximate the edges without undue tension. An incision carried away from the apex of the wound converts it from a V to a Y configuration (Fig. 37-48) (Figure Not Available) . The newly formed wound edges are undermined, and the repair is completed. A half-buried mattress stitch is placed at the fork of the Y.

**Stellate lacerations.**

The repair of a stellate laceration is a challenging problem. Usually a result of compression and shear forces, these injuries contain large amounts of partially devitalized tissue. The surrounding soft tissue is often swollen and contused. Much of this contused tissue cannot be debrided without creating a large tissue defect. Sometimes tissue is lost, yet the amount is not apparent until key sutures are placed. In repairing what often resembles a jigsaw puzzle, the physician can remove small flaps of necrotic tissue with an iris scissors; large, viable flaps can be repositioned in their beds and carefully secured with half-buried mattress stitches. If interrupted stitches are used to approximate a thin flap, small bites should be taken in the flap and larger, deeper bites in the base of the wound. A modification of the corner stitch can be used to approximate multiple flaps to a base (Fig. 37-49) . The V-Y advancement flap technique is also useful. Thin flaps of tissue in a stellate laceration with beveled edges are often most easily repositioned and stabilized with a firm dressing. [69]

Closure of stellate lacerations cannot always be accomplished immediately, especially if there is considerable soft tissue swelling. It may be best in some instances to consider delayed closure or revision of the scar at a later date. In complicated lacerations, inexact tissue approximation may be all that is possible initially.

## Repair of Special Structures

### Facial Wounds (General Features)

The ideal result in the repair of a facial laceration is an extremely narrow, flat, and inapparent scar. In addition to basic wound management, a few additional techniques can be used to achieve this result. One of the factors that contributes to wide scars is necrosis of partially devitalized wound edges. However, skin with apparently marginal circulation may survive because of the excellent vascularity of the face. SQ fat, which in other locations may be debrided thoroughly, should be preserved if possible in facial wounds to prevent eventual sinking of the scar and to preserve normal facial contours. Therefore, debridement of most facial wounds should be conservative. [72] Facial and forehead lacerations that follow natural skin creases or lines will heal with a less noticeable scar than those that are oblique or perpendicular to the natural wrinkles of the skin (Fig. 37-50) .

Converse pointed out that "precise approximation of skin edges without undue tension ensures primary healing with minimal scarring." [81] A layered closure is essential in the cosmetic repair of many facial wounds. Approximation of the dermis with an SQ stitch or a combination of SQ and subcuticular stitches should bring the epithelial edges together or within 1 to 2 mm of apposition--close enough that the use of additional sutures seems almost unnecessary. [78] If an SQ stitch is the only stitch used to close the deeper layers, it should pass through the dermal-epidermal junction or within 1 to 2 mm of the skin surface without causing a dimpling effect. The clinician must tie this stitch snugly, pulling the 2 ends of the suture in the same direction (see Fig. 37-20) . Should the first SQ stitch placed at the midpoint of a wound perfectly appose the skin edges, one can "protect" that stitch from disruption during further suturing by immediately placing a percutaneous stitch in the same location. If there is a slight gap in the wound edges after SQ closure, the skin can be partially approximated with a few guide stitches. The first is placed at the midpoint of the wound, and subsequent stitches bisect the intervening spaces. Guide stitches allow the definitive epithelial sutures to be placed with little tension on each individual stitch, and they protect the SQ stitches from disruption. Once the definitive stitches have been placed, the guide stitches, if slack, can be removed. Because a needle damages tissue with each passage through the skin, guide stitches should be used only when necessary.

The epithelial stitch should never be used to relieve the wound of tension; it serves only to match the epidermal surfaces precisely along the length of the wound. If there is significant separation of the wound edges after closure of the SQ layer, a 5-0 or 6-0 subcuticular suture can be used to eliminate the tension produced by this separation and to provide prolonged stability. Once the skin edges are apposed, the epithelial stitch can be used to correct discrepancies in vertical alignment. A 6-0 synthetic nonabsorbable suture is an excellent material for this stitch. A continuous stitch is preferable because it can be placed quickly, but interrupted stitches are acceptable. In a straight laceration, better apposition is achieved if the wound is stretched lengthwise by finger traction or by the use of skin hooks. When the needle is placed on one side of the wound, if that side is higher than the opposite side, a shallow bite is taken. The needle

is used to depress the wound edge to the proper height, after which the needle "follows through" to the other side, pinning the two sides together. If the first side entered is lower, the needle is elevated when entering the second side to match the epithelial edges.

Grabb pointed out that "the closer the needle lies to the skin edge, the greater will be its effect in controlling the ultimate position of that edge." [59] Epithelial stitches should be spaced no more than 2 to 3 mm apart and should encompass no more than 2 to 4 mm of tissue. [71] If widely spaced, the sutures will leave marks. [78] Once skin closure is complete, final adjustments in the tension on any continuous suture line are made before the end of the stitch is tied. If any level discrepancies persist, interrupted sutures or tape can be used to flatten these few irregularities.

Surgical tape is useful as a secondary support, protecting the epithelial stitch from stresses produced by normal skin movements (Fig. 37-51) . Facial wounds have a tendency to swell and place excessive stretch on an epithelial stitch. This can be minimized by applying a pressure dressing and cold

**Figure 37-51** Wound closure tape can be used to provide additional support while sutures are in place and after they are removed. This may be especially useful in cosmetic areas, such as the face.

compresses to the wound following closure. Surgical tape can serve to a limited extent as a pressure dressing.

**Forehead**

Although the forehead is actually a part of the scalp, lacerations in this region are treated as facial wounds. Vertical lacerations across the forehead are oriented 90° to skin tension lines, and the resulting scars are more noticeable than those from horizontal lacerations. Midline vertical forehead lacerations may result in cosmetically acceptable scars with standard closure techniques; those lacerations that are not centered may benefit from S-plasty or Z-plasty techniques during the initial repair or during later revision of the scar.

Superficial lacerations may be closed with skin stitches alone, but deep forehead lacerations must be closed in layers. The periosteum should be approximated before the closure of more superficial layers. If skin is directly exposed to bone, adhesions may develop that in time may limit the movement of skin during facial expressions. The frontalis muscle fascia and adjacent fibrous tissue should be approximated as a distinct layer; if left unsutured, the retracted ends of this muscle will bulge beneath the skin. If the gap in a muscle belly is later filled with scar tissue, movement of the muscle pulls on the entire scar and makes it more apparent. [72]

A U-shaped flap laceration with a superiorly oriented base poses a difficult problem. Immediate vascular congestion and later scar contraction within the flap produce the "trap-door effect," with the flap becoming prominently elevated (Fig. 37-52) . This effect can be minimized by approximation of the bulk of SQ tissue of the flap to a deeper level

on the base side of the wound; the skin surfaces of the 2 sides are apposed at the same level (Fig. 37-53 *A*). A firm compression dressing helps eliminate "dead space" and hematoma formation within the wound. Despite these efforts, secondary revision is sometimes necessary. [69] Often, swelling of the flap resolves over a 6- to 12-month period. Because flap elevation can be quite disconcerting, the physician should forewarn the patient and family about a possible trap-door effect.

When a forehead laceration borders the scalp and the thick scalp tissue must be sutured to thinner forehead skin, a horizontal mattress stitch with an intradermal component can be used (Fig. 37-53 *B*). [81]

**Eyebrow and Eyelid Lacerations**

Jagged lacerations through eyebrows should be managed with little, if any, debridement of untidy but viable edges. The hair shafts of the eyebrow grow at an oblique angle, and vertical excision may produce a linear alopecia in the eyebrow, whereas with simple closure, the scar remains hidden within the hair. If partial excision is unavoidable, the scalpel blade should be angled in a direction parallel to the axis of the hair shaft to minimize damage to hair follicles.

Points on each side of the lacerated eyebrow should be aligned precisely; a single percutaneous stitch on each margin of the eyebrow should precede SQ closure. The edges of the eyebrow serve as landmarks for reapproximation; therefore, the eyebrow must not be shaved, as these landmarks will be lost. Shaved eyebrows grow back slowly and sometimes incompletely, and shaving them often results in more deformity than the injury itself. Care must be taken not to invert hair-bearing skin into the wound. [84]

The thin, flexible skin of the upper eyelid is relatively easy to suture. A soft 6-0 suture (or smaller) is recommended for closure of simple lacerations. Traumatized eyelids are susceptible to massive swelling; compression dressings and cool compresses can be used to minimize this problem.

It is essential that the emergency physician recognize complicated eyelid lacerations that require the expertise of an ophthalmologist. Lacerations that traverse the lid margin require exact realignment to avoid entropion or ectropion (Fig. 37-54 *A*). Injuries penetrating the tarsal plate frequently cause damage to the globe. A deep horizontal laceration through the upper lid that divides the thin levator palpebrae muscle or its tendinous attachment to the tarsal plate produces ptosis. If this muscle cannot be identified and repaired by the emergency physician, a consultant should repair the injury primarily. A laceration through the portion of the lower lid *medial* to the punctum frequently damages the lacrimal duct or the medial canthal ligament and requires specialized techniques for repair (Fig. 37-54 *B*). If adipose tissue is seen within any periorbital laceration, one must assume that the orbital septum has been penetrated and that retrobulbar fat is herniating through the wound (Fig. 37-54 *C*). The repair of lid avulsions, extensive lid lacerations with loss of tissue, and any of the other complex types of lid lacerations mentioned above should be left to ophthalmologists.

**Ear Lacerations**

The primary goals in the management of lacerations of the pinna are expedient coverage of exposed cartilage and minimization of wound hematoma (Fig. 37-54 *D* and *E*). Cartilage is an avascular tissue, and when ear cartilage is denuded of its protective, nutrient-providing skin, progressive erosive chondritis ensues. The initial step in the repair of an ear injury involves trimming away jagged or devitalized cartilage and skin. If the skin cannot be stretched to cover the defect, additional cartilage along the wound margin can be removed. Depending on the location, as much as 5 mm of cartilage can be removed without significant deformity. Cartilage should be approximated with 4-0 or 5-0 absorbable sutures initially placed at folds or ridges in the pinna representing major landmarks. Sutures tear through cartilage; therefore, the anterior and posterior perichondrium should be included in the stitch. No more tension should be applied than is needed to touch the edges together.

In through-and-through ear lacerations, the posterior skin surface should be approximated next, using 5-0 nonabsorbable synthetic sutures. Once closure of the posterior surface is completed, the convoluted anterior surface of the ear can be approximated with 5-0 or 6-0 nonabsorbable synthetic sutures, with landmarks joined point by point. On the free rim, the skin should be everted if later notching is to be avoided. Care should be taken to cover all exposed cartilage. In heavily contaminated wounds of the ear (e.g., bite wounds) that already show evidence of inflammation, the necrotic tissue should be debrided, the cartilage covered by a loose approximation of skin, and the patient placed on antibiotics. [69] [93] After a lacerated ear has been sutured, it should be enclosed in a compression dressing (see Fig. 68-24) .

**Lacerations of the Nose**

In the repair of lacerations of the nose, reapproximation of the wound edges is difficult because the skin is inflexible, and even deeply placed stitches will slice through the epidermis and pull out. When the wound edges cannot be coapted easily, 6-0 absorbable sutures can be placed in the fibrofatty junction in an SQ stitch before skin closure. Because it is difficult to approximate gaping wounds in this location, debridement must be kept to a minimum. Nasal cartilage is frequently involved in wounds of the nose, but it is seldom necessary to suture the cartilage itself.

The free rim of the nostril must be aligned precisely to avoid unsightly notching. Many physicians recommend early removal of stitches to avoid stitch marks, yet the oily nature of skin in this area makes it difficult to keep the wound closed with tape. A subcuticular stitch is recommended if the `wound is gaping before closure, as this will provide support for a prolonged period. [94]

**Lip and Intraoral Lacerations**

Lip lacerations are cosmetically deforming injuries, but if the physician follows a few

guidelines, these lacerations usually heal satisfactorily.

The contamination of all intraoral and lip wounds is considerable; they must be thoroughly irrigated. Regional nerve blocks are preferred to local injection, because the latter method distends tissue, distorts the anatomy of the lip, and obscures the vermilion border. Losses of <25% of the lip permit primary closure with little deformity; losses of >25% require a reconstructive procedure. Extensive lacerations directly through the commissure of the mouth also require surgical consultation in most cases. [93] Deep scars in the vermilion of the upper lip may produce a redundancy of tissue that requires later revision.

Large through-and-through lacerations of the lip should be closed in 3 layers. With a multilayer closure, the muscle layer is approximated with a 4-0 or 5-0 absorbable suture securely anchored in the fibrous tissue located anterior and posterior to the muscle. The vermilion-cutaneous junction of the lip is a critical landmark that, if divided, must be repositioned with precision; a 1-mm "step-off" is apparent and cosmetically unacceptable. The vermilion border should be approximated with a 5-0 or 6-0 nonabsorbable stay suture before any further closure to ensure proper alignment throughout the remainder of the repair (Fig. 37-55 A). The vermilion surface of the lip and the buccal mucosa are then closed with interrupted stitches using an absorbable 4-0 or 5-0 suture. Finally, the skin is closed with 6-0 nonabsorbable sutures. [95]

Small puncture-type lacerations heal well if only the skin is closed and the small intraoral laceration is left open. Such injuries are common from a punch in the face when the victim's tooth lacerates the lip. In general, small lacerations of the oral mucosa heal well without sutures. If a mucosal laceration creates a flap of tissue that falls between the occlusal surfaces of the teeth or if a laceration is extensive enough to trap food particles (e.g., 2 to 3 cm or greater in length), it should be closed. Small flaps may be excised. Closure is easily accomplished with 4-0 Dexon or Vicryl using a simple interrupted suturing technique. These materials are soft and less abrasive than gut sutures, which become hard and traumatize adjacent mucosa. Similarly, nylon sutures whose sharp ends are annoying and painful should be avoided inside the mouth. Muscle and mucosal layers should be closed separately. Sutures in the oral cavity easily become untied by the constant motion of the tongue. Each suture should be tied with at least 4 square knots. These sutures need not be removed; they either loosen and fall out within 1 week or are rapidly absorbed.

All lacerations that penetrate the oral mucosa should be evaluated for the presence of a fragment of tooth. A retained tooth fragment should be searched for in the depths of the wound if a tooth is missing or chipped. The search should be intensified if the patient returns with an infection of a sutured wound (Fig. 37-55 B). Probing the wound with forceps may identify fragments not seen directly in the wound. In the setting of marked facial swelling, a radiograph of the soft tissue may help identify an embedded tooth fragment.

**Tongue Lacerations**

There is some controversy regarding when to suture tongue lacerations. Surprisingly

large (simple or linear) lacerations, especially those in the central portion of the tongue, heal quickly with minimal risk of infection (Fig. 37-56) . Most tongue lacerations that occur from falls or seizures do not require sutures. The majority of tongue lacerations in children heal well without sutures. Snyder suggests that only those lacerations that involve the edge or pass completely through the tongue, flap lacerations, and lacerations that continue to bleed excessively need to be sutured. All lacerations bisecting the tongue require repair. [94] Small flaps on the edge of the tongue may be excised, but large flaps should be sutured. When dilute peroxide mouthrinses and a soft diet are used for a few days, healing is rapid. Persistent bleeding from minor lacerations brings most patients to the hospital, and closure may be necessary to prevent further bleeding.

The repair of a tongue laceration in any patient is somewhat difficult, but in an uncooperative child, the procedure may prove impossible under anything other than general anesthesia. A Denhardt-Dingman side mouth gag aids in keeping the patient's mouth open. A localized area of the tongue may be anesthetized topically by covering the area with 4% lidocaine-soaked gauze for 5 minutes; the maximum safe dose of local anesthesia should be determined and exposure to greater doses avoided. Large lacerations require infiltration anesthesia (1% lidocaine with buffered epinephrine) or a lingual nerve block. If the tip of the tongue has been anesthetized, a towel clip or suture can be used to secure the tongue. Further anesthesia and subsequent wound cleansing and closure are possible while an assistant applies gentle traction to the tongue.

Size 4-0 absorbable sutures should be used to close all 3 layers--inferior mucosa, muscle, and superior mucosa--in a single stitch, or the stitch should include one half of the thickness of the tongue, with sutures placed on the superior and inferior surfaces as well as on the edge of the tongue. [94] Sutures on the tongue frequently become untied. This problem can be avoided if the stitches are buried. Do not use nylon sutures in the tongue, because the sharp edges are quite uncomfortable. [72] Closure of the lingual muscle layer is usually sufficient to control bleeding and return motor function to the lacerated tongue. Mucosal healing is rapid, and closure of the muscle layer with only a deep absorbable suture may be desirable when a surface suture is likely to be tugged at, as occurs with small children.

**Scalp**

The scalp extends from the supraorbital ridges anteriorly to the external occipital protuberances posteriorly and blends with temporalis fascia laterally. There are 5 anatomic layers of the scalp: skin, superficial fascia, galea aponeurotica, subaponeurotic areolar connective tissue, and periosteum (see Fig. 37-17 *A*). Surgically, the scalp may be divided into 3 distinct layers. The outer layer consists of the skin, superficial fascia, and galea (the aponeurosis of the frontalis and occipitalis muscles). These 3 layers are firmly adherent and surgically are considered as 1 layer. The integrity of the outer layers is maintained by inelastic, tough, fibrous septa, which keep wounds from gaping open unless all 3 layers have been traversed. Wounds that gape open signify a laceration extending beneath the galea layer. The galea itself is loosely adherent to the periosteum by means of the slack areolar tissue of the subaponeurotic layer. The periosteum covers the skull. *The periosteum is often mistakenly identified as*

*the galea*, and vain attempts are made to suture the flimsy periosteum in the hope of "closing the galea" (Fig. 37-57) . [97]

Several unique problems are associated with wounds of the scalp. The presence of a rich vascular network in the superficial fascia results in profuse bleeding from scalp wounds. Severed scalp vessels tend to remain patent, because the fibrous SQ fascia hinders the normal retraction of blood vessels that have been cut, allowing persistent or massive hemorrhage in simple lacerations (Fig. 37-58) . The subgaleal layer of loose connective tissue contains "emissary veins" that drain through diploic vessels of the skull into the venous sinuses of the cranial hemispheres. In scalp wounds that penetrate this layer, bacteria may be carried by these vessels to the meninges and the intracranial sinuses. Thus, a scalp wound infection can result in osteomyelitis, meningitis, or brain abscess. [94] Careful approximation of galeal lacerations not only ensures control of bleeding, but also protects against the spread of infection (Fig. 37-59) .

Shear-type injuries can cause extensive separation of the superficial layers from the galeal layer (Fig. 37-60) . Debris and other contaminants can be deposited several centimeters from the visible laceration. Careful exploration and cleaning of scalp wounds are of obvious importance.

Because the scalp is very vulnerable to blunt trauma and because its superficial fascial layer is inelastic and firmly adherent to the skin, stellate lacerations are common in this region. Stellate lacerations not only pose additional technical problems in closure, but also have a greater propensity for infection. Multiple scalp wounds that are hidden by a mat of hair are easily overlooked.

When scalp wounds are debrided, obviously devitalized tissue should be removed, but debridement should be conservative, because closure of large defects is difficult on the scalp. When facing profuse bleeding, especially from extensive lacerations, the physician should instruct an assistant to maintain compression around the wound during the closure rather than try to tie off bleeding vessels. Unless the vessels are large or few, ligation of individual scalp vessels seldom provides effective hemostasis, and considerable blood loss can occur during the attempt. Bleeding from scalp lacerations is best controlled by expeditious suturing. [89] A simple procedure that often provides hemostasis of scalp wounds is

**Figure 37-61** *A,* To achieve hemostasis of a scalp laceration, a wide, tight, sterilized rubber band or Penrose drain may be placed around the forehead and occiput. This compresses the arterial supply to the scalp. *B,* Raney scalp clips and instrument for application to scalp wound edges.

placing a *wide*, tight rubber band around the scalp, from forehead to occiput (Fig. 37-61 *A*). Sterile rubber bands may be kept on the suture cart for this purpose. The clinician also may control bleeding temporarily in some cases by grasping the galea and the dermis with a hemostat and everting the instrument over the skin edge. The disadvantage of this technique is that tissue grasped by the hemostat may be crushed and devitalized, [89] and if the SQ tissue also is everted for a prolonged period, necrosis

can occur.

If an assistant is not available to apply direct pressure, local anesthetics containing epinephrine are sometimes effective in controlling the persistent bleeding from small vessels in scalp wounds. If bleeding from the edge of the scalp wound is vigorous, and definitive repair must be postponed while the patient is resuscitated, Raney scalp clips can be applied quickly to the edge of the scalp wound to control the hemorrhage. The applicator is loaded by inserting the tip of the instrument into the back of the clip and then locking the handles. The clip is slid onto the bleeding wound edge and released from the applicator. When the wound is repaired at a later time, the clip is removed by reversing the procedure. The plastic clips are radiolucent and do not interfere with plain radiography or computed tomography scanning (Fig. 37-61 *B*). [98] [99]

Before wound closure, the underlying skull should be visually examined and palpated in an attempt to detect fractures. More small skull fractures are detected with the physician's eyes and gloved finger than with radiographs. A common error is to mistake a rent in the galea or the periosteum for a fracture during palpation inside the wound. Direct visualization of the area should resolve the issue. In wounds that expose bone but do not penetrate the skull, prolonged exposure may leave a nidus of dead bone that may develop osteomyelitis. Exposed bone that is visibly necrosed should be removed with rongeurs until active bleeding appears. [89] Hair surrounding the scalp wound usually must be clipped far enough from the wound edge so that suturing can proceed without entangling the hair in knots or embedding hair within the wound. If hairs along the wound edges become embedded in the wound, they will stimulate excessive granulation tissue and delay healing. [100] Vaseline or tape may be placed on stubborn hairs that persistently fall into the wound. Although clipping scalp hair is not popular with some patients, failure to expose an area adequately is a common cause of improper preparation and closure of scalp wounds.

Unlike most wounds involving multiple layers of tissue, scalp wounds should be closed with a single layer of sutures *that incorporate skin, SQ fascia, and the galea* (Fig. 37-62). The periosteum need not be sutured. To minimize the chance of infection, SQ deep sutures generally are avoided. In superficial wounds, skin and SQ tissue should be approximated with simple interrupted or vertical mattress stitches using a nonabsorbable 3-0 nylon or polypropylene suture on a large needle. Smaller suture material tends to break while firm knots are being tied and should not be used. The ends of the tied scalp sutures should be left at least 2 cm long to facilitate subsequent suture removal. The use of blue nylon, as opposed to black, may make suture removal easier. If the galea is also torn, it should be included in the skin stitch. [101] Some investigators recommend a separate closure of the galea with an absorbable 3-0 or 4-0 suture, using an inverted stitch that "buries" the knot beneath the galea. [89] Separate closure of galea is not required in the vast majority of scalp lacerations sutured in the emergency department. When performed, separate closure of the galea introduces additional suture material into the wound, but in extremely large wounds provides a more secure approximation of the galea than obtained with large needle single layer closure.

Stellate lacerations or crush lacerations may be excised to produce elliptical incisions if

the area involved is not extensive. With microvascular techniques, large sections of skin avulsed from the scalp can be reimplanted. The emergency physician should use the same techniques in salvaging avulsed scalp as are used for amputated extremities. [101] (See Chapter 50 for further discussion.)

It has been suggested that there is no absolute time interval between injury and closure that automatically precludes primary suturing of scalp lacerations. Because of the extensive collateral blood supply of the scalp, most lacerations in this area heal without problems. Nonetheless, wound care must be thorough if the devastating complication of scalp infection is to be avoided.

Sutured scalp lacerations need not be bandaged, and patients can wash their hair in 24 hours. If bleeding is persistent, an elastic bandage can be used as a compression dressing. Gauze sponges are placed over the laceration to provide direct local pressure beneath the elastic bandage.

**Figure 37-63** Anatomy of the fingernail. The fingernail rests on the nailbed, also termed the *matrix.* The distal nail covers the sterile matrix; the proximal nail arises from and covers the germinal matrix. The tissue adherent to the proximal dorsal nail is the eponychium (also termed the *cuticle*), and the potential space between the nail and the eponychium is the nail fold.

**Nail Lacerations**

Injuries to the nail and nailbed (also called the *nail matrix*) are common problems in emergency medicine, yet controversy exists over proper management (Fig. 37-63) . Sixty percent of patients with subungual hematomas that are greater than one half the size of the nailbed and with associated fractures of the distal phalanx have a nailbed laceration. [102] [103]

In the case of a simple subungual hematoma (even in the presence of a tuft fracture) in which the nail is firmly adherent and the disruption of the surrounding tissue is minimal, the nail *need not be routinely removed to search for nailbed lacerations* (Fig. 37-64) . Despite the presence of a nailbed laceration, a good result can be expected as long as the tissue is held in anatomic approximation by the intact fingernail. Nail trephination is discussed in Chapter 40 . If the nail is partly avulsed (especially at the base) or loose, or if there are deep lacerations that involve the nailbed, the nail should be lifted to assess and repair the nailbed (Fig. 37-65) . When the integrity of the fingernail is disrupted and the nailbed is not approximated anatomically, a rippled nail is believed more likely as the new nail grows over the rough, scarred surface (Fig. 37-66) .

If the nailbed is exposed and has been extensively lacerated or partially avulsed, it may be necessary to refer the patient to a hand surgeon who can raise a flap of tissue extending from the proximal nail fold, explore the wound for foreign bodies, and clean under the nailbed. A simple nailbed laceration should be approximated with 6-0 or 7-0 absorbable sutures (to obviate the need for suture removal), generally using loupe magnification and a finger tourniquet to maintain a bloodless field (Fig. 37-67) . The

exposed nailbed should be protected by reapplying the avulsed nail (best choice) or by applying a nonadherent dressing or Silastic sheet for approximately 3 weeks. After cleaning, the avulsed nail may be sutured in place or secured with wound closure tape.

The replaced nail serves three purposes: (1) it acts as a splint or mold to maintain the normal anatomy of the nailbed, (2) it covers a sensitive area and facilitates dressing changes, and (3) it maintains the fold for new nail growth. Splinting should be maintained for 2 to 3 weeks. If longitudinal scar bands are formed between the proximal nail fold and the matrix, a permanently split or deformed nail may result.

A nail that is partially avulsed distally can be used as a temporary splint or "dressing" that protects and maintains the integrity of the underlying nailbed. When the base of the nail is avulsed from the germinal matrix, its replacement may occasionally result in infection. Some authors advocate trimming the proximal portion of the traumatized nail so that it can be placed more easily in the nail fold. [104] If the *germinal matrix* of the nail is avulsed intact, the nail should be reimplanted using a 5-0 or 6-0 absorbable suture in a mattress stitch (Fig. 37-68) (Figure Not Available) . [72] [105] If the root is not replaced, the space between the proximal nail fold and the nailbed is obliterated within a few days. [106] [107] If an open fracture exists, the matrix must not be allowed to remain trapped in the fracture line. [108] A replaced nail may grow normally, acting as a free graft, but often it is dislodged by a new nail. Nails grow at a rate of 0.1 mm/day, and it requires approximately 6 months for a new nail to reach to the finger tip.

If part of the nailbed has been lost, the patient should be referred to a surgical consultant for a matrix graft. Conservative therapy that allows large portions of an avulsed nailbed to granulate is inadvisable, although this is quite acceptable therapy for a fingertip avulsion that does not involve the nailbed. If the exposed nailbed is left open to granulate, it will heal with scar tissue and could produce a distorted and sensitive digit.

Wounds should be rechecked in 3 to 5 days following repair. At that time the nail fold may be repacked if nonadherent material was used, and the wound is assessed for infection. The use of absorbable suture for nailbed repair makes suture removal unnecessary. Tape or sutures are removed from any replaced nail in 2 weeks, and the old nail is allowed to fall off as the new nail grows. The value of antibiotics is unproven. All patients with nail injuries should be advised of a possible cosmetic defect in the new nail.

When repairing distal digit lacerations involving a nail, the physician should first approximate the onychial fold (Fig. 37-69) (Figure Not Available) . A sturdy needle attached to a 4-0 thread is recommended for suturing lacerated nails. Needles seem to penetrate nails with the least difficulty when they enter at 90°. The point of the needle carves a rigid path through the nail. Unless the entire length of the needle is allowed to follow this path as it passes through the nail, the needle is likely to bend or break. Alternatively, an electrical cautery instrument or a heated paper clip can be used to perforate the nail, thus permitting easy passage of the needle. The method for atraumatically removing a nail is demonstrated in Fig. 37-70 .

**Drains in Sutured Wounds**

Drains do not prevent infection; they primarily keep wounds open to encourage drainage of purulence or blood that may otherwise collect in the wound. When no infection exists and drains are used in soft tissue wounds "prophylactically," they are more harmful than beneficial. Edlich and coworkers state that "drains act as retrograde conduits through which skin contaminants gain entrance into the wound. Furthermore, the presence of a drain impairs the resistance of the tissue to infection." [63] Magee and colleagues found that drains placed in experimental wounds contaminated with subinfective doses of bacteria greatly enhanced the rate of infection, whether the drain was placed entirely within the wound or was brought out through the wound. [110] Drains behave as foreign bodies, provoking rather than preventing infection. If the wound is considered at high risk for infection, instead of suturing the wound with a drain in place (in anticipation of disaster), the physician should leave the wound open and consider delayed primary closure later when the risk of infection is minimal. Furthermore, drains should not serve as substitutes for other methods of achieving hemostasis in traumatic wounds.

## SUMMARY

A variety of techniques are available for reapproximating wound edges. Stapling is fast, but this technique does not allow meticulous control of wound edges, as may be necessary for a cosmetically appealing repair. Tape and tissue adhesive are the quickest and least painful methods of wound closure. Both eliminate the risk of self-injury with suture needles. These techniques can be used only on small superficial wounds or after approximation of the SQ layer.

The traditional and most commonly used method of closure is suturing. Stitches provide the most secure closure initially, but placement of sutures is time consuming and technically more difficult than other methods. All suture materials provoke inflammation and increase the risk of infection. Suture repair is the most appropriate method for wounds with complex configurations, those that extend into SQ tissue, and those in mobile areas.

At the conclusion of any wound repair, dried blood on the skin surface should be wiped away gently with moistened gauze, and the wound should be covered with a dressing. Dressing techniques, wound care instructions, and suture removal skills are discussed in Chapter 36 .

# Chapter 38 - Skin Grafting in the Outpatient

**Jose Eric Diaz Alcala**

Epithelial grafting is best defined as detaching a portion of epithelium from its original blood supply and placing it in a new area where the recipient site blood supply nourishes the grafted tissue. Free epithelial grafts allow one to close a clean wound that cannot be closed primarily because of an insufficient epithelial cover. The wound could be traumatic or secondary to surgical excision or tissue loss caused by a burn injury or chronic nonhealing ulcer.

The epithelium covers the internal and external surfaces of the body. The internal epithelium is mucosa, and the external epithelium is skin. Both can be used as free tissue transplants, but only skin grafting is discussed in this chapter. The outer covering of the body is a natural barrier between the body and the environment. Early coverage of an open wound and reestablishment of this barrier function are essential in the restoration of the internal equilibrium and the prevention of further wound complications. Epithelial transplantation serves many purposes, including enhanced cosmesis, reduced scar contracture, improved function, accelerated wound healing, and reduced fluid loss.

Epithelial transplantation originated approximately 3000 years ago. Currently, free tissue transplant has evolved from a reconstructive option of last resort to one that is routinely and sometimes preferentially used during soft tissue reconstruction. Although uncommonly performed in the emergency department, minor skin grafting can be done easily in the outpatient setting with minimal equipment. Results are excellent with knowledge of the physiology of skin grafting and when careful attention is given to proper site preparation for both recipient and donor sites; appropriate selection of the type of skin graft; precise techniques for harvesting and insetting the graft; proper dressing techniques; and adequate postoperative care.

While many emergency physicians would not consider skin grafting because of time constraints, lack of familiarity with the procedures, or local protocols, in the proper circumstances the procedure is readily adaptable to emergency medical practice. In reality, minor skin grafting is not as difficult or exotic as once promulgated and is well within the expertise of the emergency physician. Obviously one would not consider grafting of facial or other exquisitely cosmetic or functionally complex areas in the emergency department; however, uncomplicated wounds, usually of the extremities, represent candidates for emergency department skin grafting (Fig. 38-1) .

## TYPES OF SKIN GRAFTS

The skin is composed of the epidermis and the dermis resting on the subcutaneous tissues. The epidermis is composed of squamous epithelial cells in progressive stages of differentiation. The epidermis lacks a direct blood supply and receives its nutrients by diffusion from the dermis. A vascular plexus and many of the skin appendages, such as thermoregulatory sweat glands, sebaceous glands, and hair follicles, are located within the dermis (Fig. 38-2) . Free epithelial grafts can be classified according to site of origin

(skin or mucosa). Graft thickness is classified as follows: thin skin, full-thickness, split-thickness, dermal, and composite.

A *thin skin graft* consists only of epidermis. It is not routinely used and therefore will not be discussed here. A *full-thickness skin graft* contains the entire thickness of the epidermis and the dermis, including the adnexal structures such as hair follicles, but not subcutaneous tissue. It is most useful in repairing defects of limited size in cosmetically or functionally important sites and provides better wear and acceptable color and texture match. Other than for occasional use in resurfacing of the palmar aspect of the hand and the fingers, a full-thickness skin graft is rarely indicated in emergency situations.

A *split-thickness skin graft* includes the full thickness of the epidermis and a partial thickness of the dermis. Depending on the thickness, these grafts can be further subdivided into thin (0.005 to 0.012 in.), intermediate (0.013 to 0.018 in.), or thick (0.019 to 0.028 in.). The thinner grafts have the advantage of requiring less revascularization and consequently have a more rapid and complete adherence, thus improving the chance for graft survival. An intermediate split-thickness skin graft is preferred, because it may offer both improved graft survival and durability.

A *dermal graft,* which includes only the dermis, is a collagen-rich layer. It is used either as a buried reinforcing layer for protection of other structures or as an exposed epithelial graft for resurfacing in reconstructive surgery. It is harvested after cautious elevation of the overlying epidermal layer. However, the high incidence of defect during harvesting, the variability of graft resorption, and the availability of more predictable alloplastic implants have diminished the use of dermal grafts.

A *composite graft* is composed of at least 2 different tissue types or several tissue layers transplanted as a unit. Most commonly, this graft is composed of skin and cartilage and is used for facial reconstructive surgery. Dermal and composite grafts are not indicated in the emergency department, and further discussion is beyond the scope of this chapter.

Graft thickness can be judged by the appearance of the graft and donor site. Thicker grafts are more opaque. Immediately after elevation the edges of the thicker graft tend to curl up more toward the raw surface as a result of shortening of the elastic fibers in the skin. The donor site for a thin split-thickness skin graft shows a velvety field of numerous very fine bleeding points (Fig. 38-1 *B*). The bleeding pattern of a thick split-thickness skin graft donor site is much coarser. The punctuate bleeding points are larger in size and fewer in number.

Once skin grafts are harvested, they tend to shrink quickly as a result of the inherent elasticity of the dermal element, a process known as *primary contraction*. The thicker the graft, the greater the potential for early shrinking. A thin split-thickness skin graft barely shows evidence of early shrinking, whereas the full-thickness skin graft shrinks markedly. As skin grafts heal and undergo further maturation, progressive contraction of the graft-recipient complex occurs, a process known as *secondary contraction*. The recipient bed, not the skin graft itself, is usually the site of the most significant secondary contraction. The degree of delayed contraction depends on the thickness of the

transplanted graft. Thin split-thickness skin grafts contract the most, whereas full-thickness skin grafts show minimal evidence of delayed contraction. In a split-thickness skin graft, the contrast between the graft and its surrounding skin can create a "tire patch" appearance that is somewhat more pronounced than that seen in full-thickness skin grafts. This secondary contraction phenomenon usually starts at approximately 10 days after grafting and may continue for up to 6 months.

Skin grafts acquire pigmentation as they mature. The degree of pigmentation is also dependent on the thickness of the skin graft. A thin split-thickness skin graft is more apt to develop a dark pigmentation than is a thicker split-thickness skin graft or a full-thickness skin graft. The hyperpigmentation is thought to be secondary to stimulation of melanocytes by hormones and ultraviolet ray exposure. The problem of pigmentation is most pronounced in skin grafts on sun-exposed areas of the body.

## SKIN GRAFT HEALING

Graft healing proceeds through a series of unique stages that are not well appreciated in sutured wounds. Initially a skin graft is anchored on a temporary basis to the recipient bed by a *fibrin network glue* from the recipient bed. For the first 48 hours, survival of the skin graft is largely dependent on *serum* or *plasmatic imbibition*. The endothelial channels of the skin graft become filled with serum-like transudate fluid from the recipient bed. This is likely due to capillary action and diffusion within the skin graft. The nutritional supply and metabolic exchange are mediated through this fluid medium. This process prevents the graft from drying and serves to keep the vessels open for later communication with the recipient vessels. [1] [2] [3]

During this early phase of serum imbibition, restoration of circulation proceeds concurrently, as seen by a gradual change of the color of the graft from pale white to pink. Concurrently the fibrin glue is replaced by granulation tissue, which provides permanent adherence of the graft to the recipient bed. Vascular anastomoses that arise from the vascular-rich recipient bed allow percolation of blood through the preexisting graft vasculature. In ideal circumstances this collateral circulation bridges a gap of 0.5 cm from any wound margin. The bridged area will require more time to regain full vascularity.

Normal circulation is restored in the skin graft in 4 to 7 days. Epidermal proliferation and hyperplasia are seen in 4 to 8 post-transplant days and persist for several weeks. Restoration of the lymphatic circulation parallels reestablishment of the blood supply. Graft reinnervation begins as early as 2 to 4 weeks after transplantation, but full sensation may not occur for several months to years later. However, in partial thickness grafts, specialized dermal adnexa structures, such as those responsible for hair growth or sweating, will not be evident upon healing. Rapid gain in adherent strength in most skin grafts occurs within the first 8 hours after grafting and continues to gain slowly with each subsequent 24-hour period.

The viability of a thin split-thickness skin graft can be maintained longer by imbibition alone than can a thick split-thickness or a full-thickness graft. This is attributed to the shorter distance for diffusion in thinner grafts and the fact that there is less cellular mass

to be nourished. Revascularization of a thin split-thickness skin graft is also more rapid than revascularization of a thick split-thickness graft or a full-thickness graft for the same reason.

## DONOR SITE HEALING

The surface of the split-thickness graft donor site is a raw dermal surface with multiple openings into the remaining portions of the transected skin appendages, such as sweat ducts and glands, sebaceous glands, and hair shafts and follicles. These appendages are lined with squamous epithelium and are the source of epithelial cells for resurfacing of the exposed dermal surface. The epithelial cells proliferate, migrate out to the dermal surface, and spread radially until they become confluent and cover the entire raw surface. The dermal layer itself may become thicker with scar tissue but does not regenerate. The greater the thickness of the split-thickness skin graft, the fewer the number of skin appendages left in the donor surface. For this reason, the donor site of a thick split-thickness graft takes longer to heal than the donor site of a thinner split-thickness graft. Generally, the donor area is treated as an open wound and is allowed to reepithelialize from its margins and form its dermal rest. Depending on the thickness of the split-thickness graft, the donor site is usually healed within 7 to 56 days (thin, 7 to 10 days; intermediate, 10 to 21 days; thick, 21 to 56 days).

Once the donor site is reepithelialized, its appearance is largely dependent on the graft thickness. A thin split-thickness skin graft donor site containing a thicker residual dermis will have an appearance closer to that of its surroundings and will be less conspicuous than a thicker split-thickness graft donor site. For the most part, the final appearance of both the recipient and the donor site reflects the amount of dermis that has been transferred and the amount of dermis that is left behind. However, *the donor site of full-thickness skin graft should not be allowed to heal by secondary intention.* The removal of the dermis prevents epidermal regeneration, resulting in significant scar contracture. The donor site of

full-thickness skin graft must be either closed primarily or covered by a split-thickness skin graft.

## UNFAVORABLE FACTORS

Many clinicians are overly concerned with skin graft aesthetics (e.g., size and shape) and are less concerned with the more important final outcome. Even partial success is better than the alternative of nongrafting for the majority of candidate wounds. Several factors adversely influence the outcome of skin grafting by either interfering with the revascularization process or disturbing the neovascular network that has already been formed; these include hematoma and seroma, graft movement, necrosis, and infection. [4] Viability of a salient thickness graft can be best enhanced by preventing unfavorable conditions, especially minimizing the buildup of fluid between the graft and recipient site and movement of the grafted tissue. In the emergency department, attention to these factors is paramount.

**Hematoma and seroma**

Hematoma formation between the graft and recipient site is the most common cause of skin graft failure. A hematoma under the skin graft literally causes separation of the skin graft from its nutrient bed. Consequently, revascularization of the graft is delayed or altogether prevented by the space-occupying effect of this intervening layer of blood or blood clot. The skin graft may survive for a short period on serum imbibition alone. If revascularization is delayed beyond this period of serum imbibition, however, the graft is doomed to fail. To prevent hematoma, one should obtain complete hemostasis of the graft bed prior to application of a skin graft. If complete hemostasis is not possible, the wound should be dressed, and skin grafting should be delayed until there is no further hemorrhage. One loses little clinical advantage by waiting a few days to perform the graft until conditions of the recipient site are more favorable. A seroma also prevents graft adherence and revascularization through a mechanism similar to that of a hematoma.

**Movement**

Graft survival depends on reconstitution of the capillary vascular network between the recipient bed and the skin graft. Even minimal motion of the skin graft relative to the recipient bed could disrupt and interfere with the formation of these fine, early vascular connections. Movement also promotes formation of a seroma or hematoma. For these reasons, immobilization of the graft to its bed is essential in graft healing. Immobilization is enhanced by suturing; stent or pressure dressings; and bandaging and splinting techniques, discussed later.

**Necrosis**

Any residual nonviable tissue left in the recipient bed will undergo necrosis and will lead to failure of the overlying graft. In particular, the vascularity of fatty tissue in a traumatic wound is often uncertain. Bits of fat may die and necrose, and therefore, it is important to debride all tissues of questionable vascularity before grafting. If the vascularity of the recipient bed is uncertain, it is best to delay skin grafting. Likewise, it is prudent to carefully debride or defat the undersurface of the graft prior to placement. Minor necrosis of portions of the graft, such as the edges, are not disastrous, and do not necessarily predict failure of the entire graft. Often even only a partial take is better than the alternative of relegating total healing to secondary intention without grafting.

**Infection**

Despite its devastating effect on skin grafting, infection is the least common cause of graft failure. In addition to the obvious space-occupying effect of infection, a purulent collection actively separates the graft from its bed and destroys the newly formed vessels. The abundant proteolytic enzymes from inflammation and from the microorganisms are responsible for the lysis of the fibrin adhesion and the destruction of vascular connections. The most notable virulent organism that affects skin grafting is

group A beta-hemolytic streptococcus *(Streptococcus pyogenes),* which rapidly destroys the skin graft and literally melts it away. Topical or systemic antibiotics effective against common microorganisms (i.e., staphylococcal, streptococcal, or *Pseudomonas* organisms) may be helpful in selective situations if administered early on, particularly for patients with diabetes or impaired immunosuppression or after a prolonged intraoperative time. However, prophylactic antibiotics need not be administered routinely in immunocompetent individuals and do little to guarantee ultimate graft survival in questionable cases.

## INDICATIONS AND CONTRAINDICATIONS FOR SKIN GRAFTING

Whether to graft a wound or to leave it open to heal by secondary intention depends on the nature and history of formation of the wound. Traumatic open wounds may be classified as either clean or contaminated. The majority of traumatic wounds can be considered clean, except for those resulting from bites, those made in a grossly contaminated environment, and those that are 24 hours old. Of particular concern is wound contamination by microorganisms. Wounds that contain inorganic material, such as gravel, glass, metal, industrial lubricants, and the like, are not necessarily extensively contaminated. As long as there is no suspicion of major contamination by microorganisms, these wounds can be made clean by debridement and removal of foreign substances. Untidy wounds resulting from tearing, crushing, or mangling or explosive injuries with devitalized tissue debris can also be debrided and converted to clean, tidy wounds. In general, all clean wounds should be closed when possible, and contaminated wounds should be left open (see Chapter 36) .

A skin graft should be contemplated only when primary wound closure or flap reconstruction is impractical or contraindicated. Grafts are more likely to fail than flaps. In general, grafting is the best possible dressing material for an open wound when an increased risk for additional cosmetic deformity is not avoided with primary closure or flap repair. Another common indication for grafting is a wound that is healing too slowly by granulation. In many cases grafting can be a life-saving procedure, such as with burn care.

When there is full-thickness skin loss of a small area and the wound cannot be closed with local tissue alone, closure with a distant tissue is considered. In these circumstances a full-thickness skin graft may be indicated. To prevent failure in revascularization, a full-thickness skin graft should not exceed 2 cm at its largest cross-sectional width, since its circulation relies on invasion of vessels from the periphery. Relatively superficial and large wounds are most often closed with split-thickness skin grafts. For those wounds in which graft color, texture match, and graft contraction

are not of concern, a split-thickness skin graft is an ideal wound cover. In fact, a split-thickness skin graft is often used because of this propensity to undergo secondary contraction. For instance, a split-thickness skin graft on a fingertip wound would contract, pulling the surrounding healthy pulp pad skin over the tissue defect, thereby minimizing the size of the wound. On the other hand, certain anatomic areas, such as the face and the flexion surfaces of the hand and fingers, require coverage that

produces minimal secondary contraction. Facial wounds also demand color and texture match, which are best met by full-thickness skin grafts. If deep structures, such as neurovascular bundle, tendon, bone, and joint, are exposed, some other means of distant tissue cover in addition to skin grafting may be considered.

Skin grafting of selected acute wounds in an outpatient setting is advantageous. Grafting affords early closure of the wound and obviates further wound complications, such as desiccation of exposed structures, repeated trauma to tissue, and infection. Grafting may considerably decrease the degree and the length of the convalescence period. For example, when grafted, a fingertip wound >1 cm2 would heal much sooner than a wound of equal size that is allowed to close by contraction and epithelialization. Grafted wounds are also more comfortable for the patient than open wounds and may allow an earlier return to work.

## RECIPIENT BED

One can best ensure that the graft will "take" by carefully preparing the recipient bed and keeping it clean and relatively dry at all times. All the nonviable tissue and granulation tissue must be removed surgically from the wound, creating a well-vascularized surface without barriers to graft adherence.

Meticulous hemostasis of the recipient bed is also a key to a successful graft. A common mistake is to attempt grafting immediately after wound cleaning/debridement when the site is still actively oozing. Often merely waiting 15 to 20 minutes for normal clotting to occur will create a dry surface. Gentle pressure applied with a warm, saline-soaked gauze is often all that is required. In an extremity, a tourniquet may be used to provide hemostasis, but the graft should be sutured into place and a compression dressing applied before the tourniquet is removed. Judicious use of topical vasoconstrictors and cautery may help if active bleeding persists.

Skin grafts can be located on any exposed vascular surface. This includes all musculoskeletal tissue and all internal organs. Skin grafts do not survive on nonvascular surfaces, such as bare bone without periosteum, bare cartilage without perichondrium, bare tendon without peritenon, and bare nerve without perineurium cover. However, a skin graft can serve as a bridge over a nonvascular area by vascularization through the graft edge, a process known as *bridging phenomenon.* Theoretically, a maximum of 10 mm of nonvascular area can be covered by bridging, provided that there is an adequate vascular rim of recipient bed at the perimeter of the skin graft.

## SELECTION OF THE DONOR SITE

In theory, a skin graft should be able to be taken from anywhere on the body. Indeed, numerous donor sites are necessary when large areas must be covered, as in a major burn injury. As a general rule, it is crucial to inspect all available donor sites, and it is best to use tissue as close as possible to the site of the defect, looking for the most appropriate texture-color match. In addition, proper selection of a well-vascularized donor site is important, because such skin becomes vascularized more rapidly once

transplanted. However, the donor tissue should not be obtained from areas that will be irritated when the patient sits or lies supine, and all feasible attempts to minimize scar formation upon healing should be pursued. In young women, it is preferred to take the graft from within the bikini line to conceal the donor site scar. Another concern in choosing the donor site is that someone may misinterpret the donor scar as a self-inflicted wound (i.e., wrist slashing).

For smaller wounds, the functional and cosmetic quality of the skin graft at the recipient site and the resulting donor site deformity dictate the selection of the donor site (see Fig. 38-2) . A full-thickness skin graft is usually taken from a glabrous area of the body. There are many favored areas from which to take full-thickness skin grafts (Fig. 38-3) . Among them, the hypothenar skin, antecubital flexion creases, the wrist flexion crease, the medial arm, and inguinal flexion creases are most readily accessible and appropriate for outpatient situations. For completeness, other donor sites for full-thickness skin grafts include the preauricular groove, postauricular groove, mentolabial groove, supraclavicular skin, and upper eyelid skin. These donor sites are mainly used for facial reconstruction surgery and are rarely indicated in the emergency department.

When a thin split-thickness skin graft is desired (as is the case for most emergency department skin grafting), the anteromedial aspect of the forearm is suitable (see Fig. 38-1 C). One objection to this site is obvious scarring in a frequently exposed area. However, in experienced hands a very thin split-thickness skin graft from this area leaves minimal to no appreciable scarring. The anterior, medial, and lateral aspects of the upper thighs, the inner aspect of the upper arms, the back, the buttocks, and the lower abdomen are also available for skin grafting. Relatively thick and large skin grafts can be taken safely from these areas. In addition, the instep of the plantar foot could be contemplated as an applicable donor site for split-thickness skin graft for selected cases of soft tissue defects of the palmar aspect of the hand that require either primary or secondary surgical reconstruction.

## HARVESTING EQUIPMENT (DERMATOMES)

A wide variety of instruments can be used to harvest the graft. These can be classified into freehand, drum-type, and powered dermatomes. For outpatient use in an emergency facility, freehand dermatomes are quite adequate to harvest a small skin graft. Only those that are useful for outpatient skin grafting are described here.

### Freehand Dermatomes

Freehand dermatomes include scalpels, razors, and knives. Even though acceptable small grafts can be obtained using these devices, they are often uneven in thickness and size and have irregular edges, despite the adjustable roller mechanism fitted in some of them. Undoubtedly, the final product depends on the physician's touch, experience, and sense of self-confidence.

A *scalpel blade* is the simplest dermatome. A No. 10 or 20 blade is quite effective for

taking a small split-thickness skin graft (Fig. 38-4 *A*). A sterile, regular single side of a *double-edged razor blade* can be used to take a small skin graft. The razor blade is held with a straight clamp and is used in a freehand manner (Fig. 38-4 *B* and *C*). Various thicknesses of split-thickness skin grafts can be cut with this dermatome. The thickness of the skin graft can be judged by the clarity with which the writing on the blade can be read through the skin graft. One can see through the usual thin and intermediate-thickness skin grafts quite readily.

## Silver Dermatome

This dermatome consists of a handle and a platform at the opposite end that holds a regular, double-edged razor blade. The razor blade is fixed in place with a cover plate, which is secured with a wing nut. There is a roller just above and parallel to the blade edge. The thickness of the graft can be adjusted roughly by setting the distance between the roller and the blade, which is accomplished by turning the knobs at the ends of the blade (Fig. 38-5) .

## Goulian-Weck Knife

The Goulian-Weck knife consists of a handle with a metal slot at one end that holds a safety razor blade 5.5 cm long. A blade guard is placed over this assembly (Fig. 38-6) . The blade guard determines the thickness of the skin graft. Three different blade guards with thicknesses of 0.008, 0.010, and 0.012 in. are available. Although these guards are made to cut skin grafts of fixed thicknesses, after repeated use they become sprung and are not very reliable. These blade guards should be used as rough guides to the thickness of the skin graft. The actual thickness should be controlled visually and by touch.

## Davol Disposable Dermatome

This dermatome is a sterile, disposable blade unit that cuts a split-thickness skin graft of intermediate thickness and 3.5 cm in width (Davol Inc., Providence, RI). This blade unit is driven by a battery-operated motor in an electric toothbrush handle unit (Fig. 38-7) . The handle is placed in a sterile plastic bag. The blade unit is then pushed onto the handle, puncturing the plastic bag, and is attached to the handle. The device is operated by pressure placed on the button switch while the blade unit is kept lightly pressed against the skin with an equally light forward force to advance the dermatome. The battery in the handle is rechargeable through a recharging unit.

# HARVESTING THE SKIN GRAFT

After meticulous preoperative evaluation has been rendered, the defect should be measured and the surrounding tissue shaved if needed. Measurements should not be done by estimation or "eye-balling"; rather, the recipient and donor sites should be carefully plotted out using templates and marking pens. Ideally a material that is flexible, transparent, not stretchable, and able to be sterilized and that can conform to the defect is the best for making a pattern or template (i.e., sterile glove, paper, aluminum foil,

gauze or Telfa dressing).

Once the donor site is outlined, local anesthesia is administered, thereby avoiding the inexact sizing caused by tissue stretch resulting from the infiltration. The donor site is scrubbed with any of a number of common antiseptic solutions. The area should be rinsed with saline and draped with sterile towels. Some physicians advocate wiping the area with acetone or ether to remove oil from the skin surface prior to donor site anesthetization. Local anesthetic (0.5% or 1% lidocaine [Xylocaine] and 1:100,000 or 1:200,000 epinephrine solution) is used for anesthesia and hemostasis. Epinephrine in the anesthetic will not compromise the procedure. An area larger than the size of the desired graft is locally infiltrated. A plane of anesthetic infiltration is placed in the deep dermis, allowing the surgical separation of the dermis from the underlying fat. The anesthetic solution is injected continuously while the needle is being passed back and forth in multiple parallel passes. A 25- or 27-ga hypodermic needle 3.8 cm (1.5 in.) long is used. This results in uniform infiltration of the entire area, forming an evenly elevated plateau and facilitating the harvesting of the skin (Fig. 38-8) .

**Full-Thickness Skin Graft**

One should plan carefully to orient the long axis of the true template parallel to the adjacent skin creases. This allows primary closure of the donor defect and produces favorable orientation of the resulting scar. Using a scalpel, a skin incision is made along the lenticular pattern (Fig. 38-9) . The graft should be 3% to 5% greater in size than the true template to compensate for natural graft shrinking and contraction after its removal from the donor site. The full

**Figure 38-8** Infiltration of anesthetic in the donor site.

thickness of the skin is removed by running the blade of the scalpel along the junction between the dermis and the subcutaneous fat. *Fat is then removed from the graft by stretching it over a finger with the epithelial side down and snipping the bits of fat from the dermal surface with a pair of small, curved sharp scissors*. Defatting is completed when all the yellow globular material is gone and the white, glistening surface of the dermis remains. The graft pattern is cut, and the skin graft is then ready for placement. In the meantime, it can be placed between layers of saline-moistened gauze, where it will remain viable for approximately 1 to 2 hours until the recipient site is prepared for transplantation. The graft may be used up to 21 days later if properly stored (i.e., refrigerated or kept on ice at 4 °C and with antibiotic solution [e.g., Hank's tissue culture solution], if warranted).

Often the patient presents to the emergency department with a portion of skin or an amputated fingertip that has been removed in a slicing injury. The amputated tip or skin, if relatively superficial, can be treated as a full-thickness skin graft. The tissue should be irrigated with saline to remove debris, defatted, and placed in saline-soaked gauze while the recipient bed is being prepared. The thick epidermis of the fingertip is often centrally cut 5 or 6 times with a scalpel to avoid separation of the graft and recipient bed as the fingertip dries. The graft is then attached by one of the techniques discussed in the

following sections.

## Split-Thickness Skin Graft

It is easiest and preferable to perform split-thickness skin grafting using one of the specialized cutting apparatuses previously described. [6] This can be done with a freehand device or with a powered dermatome, with or without a

**Figure 38-9** To harvest a full-thickness skin graft, the donor site is outlined with a marking pen. Note that a template of the area to be grafted has been placed over the donor site to ensure full coverage *(A)*. An elliptical site is removed so that the donor site can be closed primarily with sutures, leaving a linear scar. A minimal amount of subcutaneous skin is removed using a scalpel and forceps *(B)*. The graft is rolled back *(C)*. The underside of the graft is fully defatted with scissors *(D),* and the graft is placed in saline-soaked gauze, where it will remain viable for 1 to 2 hours.

predetermined depth to be harvested, depending on the instrument used. The skin graft donor site is first lubricated so that the dermatome can glide smoothly. This facilitates taking an even skin graft. Sterile substances such as mineral oil, petrolatum, saline, or any of the usual topical ointments can be used. The lubricant is applied thinly over the donor skin surface. The donor skin is stretched manually and placed under tension. The skin is held proximally and distally to the graft donor site by firmly pressing the skin with either a sterile wooden tongue blade or a piece of gauze. Traction is applied by pulling away from the donor site in both directions (Fig. 38-10 *A*). This maneuver usually requires 2 people. The operator's free hand should hold the traction toward the direction of the dermatome movement. An assistant applies countertraction, and the operator cuts the skin graft toward the free hand.

The dermatome is set for the desired thickness; it should be held lightly with the fingers and the thumb. During the procedure, the operator's upper extremity is relaxed, and the wrist is maintained in a relatively fixed position. The cutting motion is mainly at the elbow and the shoulder and consists of frequent to-and-fro strokes with minimal forward pressure. Too much forward pressure may result in irregular thickness of the graft and possible interruption of graft continuity. The dermatome should contact the skin at an angle when the cut is begun. Once the cut is initiated, the dermatome is flattened out to effect tangential excision of the skin (Fig. 38-10 *B*). The downward pressure on the dermatome determines the thickness of the skin graft. For thin and intermediate-thickness skin grafts, the weight of the dermatome itself, without much additional external pressure, is sufficient to cut the desired thickness. Tissue forceps or hemostats may be used to advance the graft into the dermatome and away from the blade. When the appropriate length of harvest tissue is reached, the dermatome is then tilted away from the skin, cutting the skin and completing the harvesting phase. At this time, the harvested graft can be washed in normal saline to remove any excess povidone-iodine or lubricant previously applied. Concurrently, the donor site should be temporarily covered with a pressure dressing.

Once harvested, the skin naturally curls and shrinks immediately. Uncurling the harvested graft by placing it on moist gauze until time for transplantation is helpful. A sheet of sterile semipermeable film (i.e. Op-Site, Tegaderm) may be applied to the

donor site prior to the harvesting phase. When removed in conjunction with the skin graft, it will prevent to some extent curling of the harvested graft because of its adhesive qualities. Of importance, its thickness (0.002 in) should be accounted for (i.e., added to the desired tissue depth) when setting the dermatome depth to ensure adequate tissue harvesting. In addition, when transferred to the recipient site, the semipermeable film can serve as a primary dressing for 1 to 2 weeks postoperatively. In split-thickness skin grafts, primary contraction of the harvested tissue is not as clinically significant as that in full-thickness

**Figure 38-10** *A,* The cutting of a split-thickness skin graft with a double-edged razor blade held in a straight clamp. The skin is held taut by traction and countertraction. *B,* A tangential excision producing a skin graft of uniform thickness.

skin grafts. Commonly, in split-thickness grafting there is no need for harvesting more tissue than is required.

# APPLICATION OF THE SKIN GRAFT

## Full-Thickness Skin Graft

Following meticulous hemostasis of the recipient bed, the previously cut skin graft is laid on the recipient bed at the desired position. At all times one should *avoid raising the graft from its bed once it has been applied.* If needed, the graft may be stretched or rolled to remove wrinkles, or trimmed to fit. As the graft is being sutured into place, a cotton applicator stick can be twisted underneath the graft to remove any remaining clots. The sutures used to approximate the skin graft to the margins of the wound are denoted as anchoring sutures, basting sutures, and circumferential sutures. Several *anchoring sutures* (4-0 polypropylene or nylon) are placed on the four quadrants of the graft for proper fitting. Alternatively, stainless steel staples provide rapid and precise edge eversion of both the graft and the recipient borders. Also, additional interrupted sutures can be placed between the primary anchoring sutures as needed. These sutures are left long (4 to 6 cm) for a tie-over dressing, although a tube gauze dressing is also effective for digits. *Basting sutures* (5-0 or 6-0 chromic gut) are necessary if the central portion of the graft requires more bed contact for additional graft stability and to prevent further hematoma formation. In some situations a properly applied basting suture may obviate the need for a sophisticated and time-consuming graft dressing. The remaining edges are sutured with a simple *running circumferential suture* (5-0 to 6-0 nylon or polypropylene) for further epidermal wound approximation. Sutures are easier to place if the needle is driven through the graft first at a slightly higher level in the dermis and then through the skin edge of the recipient area at a slightly deeper level in the dermis in an almost epicuticular fashion. This prevents tenting of the edges of the graft, thus enhancing contact for revascularization.

## Split-Thickness Skin Graft

The split-thickness skin graft is placed on the recipient bed with the dermal side down, similarly to a full-thickness skin graft. The dermal side of the split-thickness skin graft is

characterized by the wet, glistening sheen, as opposed to the relatively dry, dull appearance of the epithelial surface. If the recipient bed is larger than a single sheet of graft, several sheets of graft may be required. Any overlapping of skin grafts will not influence the graft take. On the other hand, it is important to make sure that the free edges of the graft are fully uncurled (i.e., the graft should *not* be doubled over on itself). Obviously, any curled portion will not get revascularized, and sectional failure of the graft will result.

The split-thickness skin graft should be laid in such a way that it follows all the "hills and valleys" of the wound and is in contact with the entire raw surface of the recipient bed. If the skin graft tends to tent over a deep concavity, it is useful to tack down the tented portion to the base of the concavity using 1 or 2 through-and-through stitches (5-0 or 6-0 nylon or polypropylene). This is supplemented by a conforming dressing. The overhanging edges of the skin graft beyond the wound margin are trimmed. Suturing of the skin graft at the edges is not always necessary but may be desirable to offset the curling of the skin edges. Often adhesive

closure tapes are used rather than sutures at the skin graft edge, with satisfactory results.

When there is a need for the skin graft to conform better to the recipient site surface owing to the site's irregularities, or when a larger surface area must be grafted from a smaller donor site, the necessity of meshing the graft obtained should be contemplated. This is accomplished by passing the skin through a mechanical device that produces multiple uniform slits in the graft. However, slight meshing of the graft with simple scalpel slits also can be done. When tension is applied perpendicular to these incisions, they open and increase the surface area that can be covered by the graft. Another relative indication for meshing the graft is to allow any blood or serous fluid to drain through the incisions without compromising graft vitality. These openings eventually epithelialize, but the aesthetic appearance is usually not as good as that achieved with an unmeshed graft. For the most part, the use of this method in the emergency department is rarely indicated, because it may implicate a complicated and larger wound that requires an in-patient approach.

## DRESSINGS

### Full-Thickness Skin Graft

A full-thickness or partial thickness skin graft is classically dressed with a tie-over bolster dressing. This dressing facilitates immobilization and affords moderate, even graft site pressure to prevent collection of blood or serum under the skin graft during the critical period of revascularization. Once the graft has been secured, the skin graft could be covered with a single layer of nonadhesive dressing. The nonadhesive dressing will minimize the adherence of the graft to the bulky pressure dressing that follows, which, at the time of dressing removal, tends to pull the graft away. It is followed by Xeroform, petrolatum, or antibiotic-petrolatum-impregnated fine gauze, which is cut larger than the grafted area. A bolster that fits the size of the graft is formed using a moist gauze followed by a dry wad of cotton, lamb's wool, gauze, or a polyurethane foam pad. The

overhanging edges of the nonadherent gauze dressing are swept up to cover the rough edges of the bolster. The long tails of the sutures are tied, and the bolster is secured in place providing molded constant pressure over the graft (Fig. 38-11) . An additional soft dressing can be applied to protect the bolster dressing as necessary. After this step, the graft is not disturbed until dressing removal 5 to 10 days later, depending on recipient site conditions. If motion of the graft bed is an anticipated problem, immobilization of the parts involved with a loose plaster cast, splint, or sling may be necessary.

**Split-Thickness Skin Graft**

There are numerous individual variations among physicians in dressing split-thickness skin grafts, but all approaches can be classified as closed or open. All closed techniques are all quite functional, as long as they meet several basic requirements. The skin graft dressing should be nonadherent, absorptive, immobilizing, and protective. A split-thickness skin graft on a concave surface can be effectively immobilized with a tie-over bolster technique, which has been described previously. For uncomplicated small split-thickness skin grafts, there are much simpler and easier methods of applying dressings. Ordinarily, open techniques of skin grafting dressing (e.g., a wire cage or a cardboard box placed over the graft) are not used routinely. They are not suitable for commonly encountered wounds in emergency situations and are not used in any patient whose willingness or ability to cooperate is in doubt.

A small and dry split-thickness skin graft on a flat surface can be secured with several strips of adherent porous adhesive closure strips without sutures (Fig. 38-12) . The strips can also be positioned across the wound margin in a radial fashion, like spokes on a wheel. The graft is then covered with nonadherent petrolatum gauze sufficient to cover the entire extent of the wound closure strips. If the strips are not covered entirely by the greasy dressing, they may get lifted off as the dressing is removed, which in turn may disrupt the skin graft. Next, a couple of layers of saline-moistened gauze are applied. This layer of dressing aids in absorption of the early drainage through capillary action of the moist gauze fibers. The entire area is then covered with an oversized, thin sponge with an adhesive backing, such as a Reston pad (3-M Company, St. Paul, Minn). The overhanging portion of the sponge is pressed and allowed to adhere to the surrounding skin, which has been wiped dry.

If an adhesive sponge dressing is not available, the last step can be modified as follows: A surgical adhesive such as tincture of benzoin or Aeroplast is applied to the skin surrounding the moist gauze dressing. An oversized dry gauze is applied over the entire area, and the edges are pressed to stick to the skin, which has been painted with surgical adhesive. The edges of the dressing can be reinforced with tapes if necessary. This last layer of dressing protects the graft from external shearing forces. The entire dressing and the entire graft-recipient unit may move, but the graft is not allowed to move relative to the recipient bed. This layer should not be occlusive, as such a layer (e.g., an adhesive plastic dressing) would prevent evaporation and would allow collection of drainage in the dressing and consequent maceration of the graft.

Contrary to common impression, a split-thickness skin graft on a fingertip wound is quite simple to dress. Fixing the skin graft with sutures is not always necessary; this extra

step is time consuming and does not add to the outcome of grafting. The skin graft is simply kept in place with several strips of petrolatum gauze that are placed across the skin graft and the adjacent skin in a crisscross fashion. The strips should be narrower than the thickness of the finger, so that the gauze strips can be molded to the contour of the fingertip without folding or pleating. A thin layer of moist dressing gauze cut to size is placed next. Two strips of adhesive sponge foam pad are laid across each other over the fingertip. Dry gauze strips can be used in place of the foam pad. The entire assembly of dressing is then covered with tube gauze, if available. No more than 2 layers of tube gauze should be used so that the dressing is not constricting.

The final, and often most critical, procedural step of skin graft dressing is immobilization of the graft site. External splinting is important for grafts that are on parts of the body that normally are in motion (e.g., the extremities [see Figure 38-1 *E*]). The best way to splint an extremity with a skin graft is with a plaster cast. Although a cast may appear to be too much for a fingertip graft, in the long run, patients are more comfortable and tolerate it well. Small individual finger

**Figure 38-11** A method of applying a tie-over bolster dressing to provide pressure to the graft site to minimize movement and fluid accumulation. While tying the bolster, the gauze is depressed with broad forceps or an assistant's finger.

splints that are taped on are not as reliable. These splints allow much motion and, hence, discomfort and graft disruption. With reinforcing strips of plaster in key areas, a relatively light cast that does the job can be fabricated.

The skin graft on the forearm can be splinted with a long arm cast that includes the wrist and the elbow. The wrist should be kept in 20° to 30° of extension and the elbow in 90° of flexion. A skin graft on a digit or the hand can be effectively immobilized with a short arm cast or bivalve plaster splint. Even if only a single digit is injured, it is advisable to immobilize the fingers in groups, such as the index and middle fingers or the ring and little fingers. The thumb is immobilized with a thumb spica cast. The hand and the fingers are kept in the functional position. All casts should be generously padded with cast padding. The plaster roll should be applied loosely and without constriction, since excessive pressure could cause vascular or neurologic damage and even loss of the extremity.

## CARE OF THE SKIN GRAFT

Postoperatively, skin grafts should be examined at frequent intervals. It is reasonable to check the grafted area within 24 to 48 hours for possible infection, seroma, or hematoma. If no complications are apparent or suspected, the graft should be reexamined within a week for the first dressing change and suture removal, if indicated. Early dressing change just for curiosity may damage the graft. Therefore, re-dressing the grafted area within the first 48 postoperative hours is a matter of judgment. The ideal graft is pinkish-red in appearance at early stages of healing. Often the graft may appear darker blue or purple, depending on the extent of revascularization, and will spontaneously revitalize. Certainly, a black graft is considered indicative of necrosis,

and aggressive therapy is justified.

Seromas and hematomas are to be avoided, and if they occur, they should be drained by a small stab incision made in the overlying skin graft with a No. 11 scalpel blade or a pair of scissors. The incision should be made directly over the center of the fluid collection. One then expresses the fluid by rolling 2 cotton swabs from the periphery to the center of the collection (Fig. 38-13) . Each seroma or hematoma should be drained individually; if more than 1 are drained through a single stab incision, the intervening portion of adherent skin graft may be lifted off the bed and disturbed. Following this, the skin graft is redressed and examined every 1 to 2 days and is drained if necessary until the graft is fully taken. If there is gross infection, the involved portion of the skin graft is debrided and lavaged, and appropriate dressing change is initiated.

Dressings on small split-thickness skin grafts (e.g., fingertip grafts) and tie-over bolster dressings on full-thickness skin grafts are left undisturbed for 5 to 7 days. Nonetheless, the patient should be seen 2 to 3 days following the operation, and the area of the graft dressing should be examined for signs of infection. Signs and symptoms that should alert the caregiver to the need for prompt intervention to try to save the graft include severe increasing local pain, soft tissue redness and swelling, local warmth, purulent malodorous drainage, and unexplained low-grade fever. If any combination of the above signs or symptoms is present, the graft should be exposed and examined in its entirety. Early debridement, culturing, and treatment with an antibiotic are recommended to reduce the risk of graft loss and to avoid enlargement of the defect. If the infection is local, controlling the local flora with topical antimicrobials (e.g., silver sulfadiazine, mafenide acetate, silver nitrate, or norfloxacin) may

**Figure 38-13** Techniques for draining a hematoma or seroma. Fluid collections under the graft are great deterrents to proper take and should be drained.

be sufficient in selected cases. On the other hand, if the infection is considered cellulitic in nature, a combination of topical proteolytic-antimicrobial ointment plus a systemic antibiotic is usually warranted. Prophylactic antibiotics are *not* routinely used with outpatient skin grafts. The pain from a skin graft is usually minor and seldom requires opioid analgesics.

## CARE OF THE SPLIT-THICKNESS DONOR SITE

The split-thickness donor site requires proper attention. The donor defect can cause more postoperative discomfort than the grafted area itself, so proper care must be given to hasten the healing process and prevent the donor site from becoming infected. Current donor site dressing approaches can be classified as open, semi-open, occlusive, and semi-occlusive.

The open technique, in which the wound is left uncovered, is associated with significant prolonged healing time and discomfort when compared with a dressed site.

The semi-open technique using fine mesh gauze impregnated with various substances

(e.g., Xeroform [Chesebrough-Ponds, Inc., Greenwich, Conn], bismuth, scarlet red) is frequently used for a large donor site. It is considered relatively inexpensive, easy to manage, and associated with a low infection rate. A nonadherent gauze pad (e.g., Telfa [The Kendall Company, Boston]) can be used between the fine mesh gauze and the dry-absorbent outer gauze. By doing so, the fine mesh wound gauze will not stick to the outer gauze when it dries. This minimizes discomfort when the dressing is removed at wound check. Alternatively, Biobrane (Winthrop Pharmaceuticals, a division of Sterling Drug Inc., New York), a transparent collagen-synthetic composite membrane, may be used as a semi-open donor site dressing. In general, Biobrane has been found to be more comfortable, but unfortunately, it is associated with a higher rate of infection and has a higher cost when compared with fine mesh gauze dressings. [7] For the most part, in semi-open techniques the outer absorbent gauze is removed after 24 to 48 hours, leaving the impregnated gauze now adherent to the wound undisturbed. The wound is then lightly dressed or left open to dry. The fine mesh gauze along with a thin layer of scab is gradually separated from the healing donor site in 10 to 21 days when reepithelialization is completed. Once it is completely exposed, the donor site can be lubricated with either lanolin or cocoa butter.

Early occlusive dressings consisted of fine mesh gauze covered with an impermeable dressing. These dressings had a greater risk of bacterial proliferation and fluid collection. More recently, DuoDerm (ConvaTec) dressings have been used. DuoDerm, an opaque oxygen-impermeable hydrocolloid membrane, does not adhere to the wound, is pain free upon dressing changes, and permits rapid infection-free reepithelialization. The semi-occlusive technique using synthetic transparent adhesive moisture vapor-permeable membranes (e.g., Op-Site [T. J. Smith & Nephew Ltd., Victoria, Greater London], Tegaderm [3-M Company, St. Paul]) covered with loose gauze is an acceptable alternative method that is easy to care for and provides rapid healing. Care is taken to dry the surrounding skin well so that there is good contact between the adhesive plastic sheet and the skin.

Overall, if an occlusive or semi-occlusive technique is preferred, the dressing should remain in place until reepithelialization has occurred or for approximately 10 days, unless there are signs of infection. In the case of infection, the plastic dressing is removed, and frequent moist saline dressing changes are started. Once gross signs of infection have cleared, petrolatum gauze dressings may be applied and the wound cared for as described previously.

Another technique for donor site care is the use of previously harvested residual tissue. If the harvested tissue has been meshed, then residual tissue may be reapplied to the donor site. Doing so shortens the time required for healing.

# Chapter 39 - Soft Tissue Foreign Body Removal

**Gail S. Rudnitsky, Richard C. Barnett**

The presence (or suspicion) of soft tissue foreign bodies (FBs) is a common problem seen in the emergency department. Successful identification and efficient removal of an FB is gratifying to both patient and physician. However, once identified, not all FBs can or should be removed in the emergency department. Occasionally FBs are missed during the initial emergency department evaluation, resulting in discomfort or complications. Missed FBs are one of the leading causes of malpractice claims made against emergency physicians. This chapter will provide guidelines for the evaluation and removal of a variety of FBs.

## GUIDELINES FOR APPROACHING FOREIGN BODIES

Initially the physician should take a history of the method of injury to quickly ascertain the specific characteristics of the foreign material and to formulate the best plan for judicious removal. The history, physical examination, and localization techniques available significantly influence decisions about the time and place of FB removal. Some material, such as wood, should be removed immediately, because retained wood will invariably lead to inflammation and infection. Other material, such as glass or plastic, may be removed on an elective basis, whereas innocuous metallic FBs may often be permanently left embedded in soft tissue. If localization is certain and if removal can be effected under local anesthesia within a manageable period of time (1 hour is usually the upper limit of operative time using local anesthesia), an attempt at removal is generally indicated on the initial visit (given physician and support staff availability). *The patient should be informed before the procedure that the FB may not be located in the time allotted and that subsequent referral or additional procedures may be necessary*.

If, in contrast, the material is deeply imbedded, relatively inert and small (such as a BB), and not located near any vital structures, the time, energy, and effort involved in the removal may be excessive compared with the possible adverse effects of the foreign material remaining in the soft tissue. An ill-conceived extended search for an elusive but otherwise innocuous FB often results in frustration for the physician and discomfort and dissatisfaction for the patient.

The possibility of the FB migrating to involve vital structures is quite remote but should be reviewed in the decision of when and how to remove the FB. Cases of reported missile embolization in the vascular system are influenced by the missile caliber, impact velocity, physical wound characteristics, point of vessel entrance, position and movement of the body, and the velocity of blood flow. [1] Retained bullets usually remain in the soft tissues but may make their way into the vascular system. This usually occurs at the time of injury. Schurr and colleagues [2] reported a paradoxical bullet embolization from the left external iliac vein to the left iliac artery via a patent foramen ovale. When the patient was first examined by physicians, a bullet was

noted on the chest radiograph, and an isolated chest wound was suspected. However,

the bullet had apparently entered the chest, traversed the abdomen to the iliac vein, and then embolized back to the chest and the arterial system.

All clinical decisions require an evaluation for the possibility of infection. Some FBs produce infection in a few days, whereas with other objects, infection or an intense inflammatory response may be delayed for weeks or months, often flaring up for no apparent reason. Some retained FBs, such as wood, will always eventually produce inflammation, while others, such as bullets, rarely do so. If the FB carries dirt particles, pieces of clothing, or other sources of bacterial contamination with it, expeditious removal of the material may be necessary, even though the FB itself is relatively small and unlikely to cause a reaction. If some time has elapsed since the initial injury, careful review of the history of the type of foreign material and the method of introduction is warranted. One should not attempt a hasty or extensive exploration for foreign material that may not exist or is best left alone. The initial history should also include any unusual medical problem that would preclude the use of adequate local anesthesia, such as allergy to local anesthetics, any bleeding diathesis, or any medical problems (including diabetes mellitus, vascular disease, uremia, or a compromised immune status) that might lead to unusual or more difficult wound management. Finally, one needs a cooperative and willing patient. Attempting to remove an FB in an intoxicated, mentally retarded, or overtly uncooperative patient is obviously self-defeating.

It is not rare to encounter a soft tissue FB even though its presence has not been suggested by the history. Anderson and associates reported that physicians who initially treated a series of hand injuries did not suspect the presence of an FB in 75 of 200 consecutive cases. [3] The patient who experiences a sharp, sudden pain in the foot while walking barefoot across the carpet may have a sewing needle or toothpick embedded in the foot, rather than the "sprained foot" that provoked the initial complaint (Fig. 39-1) . *An abscess or cellulitis that recurs or wounds that do not heal as expected should always be investigated for an initially unsuspected retained FB.* [4] [5] Finally, metallic or other FBs visible on the radiograph of a multiple trauma patient should be proved to be extrinsic to the patient--that is, present on the radiograph table or clothing rather than being embedded in the patient. Foreign objects, such as keys or coins, may be surreptitiously embedded in a trauma patient and easily mistaken for artifact (Fig. 39-2) .

If an FB was left in place, the patient should be told why it was not removed. If the patient is referred for delayed removal, this should also be carefully explained and documented. Regardless of whether the FB is removed, all wounds should be cleaned appropriately and tetanus prophylaxis updated (see Chapter 36) .

Rarely, retained lead FBs, such as bullets or shotgun pellets, may leach out lead into the general circulation and produce systemic lead poisoning (Fig. 39-3) (Figure Not Available) . Such a process may take years to develop and can cause vague or nondescript symptoms (e.g., fatigue, arthralgias, headache, or abdominal pain) many years after the offending missile has been forgotten. Elevated blood lead levels are most likely to occur if the lead object is bathed by bodily fluids, such as joint fluid, pleural or peritoneal fluid, or cerebrospinal fluid, and bullets retained in muscle or other soft tissues are not likely to produce any sequelae related to their lead content. Farrell and

coworkers [6] reported unsuspected elevated lead levels in patients with retained lead fragments who presented to the emergency department with a variety of complaints. Lead levels of up to 50 mug/dL were reported. Levels >45 mug/dL are generally considered an indication for chelation therapy. The relation between the retained lead and the presenting symptoms was unclear, but this report verifies the observations of others that retained lead FBs in selected areas can significantly elevate blood lead levels and may produce symptomatic plumbism.

Finally, if foreign material has been found and removed, or if a wound has been explored and no FB found, the patient should be clearly informed that there is *no absolute guarantee that all foreign material has been identified or extracted*. The prudent clinician always leaves open the option that an occult FB may still remain in any wound and informs the patient of signs and symptoms of problems related to retained material. Patients should be assured that additional steps will be undertaken should the presence of foreign material be subsequently suspected.

## IMAGING TECHNIQUES

A variety of imaging techniques are available to the emergency physician to help detect and localize FBs. Many emergency physicians mistakenly believe that if the base of the wound can be clearly visualized and explored and the wound has not entered adipose tissue, an FB can always be ruled out. While this is commonly true, Avner and Baker were able to detect glass by routine radiographs in 11 of 160 wounds (6.9%) that were inspected and believed by the physician to be free of glass. [7] Whenever there is an index of suspicion for a retained FB as a result of the history, mechanism of injury, patient complaint, or examination, attempts should be made to visualize it. Modalities available include plain radiographs, xeroradiographs, fluoroscopy, computed tomography (CT), magnetic resonance imaging (MRI), and ultrasound (US) (Fig. 39-4) (Figure Not Available) . Fluoroscopy is helpful for localizing FBs that are visualized on routine plain films, but even fluoroscopy under magnification will not identify plastic or wooden FBs. [8]

### Plain Radiographs

Plain radiographs are readily available and easily interpreted in the emergency department, and the cost is significantly less than for CT, US, or MRI. [9] The ability of plain films to detect FBs in soft tissues depends on the object's composition (relative density), configuration, size, and orientation. One should never rule out an FB by plain films unless *multiple projections are examined*, since many clearly radiopaque objects are obscured by superimposed bone on one view, yet are quite obvious when viewed from another angle. Metallic objects, such a pins, bullets, and BBs, are readily visualized. Aluminum, which has traditionally been deemed radiolucent, can occasionally be visualized on plain films if the object is projected away from the underlying bone. Ellis demonstrated that pure aluminum fragments as small as 0.5 mm × 0.5 mm × 1 mm could be identified in a chicken wing model simulating a hand or foot, but cautioned that other aluminum FBs, such as pull tabs from cans, may not be visualized in other parts of the body such as the esophagus or stomach. [10]

It is a common misconception that glass must contain lead to be visualized on a plain radiograph. Studies have shown that unless obscured by bone, almost all types of glass in soft tissue (bottles, windshield glass, light bulbs, microscope cover slips, laboratory capillary tubes) can be detected by plain radiographs (Fig. 39-5) (Figure Not Available) . [9] [11] Very small glass fragments (<1 mm) may be more difficult to detect by this technique, [12] but the absence of an FB on multiple projections is strong, although not absolute, evidence that glass is not contained in a wound. Other nonmetallic objects readily visualized include teeth, bone, pencil graphite, asphalt, gravel, and some plastics. [9] [13]

Detection of FBs on plain films can be enhanced by requesting that the technician use an underpenetrated soft tissue technique. [14] Plain films may provide indirect evidence of the presence of an FB if one sees trapped or surrounding air, a radiolucent filling defect, or secondary bony changes such as periosteal elevation, osteolytic and osteoblastic changes, or pseudotumors of bone. [14] Vegetative material, such as thorns, wood, splinters, and cactus spines, are not readily visualized by plain radiographs. These materials absorb body fluids as they set in situ and become isodense with the surrounding tissues. Because of their varying chemical composition and density, plastics may or may not be visible on plain films. However, plastics, which are more brittle and most likely to shatter, are for the most part radiolucent and invisible on plain films. [15]

## Xeroradiography

Xeroradiography is a technique used to enhance soft tissue images. Ionizing radiation passes through the patient and interacts with an electrostatically charged selenium plate. The image is then transferred to a permanent record using a Xerox processor. Although some authors claim this technique is superior to plain radiographs in detecting nonradiopaque FBs, Russell and associates [9] found that xeroradiography was no better than routine radiographs in detecting glass and gravel, and the technique failed to detect plastic and wood FBs. While occasionally useful, xeroradiography is not usually available to the emergency physician.

## Computed Tomography

CT is readily available to most emergency physicians. Since CT depends on x-ray absorption, it generally visualizes the same substances that are detected on plain films. [13] However, because CT can detect subtle differences in soft tissue densities, it may detect FBs not readily visible on plain films. [9] Wood may or may not be detected depending on its degree of hydration. CT produces a better three-dimensional image of tissue than plain films and may also visualize objects embedded in or behind bone.

## Magnetic Resonance Imaging

Although MRI is expensive and not readily available to emergency physicians, it may be superior to CT in detecting small, *nonmetallic*, radiolucent FBs, such as wood,

particularly in the orbit. [16] FBs, which have little or no free water, appear as a signal void on MRI. [9] Plastic is more easily visualized with MRI than with CT. MRI cannot be used for metallic objects and gravel, which contain various ferrometallic particles that produce signal artifacts on MRI. [9] Metallic objects, in addition to producing a high degree of artifact on MRI, have a theoretical risk of shifting within the magnetic field and causing structural damage to adjacent structures. This is of little importance in superficial extremity wounds, but it is particularly important when evaluating FBs in the eye, brain, or deep structures of the neck, face, or extremities.

**Ultrasound**

As US becomes more readily available in emergency departments, it is becoming the imaging modality of choice for objects not readily apparent on plain films. Like fluoroscopy, it can be used to guide the tip of a hemostat to a nonvisualized object [14] while not exposing the operator or patient to ionizing radiation. US is both highly sensitive and highly specific in localizing FBs of all types, [17] but the success of the technique is quite operator dependent. US is especially useful in detecting nonradiopaque FBs such as wood or thorns. A wooden object is usually seen as a bright hyperechoic focus when the scan plane parallels the long axis of the FB or is at right angles to its long axis. [17] US may have more difficulty in visualizing small FBs in the hand or foot, where there are many echogenic structures. [18] While most useful for larger FBs in extremities, US may be difficult to use in some sites such as the web space of the hand. The presence of gas in the surrounding tissue may be responsible for the failure of US to detect FBs; therefore, US should preferably be done before wound exploration. [18] Occasionally, calcific tissue or small sesamoid bones may give a false-positive US examination. Overall, the sensitivity and specificity of high-resolution US in detecting radiolucent FBs is about 90%.

**General Imaging Approach**

A reasonable initial approach for localizing nonvisualized FBs in the emergency department is to obtain multiple-projection plain radiographs (ideally with a soft tissue technique). This will visualize the majority of FBs. Metal and glass should be most visible with this technique. US may be considered for objects known to be radiolucent, such as wood or thorns. Both US and fluoroscopy may be used to guide FB extraction. CT or MRI probably should not be routinely ordered from the emergency department unless one suspects a particularly dangerous FB, such as an intraorbital FB ( *use only CT with a suspected intraorbital or intracranial FB*), or if a previously negatively explored wound exhibits recurrent infection, poor healing, or persistent pain.

# FOREIGN BODY REACTION

Many soft tissue FBs have to be removed because of either infection or FB reaction. A purulent bacterial infection *may* develop in the presence of any FB, but *not in all cases.* Karpman and coworkers found a 15% infection rate ( *Staphylococcus aureus* and Enterobacteriaceae) in a series of 25 patients treated for cactus thorn injuries of the extremities. [19] Certain thorns (black thorn, rose thorns), redwood and Northwest cedar splinters, toothpicks, hair, and stingray or sea urchin spines are noted for their ability to

initiate chronic FB reactions. Sea urchin spines and other marine FBs are covered with slime, calcareous material, and other debris that commonly initiates an FB granuloma. The inflammatory reaction seen with cactus thorns may be an allergic reaction to fungus found on the cactus plant. Many FB reactions are

thought to be due to the inflammatory response to organic material and may represent infection from bacteria introduced at wounding. Clinically evident reactions may be delayed for weeks or even years following injury (Fig. 39-6) . The chronic infection or inflammatory reaction may not be accompanied by the production of pus, but it may be quite painful and may result in loss of function. FBs may also be associated with the formation of a chronic pseudotumor, development of a sinus tract, or evidence of osteomyelitis-like lesions of bone and soft tissue. [4] Organic material has also been noted to induce chronic tenosynovitis, chronic monarticular synovitis, and chronic bursitis.

Rapidly traveling projectiles with considerable inherent heat (e.g., bullets) are less likely to cause infection but are more apt to cause damage during passage through the tissue. As noted above, the composition and location of the FB, as well as patient medical status and vocational/avocational activities, greatly affect decision making related to FB removal. One must judiciously evaluate and manage each FB injury individually.

## FOREIGN BODY SCENARIOS

Certain mechanisms of injury, such as punching or kicking out a window or stepping on an unknown object while walking in a field or stream, are highly suggestive of a retained foreign body (Fig. 39-7) . While not all lacerations caused by glass require a radiograph, imaging should be liberally used under such circumstances.

Generally, lacerations from metal objects do not contain foreign material. However, if the patient states that considerable force was applied during injury and the instrument is not available for inspection, radiographic imaging may be warranted. Occasionally a piece of a knife blade's tip may be broken off as the knife encounters bone. Animal or human bites often warrant radiographs to assess for associated fractures. Such films should be viewed carefully for imbedded teeth in the wound. Since the patient's perception of an FB correlates with the presence of an FB, *it is axiomatic that the clinician ask the patient if he or she thinks that a wound may harbor foreign material* and proceed more cautiously if the answer is affirmative.

Many, but not all, retained FBs will produce pain when the patient moves the injured area or when the wound edges are palpated or depressed. It is prudent to carefully palpate the periphery of all wounds to elicit such tenderness. Superficial  FBs may be palpated through the skin, but surprisingly large FBs may be found in seemingly minor wounds, without any external evidence. While puncture wounds are more likely to contain an FB than wide, gaping lacerations, the external characteristics of the wound do not yield firm evidence as to the presence or absence of an FB.

# GUIDELINES FOR FOREIGN BODY REMOVAL

Following the initial history, examination, and *preoperative and preanesthetic documentation of the neurovascular status of the patient*, a decision must be made as to the time and place of removal. If the FB is to be removed in the emergency department, it is wise to set a time limit and stick to it. Thirty minutes is probably reasonable in a busy emergency department. More difficult procedures should be referred. Many FBs appear superficial on radiographs, suggesting that removal will be quite easy. However, surprisingly large or presumed superficial FBs often prove quite elusive.

**Localization**

Superficial FBs, such as splinters, bullets, or embedded glass, may be palpated if they are near the skin surface. Deeper FBs must be localized by other techniques. A metal probe may identify the FB by feel or sound. Glass is difficult to identify by sight in soft tissue but creates a characteristic grating sound when touched with metal. Because of the increasing incidence of the human immunodeficiency virus (HIV), *probing a wound with a gloved finger to locate or identify an FB is strongly discouraged* (Fig. 39-8) , since sharp objects can easily penetrate the examining finger.

Radiographs are the best method for estimating the general location, depth, and structure of radiopaque FBs. If one strategically attaches a marker (needle or paper clip) to the skin surface at the wound entrance before taking a radiograph, the FB will be seen in relation to the entrance wound. This also helps to identify the path that leads to the FB and the relative distance from the surface to the FB. [20] Needles at 2 angles may also be passed to aid localization (Fig. 39-9) (Figure Not Available) . A special technique using measurements on anteroposterior and lateral radiographs and a blind dissection method have been advocated to remove needles from the foot. [21] When the material to be detected is suspected of being difficult to visualize on a conventional radiograph, then xeroradiography, high-resolution US, CT, or MRI should be considered.

While rarely available, fluoroscopic image-intensifying equipment may be used to follow the wound's entrance, localize the material, grasp the FB, and remove it without making a larger incision. A potential disadvantage of this procedure is the increased amount of radiation that may be required. Ariyan has described a technique in which 2 needles are placed in the soft tissue from opposite directions, pointing toward the FB. [22] The extremity is rotated while the physician watches the image under the image intensifier to obtain a three-dimensional effect. An incision is placed perpendicular to the plane of the needles, and the object is removed. US can also be used to remove an FB under direct visualization. This is particularly useful for radiolucent FBs.

Some authors have suggested injecting the entrance wound with methylene blue to outline the tract of the FB. [23] Note that excess methylene blue that spills onto the skin surface may be removed with ether. The blue line of injected dye is followed into the deeper tissues. This technique is of limited value, because the tract of the FB often

closes tightly and does not allow passage of the methylene blue.

## Equipment

A standard suture tray with a scalpel is usually adequate equipment for removal of most simple FBs. Tissue retractors, special pickups, and loupes may be added as needed. Good direct light is essential for success, and some physicians prefer to use a head lamp. Sitting on a stool makes the procedure easier on the physician's legs and back.

Local soft tissue injection with buffered bupivacaine or lidocaine (with epinephrine for other than digital blocks) is the recommended anesthesia for the removal of most soft tissue FBs. IV regional anesthesia or selected nerve blocks may be useful. *The judicious use of sedation (parenteral, rectal, or oral) is strongly advised if the clinician senses undue apprehension or anxiety in the patient.* This may be especially helpful in children or patients otherwise unable to cooperate with

the operator. If the patient is totally uncooperative, exploration should be postponed for a more opportune time.

The use of an arterial tourniquet is essential to provide a bloodless field for the removal of a soft tissue FB in an extremity. A blood pressure cuff or portable self-contained pneumatic cuff inflated above arterial pressure may be used on the upper arm, forearm, leg, or thigh. To limit bothersome backbleeding, the extremity is elevated and wrapped with an elastic bandage to exsanguinate the extremity before the tourniquet is inflated. In the digits, a Penrose drain or specialized tourniquet may be used as a tourniquet at the base of the finger or toes. A sterile glove may also be used as a finger tourniquet. The fingertip of the glove on the involved finger is cut, and the glove is rolled down to the base of the finger. Most patients can tolerate an ischemic tourniquet for 15 to 30 minutes, and it is safe to stop circulation to an extremity for this time period.

## Operative Technique

The specific technique for removal of an FB is tailored to each clinical situation. In general, FBs should be removed only under direct vision. *Blind grasping into a wound with a hemostat in an effort to remove an FB should be avoided.* This technique is especially dangerous in the hand, foot, neck, or face, where vital structures may be easily damaged.

In most cases one should *enlarge the entrance wound with an adequate skin incision.* Attempting to remove an FB through a puncture wound or an inadequate skin incision is a common error that is both frustrating and self-defeating. Following a proper skin incision, the wound is explored by carefully spreading the soft tissue with a hemostat (Fig. 39-10) (Figure Not Available) . The FB can often be felt with an instrument before it can be seen. In an extremity that has been made ischemic by a tourniquet, the tract of the FB may be followed, although the tract frequently cannot be identified in muscle or fat.

If the FB is difficult to visualize (such as with fiberglass or plastic FBs) and if it is located in the superficial soft tissue, excision of a small block of tissue, rather than removal of the FB alone, may be necessary. Block excision is also required if the FB has contaminated the surrounding soft tissue. It must be noted that excision of a block of tissue is done only under direct vision and after nerves, tendons, and vessels have been identified and excluded from the excision.

If an FB such as a thorn or needle enters the skin perpendicularly, a linear incision may pass to 1 side of the FB, and it will be difficult to determine where the FB lies in relation to the incision (Fig. 39-11 (Figure Not Available) *A* and *B*). The search must then be extended into the walls of the incision rather than through the skin. [24] In such cases it is advisable to excise a small ellipse of skin and undermine the skin for 0.5 to 1.0 cm in all directions (Fig. 39-11 (Figure Not Available) *C*). The tissue is then compressed from the sides in hopes that the FB will extrude and can be grasped with an instrument.

Following FB removal, the wound is irrigated under pressure with saline. If a small incision has been made in a noncosmetic area (such as the bottom of the foot), the incision is left open and bandaged. The area may be soaked in hot water for a few days, and a return visit is necessary only if signs of infection develop. If a large incision has been created, the skin may be primarily sutured. In cases in which gross contamination has occurred, the wound should not be closed on the initial visit. The wound may be packed open, and the skin may subsequently be sutured, if free of signs of inflammation, in 3 to 5 days (delayed closure). Tetanus prophylaxis should be given, but the use of postoperative antibiotics is not standard. They may be indicated for immunocompromised patients, if there was excessive time between injury and removal, if the wound was obviously contaminated, or when the ability to adequately clean a wound is suspect; however, under these circumstances it is more prudent to opt for an open wound and a delayed closure. If antibiotics are prescribed prophylactically, 3 days of a penicillinase-resistant penicillin or a first-generation cephalosporin should suffice. Under certain circumstances (e.g., stepping on a nail or a marine FB), alternative antibiotics may be indicated. The majority of wounds that contained glass or metal FBs may be irrigated and closed primarily. Importantly, the presence of infection on a subsequent visit strongly raises the concern for an occult retained FB.

**Traumatic Tattooing**

Ground-in foreign material or tattooing of the skin is a troublesome problem, because foreign matter will permanently disfigure the skin. Many cases may be managed with adequate local anesthesia and meticulous debridement with a sponge, a scrub brush, or a toothbrush. If it is impossible to remove all the foreign material with these methods, careful consideration should be given to a secondary excision of the tattooed area and primary closure with subsequent plastic surgery to repair the defect. It is often impossible to safely and easily remove traumatic tattooing in the emergency department; referral for more extensive surgical treatment following local wound care is quite acceptable. Dermabrasion may be an acceptable delayed treatment when the tattooing is superficial. [25]

**Foreign Bodies in Fatty Tissue**

FBs located in fatty tissues may be removed by making an elliptical incision surrounding the entrance wound; grasping the skin of the ellipse loosely with an Allis forceps; undercutting the incision until the FB is contacted; and removing the FB, skin, and entrance tract in one block (Fig. 39-12) (Figure Not Available) . In most instances, a small portion of subcutaneous (SQ) fat should be removed along with the FB to minimize infection. FBs in fat are very mobile, and probing may displace them even more. FBs that are embedded in fat and are perpendicular to the skin may also be removed, as shown in Figure 39-11 (Figure Not Available) .

**Puncture Wounds to the Sole of the Foot**

FBs are often introduced with puncture wounds to the sole of the foot. A common scenario is stepping on a nail while wearing tennis shoes and socks. If the patient reports that the nail was removed intact, the possibility remains that small fragments or fibers from the shoe or sock were introduced into the wound. When present in the wound, this material will invariably serve as a nucleus for infection. Therefore, these wounds should always be evaluated for the presence of foreign material, and some may require exploration (see Fig. 54-18) (Figure Not Available) . Proper evaluation requires that the patient be placed prone on a stretcher and adequate lighting used. A magnifying device may be helpful. Routine radiographs are probably not helpful, as most retained material is radiolucent. If a deep FB is suspected, CT, MRI, or US may be indicated.

Initially one visualizes the punctured area searching for obvious threads or pieces of rubber by merely elevating the small flap of calloused epidermis that commonly covers the entrance wound. Superficial and readily seen FBs, including small pieces of rust or debris, are often present. If foreign material is found, it should be removed by excising a small piece of tissue. A large laceration under the skin flap also warrants local wound exploration. Although local anesthesia is often sufficient, regional nerve blocks for the sole should be considered if extensive exploration or debridement are anticipated (see Chapter 32) . The surface of the site is irrigated, left open, and appropriately bandaged. Generally this is all that is required under these circumstances.

If infection is already present, the incidence of a retained FB is high. If infection is present, often with accompanying lymphadenitis, the puncture site may be cored out with a 2- to 4-mm-diameter incision carried up to 2 cm deep. The cored-out area is superficially irrigated and left open. It is best to avoid advancing an irrigating catheter into the deep recesses of the tract, as this serves no useful purpose and may disseminate infection.

Routinely removing a plug of tissue or coring a noninfected wound with no visible foreign material is quite controversial. [26] [27] This empiric procedure has been advocated by some, but it increases patient discomfort and disability and has never been shown to reduce the incidence of serious sequelae. It is best reserved for patients with early inflammation or other findings on wound inspection that suggest a retained FB. It is

likely overtreatment in the majority of cases.

A first-generation cephalosporin or a penicillinase-resistant penicillin is a reasonable antibiotic choice for obviously infected puncture wounds. Noninfected puncture wounds have not been shown to benefit from antibiotics; routine use of antibiotics in noninfected wounds is discouraged except in high-risk patients. Empiric antibiotics may select out resistant organisms. Although rare, the most dreaded complication of a deep puncture wound of the foot is *Pseudomonas* osteomyelitis (the organism lives in the rubber soles of tennis shoes), but there is no evidence that routine use of prophylactic antibiotics will prevent this complication. The key to a successful outcome of any puncture of the foot is to realize that pain that persists for more than a few days after stepping on a nail may represent the beginning of a limb-threatening infection. In the early course of a deep *Pseudomonas* infection, fever may be absent; the complete blood cell (CBC) count and sedimentation rate, normal; and a plain film, negative. A bone scan is the best way to evaluate a foot that still remains painful or swollen 2 weeks after stepping on a nail.

**Subungual Foreign Bodies**

Special attention is required for subungual FBs that are deeply embedded in the nailbed. [28] In some instances this may require removing a small portion of the nail with double-pointed heavy scissors and grasping the foreign material with the splinter forceps (Fig. 39-13) (Figure Not Available) . Occasionally, complete removal of the nail may be required. Obviously, a digital block is needed for techniques involving manipulation of the nail or nailbed. An interesting technique has been suggested in which a sterile hypodermic needle, bent at its tip, is slid under the nail and hooks the FB, allowing its withdrawal. Alternatively, a 19-ga hypodermic needle can be slid under the nail to surround a small splinter. The needle tip is then brought against the underside of the nail to secure the splinter. The needle and splinter are then removed as a unit. [29] Wooden splinters are commonly embedded under the fingernail. *Such FBs must be completely removed, because subsequent infection is certain*. The proximity of the distal phalanx to the subungual area is a constant concern for the development of osteomyelitis.

**Fishhooks**

There are several methods of removing a fishhook. The preferred method depends on the conditions under which the removal is to take place. [30] [31] [32] The traditional manner for removal requires advancement of the fishhook and cutting the fishhook proximal to the barb. Generally, local anesthesia (1% lidocaine) is infiltrated into the tissue overlying the barb, the barb is forced through the anesthetized skin and clipped off, and the rest of the hook is removed along the direction of entry (Fig. 39-14) (Figure Not Available) . In the field or stream, removal of a fishhook may be accomplished without local anesthetic using a quick tug technique. The same technique may be used in the emergency department; some prefer to use local 1% lidocaine to facilitate removal. This "stream" technique (Fig. 39-15) (Figure Not Available) uses a looped string or fishing line passed around the belly of the hook at the point at which it enters the skin. Approximately 1 foot of string should be wrapped around the dominant hand to give strong traction. The shank of the hook should be held parallel to and in approximation with the skin by the

index finger of the opposite hand. The thumb and middle finger of the opposite hand stabilize and depress the barb, which helps the index finger to disengage the barb from the SQ tissue. When the barb has been disengaged, a *sharp* pull (i.e., a quick tug with a snapping motion) with the dominant hand removes the hook through the wound of entry. The hook often flies out of the patient, and care should be taken to keep bystanders out of the expected path. A commercial fishhook extractor device that is based on this method but grasps the hook during removal is available (Minto Research and Development Inc., Redding, Calif.).

Alternatively, an 18-ga needle may be used to cover the barb (Fig. 39-16) (Figure Not Available) . After adequate local anesthetic has been administered, the needle should be passed through the entrance wound of the hook parallel to the shank of the hook to sheath the barb and allow the hook to be backed out while the barb is covered. An alternative to this procedure is to insert a No. 11 blade parallel to the shank of the hook down to the barb. Using the point of the blade, free the SQ tissue that is engaged on the barb, sheath the barb with the point of the No. 11 blade, and back the hook out, with the blade protecting the barb.

The retrograde techniques may cause less tissue trauma, if the barb does not already protrude from the skin. Tetanus prophylaxis should be given. Antibiotics are not necessary.

**Wooden Splinters**

Because of the potential for inflammation, *pieces of wood must be completely removed from soft tissue*. Simply grasping the end of a superficial protruding splinter may be adequate, but care should be taken not to leave small pieces of the FB in the wound. Some splinters cannot be visualized at the point of entry but can be easily and readily palpated beneath the skin. When a wooden FB is SQ, it is advisable to cut down on the long axis of the FB to remove it via a skin incision, rather than pulling it out through the entrance wound (Fig. 39-17) . An incision may seem extensive, and it does create a laceration where only a puncture wound existed, but opening the tract allows for thorough cleaning and mandatory removal of small pieces of the splinter that may otherwise remain. The linear skin incision may then be sutured.

Particular mention should be made of certain wood splinters that are reactive and pliable, such as California redwood and Northwest cedar. Any wood that is easily fragmented requires meticulous care to ensure removal of all the material. Wood is generally not visible on a standard radiograph or xeroradiograph unless it is covered with lead paint. US is the technique of choice to evaluate for a wood FB, but CT or MRI may also be helpful.

Occasionally, the most expeditious method of removing small wooden splinters is complete excision of the entrance tract and the FB en bloc, followed by a linear closure.

**Pencil Lead**

Good judgment must be used in removing graphite from pencils when lodged in the skin. Because graphite invariably leaves a pigmented tattooing in the soft tissue, it is preferable to excise the material en bloc when pencil lead is found in a cosmetic area. The graphite specks cannot be irrigated or scrubbed off, and tattooing results if they are not removed.

**Metallic Fragments**

High-velocity fragments (e.g., bullets, BBs, chips of wood-splitting mauls, or other metallic particles caused by metal striking metal) are easy to visualize radiographically and relatively simple to remove unless they are embedded in areas that are anatomically difficult to approach. Before removal of the FB, the area in which the fragment is embedded should be assessed to determine which structures are involved along the wound entrance, which structures might be encountered in attempting to cut down on the FB, and which of those structures can be sacrificed to allow for adequate removal of the foreign material. It is preferable to defer the removal of deeply embedded metallic FBs unless symptoms of infection develop.

The treatment of high-velocity FBs (as seen with modern military or sporting ammunition) and their associated wounds is beyond the scope of this discussion. Retained nonexplosive metallic fragments are inert and rarely cause infection. They usually become encysted after a period of

time. Lead toxicity from bullet fragments is rare and generally only of concern when the fragment is in contact with synovium (see previous discussion). The value of routine prophylactic antibiotics for metallic FBs left in the soft tissue has not been proved.

A sterile magnet may be used to facilitate removal of small metallic fragments. [33] The magnet is introduced into the entry site using a small scalpel and hemostat to extend and open the wound, respectively, as needed. When the magnet contacts the metal, a click is heard. The FB is then removed, attached to the magnet. If there is resistance to extraction of the object, directed exploration can be done with the FB attached to the magnet.

Taser darts used for immobilization of violent individuals are like fishhooks and must be removed under local anesthesia after the attached wires have been cut. Backing the dart out as with a fishhook is not possible owing to the dart's unique construction. Cutting down on the dart under local anesthesia is the preferred method of removal. [34] [35]

**Marine Foreign Bodies**

While most marine FBs, such as shell fragments, may be treated like other FBs, a number of marine animals carry toxins and may leave FBs that require special consideration.

**Catfish.**

Several species of catfish in North America contain toxic venom, and their sting can imbed an FB. Envenomation occurs when the fish becomes agitated and the dorsal and pectoral spines become locked in the extended position and penetrate the hand or foot. The venom is then secreted from an epidermal gland at the base of the spine. [36] This is initially quite painful but is usually transient, and there is no specific antitoxin. Treatment consists of local care and analgesics. The affected part should be immersed in hot water (approximately 43.3 °C [110 °F]) for at least 30 minutes if pain is severe. This is believed to provide symptomatic relief by decreasing vascular and muscle spasm. [37] Local injection of the wound with alkalized bupivacaine provides local analgesic and may also neutralize the toxin. [36] The wound should be inspected and any remaining spine removed. A radiograph may be taken to confirm the absence of FBs, but catfish spines and cartilage may be radiolucent. The wound should be thoroughly cleaned and irrigated. Tetanus prophylaxis should be updated. Some authors [36] recommend empiric antibiotic therapy to cover gram-negative bacilli (e.g., *Aeromonas hydrophila*), but infection is quite rare, and routine antibiotic prophylaxis is not standard.

**Coelenterates.**

Coelenterates, including the Portuguese man-of-war, the true jellyfish, fire coral, the box jellyfish, and sea anemones, inject a toxin that is responsible for many marine envenomations. Reactions to the toxin may be local or systemic, and pain may be severe. Systemic reactions usually consist of fever, chills, and muscle spasm, but severe reactions may result in neurologic sequelae ranging from malaise and headache to coma and paralysis. Cardiopulmonary manifestations include dysrhythmias, hypotension, syncope, bronchospasm, laryngeal edema, and cardiorespiratory failure. [38] [39]

Coelenterates do not inject FBs per se, but deposit their venom-containing organelles, called *nematocysts,* on the skin of the unfortunate victim (Fig. 39-18) . Nematocysts are present by the thousands on the tentacles of coelenterates, and even minimal contact with the animal causes the tentacles to break off and adhere to the skin. Nematocysts are released from the tentacles, cling to the skin, and discharge venom. An injection apparatus in the nematocyst penetrates the dermis and diffuses the venom into the soft tissue. Decontamination and removal of the nematocysts decrease pain and systemic reactions.

Vinegar (5% acetic acid) is the initial decontaminating agent of choice and will inactivate nematocystic discharge of most species of jellyfish, Portuguese man-of-war, and sea anemones. [38] This should be applied continuously for 30 minutes, or until the pain is gone. [39] Fresh water should not be applied to the wound, as this may worsen the envenomation by stimulating nematocyst discharge. Isopropyl alcohol has also been used as a decontaminating agent but has been shown to cause nematocyst discharge in vitro. [38] Other suggested but unproven remedies include meat tenderizer, ammonia,

baking soda, urine, olive oil, sugar, and papaya latex.

After the wound has been decontaminated, remaining tentacle fragments should be removed. Large fragments may be removed with forceps. Individual nematocysts are quite small (<1 mm in length) and are not easily visible to the eye, but if there is local pain, it should be assumed that the organelles remain on the skin. Some nematocysts are so small that they must be seen with a microscope on skin biopsy. Nematocysts should not be removed by rubbing the

skin with sand, as this increases venom discharge. To remove remaining nematocysts, the skin should be scraped with a hard edge, such as a knife blade or credit card held perpendicular to the skin. Remaining fragments may also be removed by applying shaving cream and shaving the area gently. [39]

After decontamination, topical anesthetics or steroids may be used. Prophylactic antibiotics are not necessary. Routine wound care should be followed. Allergic and systemic reactions should be treated appropriately. Antivenin is available only for box jellyfish envenomation (1 to 3 ampules).

**Coral.**

Fire corals are another type of coelenterate that produces toxicity by stinging. Other corals are sharp and irritating if touched. "Coral cuts" occur in divers and snorkelers exploring coral reefs. These organisms can produce deep cuts, with local reactions such as pruritus, erythema, and urticaria occurring at the site of the injury. Delayed healing may occur with secondary development of cellulitis or ulceration, perhaps as a result of contamination of the wound with bacteria or microparticles of coral. [38] Treatment consists of saline irrigation. Hydrogen peroxide may be used to remove small coral particles from the wound. [38] The wound should not be closed; wet-to-dry dressings may be used.

**Sponges.**

Sponges produce both an irritant and a contact dermatitis. The irritant dermatitis occurs as a result of sponge spicules embedded in the victim's skin. These spicules may be removed with adhesive tape (applied to the skin and then peeled back) and the area should then be bathed with vinegar. [38] The contact dermatitis which is believed to be caused by a toxin produces erythema, pruritus, and vesicles similar to poison oak. [38] Treatment is initial immersion in vinegar followed by local steroid creams.

**Sea urchins and starfish.**

Sea urchins and starfish are free-living echinoderms covered with spines that may contain toxins. These spines are sharp and brittle and may become embedded in victims if they inadvertently step on or handle these creatures. A severe local reaction may result from the venom contained in the spines. Local pain usually responds quite well to immersion in hot water (43.3 to 46.1 °C [110 to 115 °F]) for 30 to 90 minutes.

Retained spines may become infected or cause delayed (up to 1 to 2 months) FB granulomas. This reaction is poorly understood but may produce an intense and persistent inflammatory reaction. Spines that penetrate joints may induce a synovitis. Echinoderm spines may discharge a purple dye that may be mistaken for a retained spine. [39] Spines are usually visible on radiographs and should be removed if possible, although they are brittle and can break off in the skin. If located in a joint or near a nerve, surgical extraction may be necessary using an operative microscope. [38] Otherwise, if removal is difficult, the spine may be left in place until it resorbs or a local reaction takes place. The wound may then be opened and drained and allowed to close by secondary intention.

**Stingrays.**

Stingray envenomation usually occurs when a bather accidentally steps on an animal that is resting on the bottom in shallow water, covered by sand (Fig. 39-19) . This causes the stingray to lash out its whip-like caudal appendage, or tail, which contains 1 to 4 venom-containing stinging spines. The spines are sharp and jagged and can produce a significant laceration. Portions of the spine may become buried in the victim's skin. Each spine is covered with a sheath containing venom glands, and in addition to immediate toxin-induced pain, pieces of the spine or sheath may be left in the wound. These fragments do not dissolve and must be removed, but they are often difficult to locate. Persistent pain and inflammation (weeks to months after the attack) mandate consideration of a retained FB, but a persistent and difficult-to-treat irritative process can occur in the absence of a retained spine or sheath. Immediate local and systemic reactions occur as a result of injection of a complex toxin. Systemic reactions may be severe and can include muscle cramps, vomiting, seizures, hypotension, arrhythmias, and (rarely) death.

Treatment consists of irrigation with saline or fresh water followed by hot water immersion at 43.3 to 46.1 °C (110 to 115 °F). Local anesthetics without vasoconstrictors provide effective analgesia. All wounds should be explored and debrided, all remnants of the spine and integumentary sheath removed. [38] The wound should not be closed.

**Antibiotic therapy.**

Prophylactic antibiotic therapy for marine injuries is common. Unlike other soft tissue infections, marine injuries become infected with unusual gram-negative organisms, particularly *Vibrio* species. Although there are little data evaluating the effect of specific antibiotics, it is recommended that quinolones, trimethoprim-sulfamethoxazole, or aminoglycosides be used in lieu of penicillin, ampicillin, erythromycin, or first-generation cephalosporins. [39] It is always difficult to differentiate chronic inflammation caused by toxins and foreign material from true infection, and often surgical exploration is required in persistent cases. Tetanus prophylaxis should be routine.

**Cactus Spines**

Cactus spines vary considerably in size. The difficulty of removal is generally inversely

proportional to the FB size. [40] Larger embedded cactus spines are managed like wood splinter and sea urchin spine FBs. More advanced imaging techniques (xeroradiography, ultrasonography, CT, or MRI) may be required for localization of deeply embedded spines.

Deeply embedded cactus spines generally produce granulomatous reactions, and infections are rare. [19] Hence, efforts to remove deeply embedded spines should be made after carefully weighing the benefit and potential harm related to a deep exploration, especially in a sensitive location. [40] Superficially embedded medium- to large-sized cactus spines are best removed by direct axial traction of each spine using forceps. Smaller spines (glochids) may be difficult and tedious to remove individually. Adherent facial mask gel application and removal of spines en masse with the mask are recommended (Fig. 39-20) (Figure Not Available) . Depilatory wax melted in a microwave oven and applied warm, [41] commercial facial gels, [40] [42] and household glue (Elmer's Glue-All [Borden Inc., Columbus, OH]) have all been recommended for this purpose. [43] The practitioner should be aware that over-the-counter "home use" facial mask gels are not adherent enough to be effective without multiple applications (up to 8 or more).

### Postoperative Foreign Bodies

FBs in the form of nonabsorbed suture material are frequently encountered in the postoperative period. Drainage, localized inflammatory reaction along the suture line, and localized pain and tenderness are characteristic of a retained FB (suture abscess). In this instance, probing the wound with a sterilize needle bent in the shape of a crochet hook is frequently rewarding. Hooking the suture material through the sinus tract and removing it allows the wound to heal over the tract.

### Ring Removal

Frequently, a ring must be removed to prevent laceration of tissue or vascular compromise. If removal is not possible using thorough lubrication (a water-soluble lubricant [e.g., K-Y jelly]) and a circular motion with traction on the ring, it may be necessary to either cut the ring off or remove it by the string-wrap method. It is preferable to remove all rings before the edema is extensive enough to cause pain or vascular compromise.

#### String-wrap method.

An occasional patient can remain calm during this procedure, but if swelling is significant or the digit has been traumatized, anesthesia is necessary (Fig. 39-21) . Anesthesia is obtained by a digital or metacarpal block applied to produce minimal tissue distention. Prior to ring removal, a wide Penrose drain is wrapped circumferentially in a distal-to-proximal direction to reduce soft tissue swelling. For maximum effect, the wrap should remain in place for a few minutes. Some nonanesthetized patients panic during the procedure because of increasing pain due to

compression and unwinding. [44]

A 20- to 25-inch piece of string, umbilical tape, or thick silk suture is first passed between the ring and the finger. Shorter lengths are discouraged, as one may need to repeat the wrapping procedure midway. If there is marked soft tissue swelling, the tip of a hemostat may be passed under the ring to grasp the string and pull it through the ring. The distal string is wrapped clockwise around the swollen finger (proximal to distal) to include the proximal interphalangeal (PIP) joint and the entire swollen finger. The wrapping is begun next to the ring. The wrap should be snug enough to compress the swollen tissue. The successive loops of the wrap are placed next to each other to keep any swollen tissue from bulging between the strands. When the wrapping has been completed, the proximal end of the string is carefully unwound in the same clockwise direction, forcing the ring over that portion of the finger that has been compressed by the wrap. The PIP joint is the area that is most difficult to maneuver and that causes the most pain to the patient.

Occasionally, one must rewrap the finger if it was not carefully wrapped initially. It is not uncommon to produce abrasions or other trauma to the skin during this procedure. If the finger with the ring is lacerated or there are underlying fractures, it is prudent to cut off the ring instead of attempting this technique.

Two alternatives to this procedure have been described. The first one uses an entire skein of 2.0 surgical silk ties instead of a single piece of string. The procedure is the same: The ties are passed underneath the ring with a hemostat. The short ends of the ties are clamped with a hemostat on the hand side of the ring. The long end of the skein is wrapped around the finger and then unwound from the short end, as described above. This method may be less traumatic to the finger than a single piece of string. [45]

The second technique uses a 3- to 4-mm rubber band that is passed beneath the ring after the finger has been lubricated with soap or petroleum jelly. The physician then places a finger through both loops of the rubber band (1 above and 1 below the ring) and pulls the rubber band distally while moving the rubber band circumferentially around the ring and the finger until the ring is removed.

**Ring cutter.**

A ring cutter should be used if the ring is not of high value or there is excessive swelling. Manual and power devices are available. The ring cutter has a small hook that fits under the ring and serves as a guide for a saw-toothed wheel that cuts the metal. The cut ends of the ring are spread using large hemostats (e.g., Kelly clamps), and the ring is removed. Cut rings may be repaired by a jeweler.

**Tick Removal**

Early removal of ticks is advisable, since the hard tick of the Ixodid family is likely to transmit disease. Rocky Mountain spotted fever, Lyme disease, tularemia, and ascending paralysis have been identified as tick-borne diseases. Tick removal of Ixodid

ticks is difficult, because the mouth parts are cemented to the skin of the host (Fig. 39-22 *A*). The longer the tick is attached, the more difficult removal may become. Traditional and folk methods of forcing the tick to disengage (e.g., the use of petroleum jelly, fingernail polish, a hot match, or alcohol) are probably ineffectual, and removal by mechanical means is recommended. [47]

Protection of the operator by use of a hemostat or gloves is advisable. The tick is grasped as close to the patient's skin surface as possible and gently pulled free with steady axial traction (Fig. 39-22 *B*). The tick body should not be squeezed, crushed, or punctured. If mouth parts are left behind after removal of the body, excision under local anesthesia is needed to prevent local infection.

Many patients have great anxiety over subsequent tick-borne diseases following tick removal. There is no standard of care for the use of prophylactic antibiotics to prevent Lyme disease or Rocky Mountain spotted fever. Theoretically, a tick infected with Lyme disease must remain attached for 24 hours to transmit the disease, and even then, only 10% to 20% of bites from infected ticks will result in transmission.  Although all patients should be advised about the signs and symptoms of these diseases, routine prophylactic antibiotic use is controversial. Prophylactic antibiotics (ampicillin or tetracycline) following tick removal may be more reasonable in endemic areas.

### Zipper Injuries

The skin of the penis may become painfully entangled in a zipper mechanism. Unzipping the zipper frequently lacerates the skin and increases the amount of tissue caught in the mechanism. Although the physician may anesthetize the skin and excise the entrapped tissue, a less invasive method may be useful.

The interlocking teeth of the zipper fall apart if the median bar (diamond or bridge) of the zipper is cut in half (Fig. 39-23) (Figure Not Available) . The skin is subsequently freed. A bone cutter or wire clippers and a moderate amount of force may be required to break the bar. Patients with penile lacerations warrant urologic follow-up to assess for urethral injury.

### Hair-Thread Tourniquet

Hair or thread fibers adherent to infant clothing occasionally become tightly wrapped about the child's digits or genitals (Fig. 39-24) . [48] If these are left in place, automatic amputation may eventually occur. The offending fibers may be difficult to visualize, and the child is often brought for evaluation only after signs of distal ischemia appear. Occasionally the fiber can be grasped with toothless forceps or a small hemostat and unwrapped. More commonly the fibers cannot be identified and are deeply imbedded in swollen tissue.

A No. 11 blade can be used to cut the constricting bands under a regional nerve block. It may be difficult to identify individual hairs that are deeply imbedded in a swollen digit and even more difficult to assess the success of one's intervention. Often multiple hairs

are involved. Because the bands may be quite deep, the incision should avoid known neurovascular tracts. Barton and colleagues [48] recommend a dorsal, rather than lateral, incision on the digits. If the soft tissue of the distal digit has been rotated following a circumferential dermal laceration from the tourniquet, the distal tissue can be realigned with the proximal tissue and 2 dorsolateral sutures placed to maintain the digit in alignment.

Generally, conservative wound care is sufficient once the band has been removed. Application of an antibiotic ointment may enhance healing and allow easier removal of serous drainage from the circumferential laceration. Clinical reassessment in 24 hours will indicate whether any constricting bands remain.

# Chapter 40 - Incision and Drainage

**Howard Blumstein**

Cutaneous abscesses are among the soft tissue infections most frequently encountered in the emergency department (ED). Approximately 1 to 2% of patients presenting to the ED receive care for cutaneous abscesses. [1] [2] In contrast with most bacterial diseases, which are usually described in terms of their etiologic agent, cutaneous abscesses are best described in terms of their location. There has been little systematic investigation into the bacteriology of simple cutaneous abscesses, and there have been few new recommendations for improved management over the years. The probable reason for this lack of scientific rigor is the predictable and striking clinical recovery once a mature abscess is incised and drained. The exact reasons for this amelioration of local and constitutional symptoms are unknown; however, it is clear that the exact bacteriology of cutaneous abscesses, while academically interesting, is essentially inconsequential to the final outcome in most cases. [3]

Although most incision-and-drainage procedures are performed for decompression of purulent collections, drainage of sterile hematomas or seromas may be required in the ED. In general, the same principles used for formal drainage of pus in the soft tissues apply to drainage of a sterile fluid collection, and hence one can directly apply the principles of this chapter to the drainage of sterile fluids. In addition, when a sterile fluid collection is drained, the operator has the option of primarily closing the incision site following wound irrigation (see Chapter 37 for wound closure techniques). Drainage of a soft tissue hematoma is generally best postponed several days following an initial injury to permit hemostasis and to minimize the risk of hematoma reaccumulation following drainage. The procedure is generally reserved for those soft tissue hematomas that are large and painful (secondary to tissue distention) and are expected to either resolve slowly or result in soft tissue deformity if not drained. Drainage of a subungual hematoma represents a special case of hematoma drainage and is discussed in detail later in this chapter.

## ABSCESS ETIOLOGY AND PATHOGENESIS

Localized pyogenic infections may develop in any region of the body and usually are initiated by a breakdown in the normal epidermal defense mechanisms, with subsequent tissue invasion by normal resident flora. Thus, an abscess is likely to be caused by the flora that are indigenous to that area. An exception is direct inoculation of extraneous organisms, such as infections that follow mammalian bites.

Staphylococcal strains, which are normally found on the skin, produce rapid necrosis, early suppuration, and localized infections with large amounts of creamy yellow pus. This is the presentation of a typical abscess. Group A beta-hemolytic streptococcal infections, on the other hand, tend to spread through tissues, causing a more generalized infection characterized by erythema and edema, a serous exudate, and little or no necrosis. This is the presentation of a typical cellulitis. Anaerobic bacteria proliferate in the oral and perineal regions; produce necrosis with profuse brownish,

foul-smelling pus [3] ; and may cause both abscess and cellulitis.

Normal skin is extremely resistant to bacterial invasion, and few organisms are capable of penetrating the intact epidermis. In the normal host with intact skin, the topical application of even very high concentrations of pathogenic bacteria does not result in infection. The requirements for infection include a high concentration of pathogenic organisms, such as occurs in the hair follicles and their adnexa; occlusion, which prevents desquamation and normal drainage, creating a moist environment; adequate nutrients; and trauma to the corneal layer, which allows organisms to penetrate. [4] Trauma may be the result of abrasions, hematoma, injection of chemical irritants, incision, or occlusive dressings that cause maceration of the skin. Foreign bodies can also potentiate these infections and decrease the number of bacteria necessary for infection. An example of this is the ubiquitous suture abscess, which frequently develops in otherwise clean wounds closed with suture material.

When favorable factors are present, normal flora of cutaneous areas can then colonize and infect the skin. The bodily area involved depends primarily on host factors. In persons performing manual labor, the arms and the hands are infected most frequently. In women, the axilla and submammary regions are frequently infected because of minor trauma from shaving and garments and because of the abundance of bacteria in these areas. IV drug users may develop infections anywhere on the body, although the upper extremities are most commonly affected. [5] [6] Deep soft tissue abscesses have also been described following attempts at access to the deep venous structures in addicts who have exhausted all peripheral venous access sites. [7] In addition, areas with compromised blood supply will be more prone to infection, because normal host cell-mediated immunity is not as available. [4] Septic emboli from endocarditis may cause abscess formation by bacteremic migration of infected material into subcutaneous (SQ) tissue.

Infections in the soft tissue often begin as a cellulitis. Some organisms cause necrosis, liquefaction, and accumulation of leukocytes and debris, followed by loculation and walling off of pus, all of which result in the formation of 1 or more abscesses. There may be involvement of the lymph tissues, producing lymphangitis and subsequent bacteremia. As the process progresses, the area of liquefaction increases until it "points" and eventually ruptures into the area of least resistance. This may be toward the skin or the mucous membrane, into surrounding tissue, or into a body cavity. If the abscess is particularly deep-seated, spontaneous drainage may occur, with persistence of a fistulous tract and the formation of a chronic draining sinus. This development, or the recurrence of an abscess that has been previously drained, should always suggest the possibility of osteomyelitis, a retained foreign body, or the presence of unusual organisms such as *Mycobacterium* or *Actinomyces.*[3]

**Bacteriology of Cutaneous Abscesses**

Meislin and coworkers [1] cultured abscesses in 135 patients, and their report typifies the bacteriology and natural history of cutaneous abscesses. Their patients received simple incision and drainage, and all subjects were followed as outpatients. Both aerobic and anaerobic cultures were taken. Most (96%) cultures were positive for bacteria (Table

40-1) (Table Not Available) .

In this series predominantly mixed aerobic bacteria were isolated in abscesses of the trunk, axilla, extremities, and the hand. In pure cultures, *Staphylococcus aureus* was found in 72% of cases. One third of the cultures from the perianal region contained only anaerobes. Mixed cultures of both aerobic and anaerobic bacteria were obtained from all sites of the body, but there was a 67% incidence of such mixed cultures from the perirectal area. Commonly isolated anaerobes included various *Bacteroides* spp, peptococci, peptostreptococci, *Clostridium* spp, *Lactobacillus* spp, and *Fusobacterium* spp.

Bacteria from abscesses in areas remote from the rectum were generally aerobic strains and were primarily indigenous microflora of the skin. *S. aureus* was the most prevalent aerobic organism; it was isolated in 24% of all abscesses.

Gram-negative aerobes were isolated infrequently from cutaneous abscesses. *Escherichia coli, Neisseria gonorrhoeae,* and *Pseudomonas* spp were rarely found. The most commonly isolated gram-negative organism was *Proteus mirabilis.* This organism was found almost exclusively in the axilla, and its presence may be related to the use of underarm deodorants. [8]

Brook and Finegold [9] studied the bacteriology of cutaneous abscesses in children. Their results closely correlate with those of Meislin and associates. [1] Brook and Finegold found aerobes (staphylococci and group A beta-hemolytic streptococci) to be the most common isolates from abscesses of the head, neck, extremities, and trunk, with anaerobes predominating in abscesses of the buttocks and perirectal sites. Mixed aerobic and anaerobic flora were found in the perirectal area, head, fingers, and nailbed area. This study found an unexpectedly high incidence of anaerobes in nonperineal abscesses. Anaerobes were found primarily either in areas adjacent to mucosal membranes, where these organisms tend to thrive (e.g., the mouth), or in areas that are easily contaminated (e.g., by sucking fingers, which causes nailbed and finger infections or bite injuries).

Parenteral drug users develop somewhat atypical abscesses. Webb and Thadepalli found anaerobes to be a major pathogen, regardless of anatomic location. [10] A more recent study of IV drug users found that skin abscesses at the site of injection contained predominantly staphylococcal and streptococcal species. Four of the 20 staphylococcal isolates were oxacillin resistant. Anaerobes and gram-negative bacilli accounted for 24% of the isolates. [11] Because IV drug use is associated with immunodeficiency syndromes, unusual isolates such as *Candida albicans*[12] and acid-fast bacilli have been obtained.

The polymicrobial nature of abscesses does not lend itself to a strict scientific interpretation of culture results, but if an unexpected or atypical organism is found in an abscess culture, the clinician should consider an underlying process not readily apparent from the history or physical examination. Most typical is finding tuberculosis or fungal isolates in immunocompromised patients (e.g., those with diabetes or acquired immunodeficiency syndrome [AIDS]). Finding *E. coli* suggests an enteric fistula or even

self-inoculation of feces in psychiatric patients or in patients with Munchausen's syndrome. Recurrent abscesses without an obvious underlying cause could indicate clandestine drug use ("skin popping").

**Special Considerations**

Parenteral drug users, insulin-dependent diabetics, hemodialysis patients, cancer patients, transplant recipients, and individuals with acute leukemias have an increased frequency of abscess formation compared with the general population. Local symptoms may not be the primary complaint, and the patient may present only with an exacerbation of the underlying disease process or an unexplained fever. These abscesses tend to have exotic or uncommon bacteriologic or fungal causes and typically respond poorly to therapy. The diabetic patient with diabetes-induced ketoacidosis should be evaluated extensively for an infectious process; a rectal examination should be included with the physical examination to rule out a perirectal abscess. This also holds true for other patients with abnormal cell-mediated immunity. The increased frequency of abscess formation in these patients and in the parenteral drug user is multifactorial. There may be intrinsic immune deficiencies in all these patients; they have an increased incidence of *Staphylococcus* carriage, and they have frequent needle punctures which allow access by pathogenic bacteria.

It is important to note that a substantial percentage of abscesses in parenteral drug users are sterile and are the result of the injection of necrotizing chemical irritants. Drug users frequently use veins of the neck and the femoral areas, producing abscesses and other infectious complications at these sites. Any abscess near a vein of the antecubital fossa or dorsum of the hand should alert the physician to possible IV drug use; however, substance users may also inject directly into the skin ("skin popping"), causing cutaneous abscesses away from veins (Fig. 40-1) .

The presence of a foreign body may serve as a nidus for abscess formation. A history of a possible foreign body at the site of an abscess should be sought. Because IV drug users frequently break needles off in skin toughened by multiple injections, the clinician should maintain a high index of suspicion for retained needle fragments. If an abscess is recurrent or if the patient is a known or suspected IV drug user, radiographs or other techniques should be considered to search for foreign bodies.

## MANIFESTATIONS OF ABSCESS FORMATION

The diagnosis of cutaneous abscess formation is usually straightforward. The presence of a fluctuant mass in an area of induration, erythema, and tenderness is clinical evidence that an abscess exists. An abscess may appear initially as a definite tender soft tissue mass, but in some cases the presence of a distinct abscess may not be readily evident. If the abscess is quite deep, as is true of many perirectal, pilonidal, or breast abscesses, the clinician may be misled by the presence of only a firm, tender, indurated area without a definite mass. To avoid misdiagnosis of an early abscess, one may aspirate the suspicious infected area with a needle and syringe to confirm the presence of pus. [18] This approach also may help identify a mycotic aneurysm or an inflamed lymph node simulating an abscess. A specific entity that is commonly mistaken

for a discrete abscess is the sublingual cellulitis of Ludwig's angina (see Chapter 69) . Cellulitis and abscess formation may lead to bacteremia and sepsis, especially in the immunocompromised patient, and the presenting complaints may draw attention from the primary focus of infection

## LABORATORY FINDINGS

*Laboratory tests offer no specific guidelines for therapy of a cutaneous abscess and are not generally indicated.* An exception would be a blood or urine glucose determination to assess diabetes in patients with appropriate clinical scenarios. An abscess may produce leukocytosis, depending on the severity and duration of the abscess process; however, the majority of patients with an uncomplicated cutaneous abscess will have a normal complete blood cell (CBC) count and will not experience fever, chills, or malaise. The presence or absence of leukocytosis has virtually no diagnostic or therapeutic implications.

*Gram stain is not indicated in the care of uncomplicated simple abscesses.* Patients who appear "toxic" or immunocompromised and those patients who require prophylactic antibiotics (see Prophylactic Antibiotics) may benefit from Gram stain and cultures. Gram stain results have been shown to correlate well with subsequent culture results, and in compromised hosts the test can be used to direct antibiotic choice. [18] [19] Anaerobic infections should be suspected when multiple organisms are noted on Gram stain, when a foul odor is associated with the pus, when free air is noted on radiographs of the soft tissue, or when no growth is reported on cultures." [10]

In uncomplicated abscesses, *routine culture is unnecessary* because of the expected prompt response to surgical therapy and the polymicrobial nature of abscess formation. However, in complicated cases or in immunosuppressed patients, an abscess should be cultured. The information obtained may later be useful if there is poor response to the initial surgical drainage, secondary spread of the infection, or the occurrence of bacteremia. [3] If one takes a culture, it is best to aspirate the pus with a needle and syringe *before* incision and drainage. Material should be cultured for aerobic and anaerobic bacteria. The finding of a "sterile" culture in an abscess that has been cultured with a standard cotton swab *after* incision is frequently the result of improper anaerobic culture techniques. As a side note, there is a general misconception that foul-smelling pus is a result of *E. coli.* This foul odor is actually caused by the presence of anaerobes; the pus of *E. coli* is odorless.

## INDICATIONS AND CONTRAINDICATIONS

Surgical incision and drainage is the *definitive treatment* of a soft tissue abscess. [18] Antibiotics alone are *ineffective* in the face of a localized collection of pus. The drainage of a suppurative focus results in a marked improvement in symptoms and rapid resolution of the infection in uncomplicated cases. Premature incision before localization of pus will not be curative and theoretically may be deleterious, because extension of the infectious process and bacteremia from manipulation can result. In some cases the application of heat to an area of inflammation may ease pain, speed resolution of the cellulitis, and facilitate the localization and accumulation of pus. It must be stressed that

nonsurgical methods are not a substitute for surgical drainage and should not be continued for >24 to 36 hours before the patient is reevaluated. Diagnostic needle aspiration is recommended if one is unsure of pus localization.

# ANCILLARY ANTIBIOTIC THERAPY

The use of antibiotics remains controversial for both prophylaxis and treatment of cutaneous abscesses. As an overview, there are no data that definitively demonstrate the need for antibiotic therapy in conjunction with incision and drainage of uncomplicated cutaneous abscesses in healthy, immunocompetent patients without valvular heart disease. The specific value of concomitant antibiotics in the immunocompromised patient, while intuitively attractive, is unclear. Patients with risk factors for endocarditis comprise a third group of patients. Because of the concern of inducing a bacteremia by manipulation of infected tissue, parenteral antibiotics are commonly given *prior to the incision-and-drainage procedure* in patients at risk from such bacteremia. Transient bacteremia has been documented (incidence range, 7 to 50%) following manipulation of noninfected tissue (brushing teeth, sigmoidoscopy, Foley catheter, rectal examination) and following simple incision and drainage of abscesses. In a report by Fine and colleagues, 6 of 10 patients with cutaneous abscesses were noted to have positive blood cultures immediately following incision and drainage, whereas all cultures were negative before the procedure. [20] Blick and associates evaluated the use of prophylactic antibiotics in abscesses managed in the operating room under general anesthesia. Three of 19 patients developed bacteremia after the procedure. A similar group treated with parenteral antibiotics before the procedure had only 1 patient with a positive blood culture. That culture yielded an organism resistant to the prophylactic antibiotic used. [21]

## Prophylactic Antibiotics

### Prophylaxis for Endocarditis

The precise risk for endocarditis following incision and drainage of cutaneous abscess is unknown, and it is impossible to predict which patients will develop this infection or which particular procedure will be responsible. However, since bacteremia clearly occurs with manipulation of infected tissue, it is generally agreed that those patients at risk forcardiac complications related to transient bacteremia should be treated with appropriate antibiotics within the hour preceding the procedure. The transient bacteremia secondary to abscess drainage is probably of no concern in otherwise healthy, immunocompetent individuals without valvular heart disease.

No adequate controlled trials of antibiotic regimens appear in the literature, and data predicting the incidence of endocarditis in patients with preexisting cardiac disorders are lacking, thereby making the decision regarding antibiotic prophylaxis quite arbitrary. However, the Committee on Rheumatic Fever and Infective Endocarditis of the American Heart Association (AHA) has recommended that prophylactic antibiotics be given prior to incision and drainage of infected tissue in patients who have *cardiac lesions that place them at high risk for developing endocarditis.*[22] Table 40-2 (Table Not Available) lists conditions that may warrant antibiotic coverage. Durack also

recommends prophylactic antibiotics for a variety of procedures known to have a significant incidence of bacteremia, including incision and drainage of cutaneous abscesses. [23] Interestingly, the antibiotic regimen suggested by the AHA (Table 40-3) (Table Not Available) does not include antibiotics effective against many organisms commonly found in some cutaneous abscesses, specifically *S. aureus,* but is mainly directed against *Streptococcus viridans* and enterococcus. These latter organisms have a predilection for valvular infection. The AHA recommends the same prophylaxis for incision of soft tissue infection that is recommended for genitourinary procedures, but with additional consideration of therapy "directed against the most likely pathogen." [24]

Two clinical situations deserve special note. Because of the frequent incidence of endocarditis and previous valvular damage in the patient who uses IV drugs, prophylactic antibiotics may be indicated prior to the incision and drainage of abscesses in these patients. Clearly, any patient with a documented history of endocarditis must receive prophylactic antibiotics prior to the incision-and-drainage procedure. Because cutaneous abscesses may result from active endocarditis and prophylactic antibiotics may obscure subsequent attempts at identifying an etiologic organism, 2 or 3 blood cultures (aerobic and anaerobic) should be obtained from those at risk for endocarditis prior to antibiotic therapy. Patients with the diagnosis of mitral valve prolapse have traditionally been included for treatment with prophylactic antibiotics. The indication for this is unclear. The risk of an allergic reaction may outweigh the benefits of treatment in this group, [25] and clinical judgment is required. Kaye [26] suggests prophylaxis only for patients who have a holosystolic murmur secondary to mitral valve prolapse.

---

**TABLE 40-3** -- Prevention of Bacterial Endocarditis: Regimens for Genitourinary/Gastrointestinal Procedures *
*From Dajani AS, et al: Prevention of bacterial endocarditis. Circulation 83:1174, 1991.*

(Not Available)

---

* In the case of abscess drainage, include antibiotics directed against most likely pathogen if not included in this regimen.

**Prophylaxis for Bacteremia in Other Conditions**

Immunocompromised patients have not been adequately studied, but this is a subgroup that intuitively may benefit from prophylactic antibiotics. In contrast to patients with endocarditis risks, immunocompromised patients are at risk for developing septicemia secondary to a brief bacteremia. Since IV drug users have a high incidence of human immunodeficiency virus (HIV)-related disease, the treating physician must anticipate various degrees of immunodeficiency in these patients. Clinical judgment must determine the use of antibiotics in these situations.

No specific guidelines have been offered for the antibiotic regimen used prior to incision and drainage of infected cutaneous tissue in patients at risk for conditions *other than endocarditis*. Choice of antibiotics is guided by the organism anticipated to cause the bacteremia. Although the location of the abscess will give some clue to the organism involved, most abscesses contain multiple strains of bacteria. Not all bacteria are potent pathogens, so their mere presence does not predict their role in subsequent morbidity. Because *Staphylococcus* continues to be a significant organism in this setting, a broad-spectrum anti-staphylococcal drug is indicated. [5] Prophylaxis should consist of a single IV dose given a half hour prior to incision and drainage. A first-generation cephalosporin or penicillinase-resistant penicillin is a good initial choice. Vancomycin may also be considered. Others may prefer cefazolin (Ancef, Kefzol), 1 g, IV, given a half hour before surgery. This regimen covers staphylococcal and streptococcal species, many gram-negative organisms, and many anaerobes.

Although not widely used in the United States, parenteral antibiotics have been used to "sterilize" the abscess cavity following curettage in Great Britain. The concentration of clindamycin in the abscess cavity has been shown to be equivalent to the concentration of antibiotics found in the blood. [21] Some British articles report success with "primary closure under antibiotic coverage," although other British reports continue to recommend the traditional procedure.  One limitation to the abscess curettage technique is the need for general anesthesia, because the performance of curettage is extremely painful.

**Therapeutic Antibiotics**

In contrast to prophylaxis prior to surgery, the routine use of oral antibiotics following incision and drainage of simple cutaneous abscesses in otherwise healthy patients with no immunocompromise appears to have virtually no value, and their empiric use cannot be scientifically supported. Llera and Levy [2] performed a randomized double-blind study to compare outcomes of patients treated with a first-generation cephalosporin following drainage of cutaneous abscesses in the ED. They found no significant difference in clinical outcome between the 2 groups and concluded that antibiotics are unnecessary in individuals with normal host defenses. This confirmed previous less well controlled studies. It should be noted that high-risk patients were often excluded from these studies. The immunocompromised patient has not been adequately studied in this situation and is therefore often given antibiotics empirically, but this practice, while common, has not been supported by rigorous prospective studies.

Patients with cutaneous abscesses often have concomitant disease processes that may warrant the use of parenteral or oral antibiotics. Cellulitis or lymphangitis often accompany abscesses, and therapeutic antibiotics may have value under these circumstances, but again, this concept is not well addressed in the literature. Meislin noted that pathogen identification in cases of cellulitis without abscess can be difficult, and empiric antibiotics may be helpful. [35] IV drug users who present with an abscess and fever require parenteral antibiotic therapy after blood cultures have been drawn until subacute bacterial endocarditis can be ruled out. [36] Obviously, patients who are clinically septic require immediate IV antibiotics as well as aggressive surgical drainage

of pus.

As a general guideline, therapeutic antibiotics should be given to all immunocompromised patients (e.g., patients with AIDS or diabetes, patients receiving chemotherapy or steroids, transplant recipients, and alcoholic patients) and to the immunocompetent patient with "significant" cellulitis, lymphangitis, or systemic symptoms, such as chills or fever. Although it has not been studied, it makes sense to also give *antibiotics prophylactically, before surgery, to all patients who will obviously be given therapeutic antibiotics.* As with prophylactic antibiotics, a first-generation cephalosporin or semisynthetic penicillin is a reasonable therapeutic choice unless the specific abscess site dictates alternative therapy. The ideal duration of therapeutic antibiotics is unknown. As a general guideline, immunocompromised patients should receive antibiotics for 5 to 7 days and immunocompetent patients, for 3 to 5 days after the procedure, depending on the severity of the condition and clinical response.

Facial abscesses should be handled carefully and checked frequently. Any abscess above the upper lip and below the brow may drain into the cavernous sinus, and thus manipulation may predispose to septic thrombophlebitis of this system. Treatment with antistaphylococcal antibiotics and warm soaks following incision and drainage has been recommended pending resolution of the process. Areas *not* in this zone of the face can be treated in a manner similar to that used for other cutaneous abscesses.

## INCISION-AND-DRAINAGE PROCEDURE

### Procedure Site

Definitive incision and drainage of soft tissue abscesses is performed in either the ED or the operating room (OR). The choice of the locale for the procedure depends on a number of important factors. Location of the abscess may dictate management in the OR. Large abscesses or abscesses located deep in the soft tissues require a procedure involving a great degree of patient cooperation, which may only be possible under general or regional anesthesia. Proximity to major neurovascular structures, such as in the axillae or antecubital fossa, may necessitate specific management. Infections of the hand (with the exception of distal finger infections) have traditionally been managed in the OR because of the many important structures involved and the propensity for limb-threatening complications.

Lack of adequate anesthesia is the most common limiting factor in ED incision and drainage. If the clinician believes the abscess cannot be fully incised and drained because of inadequate anesthesia, the patient should be taken

to the OR for management under general anesthesia. In addition to limiting proper drainage, it is inhumane and unethical to subject a patient to extreme pain when alternatives are available.

When abscesses are drained in the ED, some centers prefer to use a special area of

the ED to avoid contamination of general treatment rooms.

**Equipment and Anesthesia**

A standard suture tray provides adequate instruments if a scalpel and packing material are added. Although sterility is impossible during the procedure, one should avoid contamination of surrounding tissue. Some physicians prefer to use an obligatory skin scrub with an antiseptic solution, but the value of this step is dubious.

It is often quite difficult to obtain local anesthesia by direct infiltration because of the poor function of local anesthetic agents in the low pH of infected tissue. Furthermore, the distention of sensitive structures by a local injection is quite painful and hence poorly tolerated by most patients. Skin anesthesia is usually possible, but total anesthesia of the abscess cavity itself generally cannot be achieved. If a regional block can be performed (see Chapters 32 and 33) , this type of anesthesia is preferred. Alternatively, a field block may be used. It should be noted that infected tissue is very vascular, and local anesthetics are quickly absorbed. Strict adherence to maximum safe doses of the local anesthetic is required.

The skin over the dome of an abscess is often quite thin, making skin anesthesia difficult. If a 25-ga needle is carefully used, one can often inject the dome of the abscess subcutaneously. The anesthetic solution spreads over the dome through the SQ layers into the surrounding skin and provides excellent skin anesthesia. If the needle is in the proper plane (best accomplished by holding the syringe parallel, rather than perpendicular, to the skin), the surrounding skin blanches symmetrically during infiltration without having to reposition the needle (Fig. 40-2 *A*). In the extremely anxious or uncomfortable patient, the judicious use of preoperative sedation (see Chapter 35) with IV opioids and sedatives or nitrous oxide makes the procedure easier for both patient and physician. If adequate anesthesia cannot be obtained and pain limits the procedure, the patient should be treated under general anesthesia.

Some clinicians recommend the use of topical ethyl chloride or Fluori-Methane spray for the initial skin incision, but the pain relief offered by this agent is variable and fleeting. Ethyl chloride is also highly flammable. These vapocoolant sprays may be useful to provide momentary anesthesia for local anesthetic injection or for the initial skin incision if the injection or incision is made immediately after blanching of the skin. In general, however, this agent is a poor choice for a stand-alone anesthetic agent for all but the smallest of superficial abscesses (e.g., purulent folliculitis).

**Incision**

One should make all incisions conform with skin creases or natural folds to minimize visible scar formation (Fig. 40-3) (Figure Not Available) . Extreme care should be taken in such areas as the groin, the posterior knee, the antecubital fossa, and the neck, so that vascular and neural structures are not damaged.

A No. 11 or 15 scalpel blade is used to nick the skin over the fluctuant area, and then a

simple linear incision is carried the *total length of the abscess cavity* (see Fig. 40-2 *B*). This will afford more complete drainage and will facilitate subsequent breakup of loculations. A cruciate incision or an elliptical skin *excision* is to be avoided in the routine treatment of cutaneous abscess. The tips of the flaps of a cruciate incision may necrose, resulting in an unsightly scar. A timid "stab" incision may produce pus but is generally not adequate for proper drainage. It should be emphasized that *the scalpel is used only to make the skin incision and is not used deep in the abscess cavity*.

Exceptions to this rule regarding aggressive incision are abscesses in cosmetic areas, in areas under significant skin tension (e.g., extensor surfaces), or in areas with extensive scar tissue (e.g., sites of multiple prior drainage procedures). In these special circumstances, a stab incision or simple aspiration alone may be initially attempted, with the goal of limiting tissue injury and resultant scar formation. Use of this less aggressive approach requires that the patient be counseled that multiple decompressions (e.g., via needle aspiration) or delayed aggressive incision and drainage may be required. The abscess will need reassessment in 24 to 48 hours to determine the need for additional intervention.

**Wound Dissection**

Following a standard incision, the operator should probe the depth of an abscess to assess the extent of the abscess and ensure proper drainage by breaking open loculations (see Fig. 40-2 *C*). An ideal instrument for this procedure is a hemostat wrapped in gauze (or a cotton swab for small abscesses) that is placed into the abscess and swirled around to all sides of the cavity (see Fig. 40-2 *D*). Traditionally the operator's gloved finger has been suggested as an ideal way to assess the depth of the abscess cavity and to break up loculations, but *this is a potentially dangerous practice that should be avoided* unless it is certain that the abscess contains no sharp foreign body. Of particular concern is the abscess caused by skin popping or IV drug use. Such patients have a high incidence of HIV infection, and occasionally these abscesses harbor broken-off needle fragments (Fig. 40-4) . One is often surprised at the depth or extent of an abscess during probing. Sharp curettage of the abscess cavity is usually not required and may produce bacteremia. [21] Although tissue probing is usually the most painful aspect of the technique and total local anesthesia is difficult to obtain, this portion of the procedure should not be abbreviated. If the procedure is limited because of pain, the use of appropriate analgesia/anesthesia is mandated.

**Wound Irrigation**

Following the breaking up of loculations, some physicians advocate copious irrigation of the abscess cavity with normal saline to ensure adequate removal of debris from the wound cavity. Although it may seem intuitively to be a helpful step, irrigation of the abscess cavity has not been experimentally demonstrated to significantly augment healing or affect outcome. Hyperemic tissue may bleed profusely, but bleeding usually stops in a few minutes if packing is used. Abscesses of the extremities can be drained during the use of a tourniquet to provide a bloodless field.

**Packing and Dressing**

After irrigation, a loose packing of gauze or other material is placed gently into the abscess cavity to prevent the wound margins from closing and to afford continued drainage of any exudative material that may otherwise be trapped. The packing material should make contact with the cavity wall so that upon removal, gentle debridement of necrotic tissue will spontaneously occur. A common error is to attempt to pack an abscess too tightly with excessive packing material. In essence, the pack merely keeps the incision open, and its main purpose is not to absorb all drainage--a dressing accomplishes this goal. Care must be exercised to ensure that the packing does not exert significant pressure against the exposed tissue and lead to further tissue necrosis. Some prefer to use plain gauze, some use gauze soaked in povidone-iodine, and some use gauze impregnated with iodine (iodoform). For large abscess cavities, gauze pads (without cotton backing) are ideal packing. *If gauze pads are used, the number of pads placed in the wound should be counted and charted--ideally, the corner of each pad should exit from the wound.* The clinician must ensure that all gauze pads will be removed when the packing is changed or discontinued. More commonly, thin (0.6 to 1.2 cm) packing strip gauze, either plain or iodoform, is used. The iodoform gauze may sting the patient for a few minutes after it is inserted. The value of antibiotic-impregnated gauze is uncertain.

An absorbent gauze dressing should be placed over the packed abscess, or, if an extremity is involved, a lightly wrapped circumferential dressing should be used. Generous amounts of dry gauze are used over the packing to soak up any drainage or blood. The affected part should be splinted if possible, and elevation should be routine. The dressing/splint should not be disturbed until the first follow-up visit. Drainage relieves most of the pain of an abscess, but postoperative analgesics may be required.

Following treatment, packing is often changed periodically. Most patients require a repeat visit to the physician for packing change, but if the original packing is to be removed and not replaced (paronychia, hair follicle abscess), selected patients may remove the packing and perform their own wound care totally at home.

## FOLLOW-UP CARE

Reevaluation of a drained abscess should occur in 1 to 3 days, depending on a number of parameters.  Most lesions are reevaluated 48 hours following the procedure, with the first packing change occurring at this time. Some wounds warrant closer monitoring. Diabetic patients or other patients with impaired healing capacity, mental impairment, or physical disabilities may require a home care nurse or admission for more frequent wound care/packing changes. Wounds that are at high risk for complications, such as those about the face or hands or those with significant cellulitis, require close follow-up, ideally by the same examiner. The patient should be encouraged to play an active role in wound care. During the first follow-up visit, the compliant and able patient should be taught to perform packing and/or dressing changes. If this is anatomically impossible, a

friend or family member can be instructed in the technique.

The technique of packing change is usually one of personal preference. It should be emphasized that patients often fear a repeat visit and expect significant pain with subsequent wound care, especially if the initial incision drainage was difficult. Therefore, the specifics of packing change should be addressed prior to release home after the initial drainage procedure. Some clinicians suggest an oral opioid be taken 30 to 40 minutes before the next visit or use local anesthesia or parenteral analgesia if significant pain is anticipated. Removal of packing material is often painful, but if the packing is moistened with saline prior to removal, it may be less traumatic. Once the packing is removed, the wound is inspected for residual necrotic tissue. The cavity may be irrigated with saline prior to replacing the pack if there is significant exudate, but this is often not required because the packing absorbs most debris.

The frequency of packing or dressing changes is also clinically guided. Some wounds require multiple packing changes, while other wounds require only the initial packing. In all facial abscesses, the packing should be removed after only 24 hours, at which time warm soaks should be started. Wounds large enough to require packing should be repacked at least every 48 hours (occasionally daily for the first few visits) until healing continues in a deep to superficial direction. Large wounds that are allowed to close superficially will create an unsterile dead space that will potentiate the formation of recurrent abscesses. After the first few days (and in the motivated and compliant individual), an alternative to packing is to have the patient clean the base of the abscess 3 times a day with cotton swabs soaked in peroxide (Fig. 40-5) . This promotes drainage, produces gentle debridement, and keeps the incision open. Following cleaning, the abscess can be irrigated with tap water and a dry dressing applied.

In general, once healthy granulation tissue has developed throughout the wound and a well-established drainage tract is present, the packing may be discontinued. The patient should then be instructed to begin warm soaks of the wound. [18] Gentle hydrostatic debridement may be performed by the patient in the shower at home. With this procedure the patient holds the skin incision open and directs the shower or faucet spray into the abscess cavity. Wet-to-dry normal saline dressing changes should then follow until healing is completed. When all signs of infection (e.g., erythema, drainage, pain, and induration) have resolved and healthy granulation tissue is present, the patient may be discharged from medical care.

Complicated wounds that require prolonged care are best followed by a single physician and should not be routinely referred back to the ED. These patients should receive early referral to their primary care physician or specialist. Wounds in cosmetically important areas may require revision once healing is complete. Patients should be informed of this possibility early on in their care.

In selected cases in which extensive or prolonged drainage occurs or in patients who are unable to return for proper follow-up care, a catheter system of drainage may be preferred. [37] [38] Following incision, a balloon-tipped or flared-tip catheter is placed into the abscess cavity, and pus is allowed to drain continuously through the catheter lumen. This technique has been most successful in pilonidal and Bartholin gland abscesses,

but the technique is applicable to any abscess *not* on the face.

## SPECIFIC ABSCESS THERAPY

### Staphylococcal Diseases

The *Staphylococcus* bacterium is a ubiquitous pathogen that frequently colonizes the nose, skin, perineum, and gut. The umbilicus of neonates is also commonly colonized. It grows on the skin and thrives particularly well in hair follicles, causing boils (furuncles), wound infections, and occasionally carbuncles. The pathogenesis of staphylococcal disease is a complex host-bacteria interaction. *S. aureus* invades the skin by way of the hair follicles or an open wound and produces local tissue destruction followed by hyperemia of vessels. Subsequently, an exudative reaction occurs, during which polymorphonuclear cells invade. The process then extends along the path of least resistance. The abscess may "point" or form sinus tracts. The process can disseminate by invasion of vessels and thus can infect other organs. Most cases of staphylococcal osteomyelitis, meningitis, and endocarditis occur by this mechanism. [39] [40]

*Folliculitis* represents a small abscess occurring at the root of a hair. Local measures, including warm compresses and antibacterial soaps and ointments, are the usual treatment, but systemic antibiotics may be required if multiple sites are involved or the patient is a chronic staphylococcal carrier. *Furuncles*, or boils, are acute circumscribed abscesses of the skin and SQ tissue that most commonly occur on the face, the neck, the buttocks, the thigh, the perineum, breast, or axilla. *Carbuncles* are aggregates of interconnected furuncles that frequently occur on the back of the neck (Fig. 40-6) . In this area the skin is thick, and extension therefore occurs laterally rather than toward the skin surface. Carbuncles may attain large size and can cause systemic symptoms and complications. They are found in increased frequency in diabetics, and all patients with a carbuncle should be evaluated for this underlying disease. Treatment should consist of surgical drainage and administration of systemic antibiotics. Large carbuncles may be impossible to drain adequately in the ED. Carbuncles usually consist of many loculated pockets of pus, and simple incision and drainage are often not curative. Occasionally, wide excision and skin grafting are required.

Most cases of recurrent staphylococcal skin infections are caused by autoinfection from existing skin lesions or nasal reservoirs. Prevention is directed at eliminating the organism. This is accomplished by application of bacitracin to the nares and by good hygiene, including frequent cleansing with antibacterial soap. If these measures are unsuccessful, then systemic oral antistaphylococcal treatment is instituted for 2 to 3 weeks. Detection and treatment of infection in family members may be necessary.

*S. aureus* produces the ubiquitous suture abscess. A *suture abscess* is often misdiagnosed as a wound infection, but in fact, it is a local nidus of inflammation and/or infection caused and potentiated by suture material. Such an abscess usually appears after sutures have been present for at least 3 to 5 days, with single or multiple discrete areas of redness and tenderness noted at the site of suture penetration of the skin. Simply removing the suture (a drop of pus may be expressed) and providing warm compresses and topical antibiotic ointment is usually all that is required. Wide opening

of the wound and systemic antibiotics are seldom required. When the suture is buried, a small incision should be followed by probing of the wound with a small hook or bent needle (see Chapter 39) to snare the suture for its removal.

## Hidradenitis Suppurativa

Hidradenitis suppurativa (Greek hidros = sweat, aden = gland) is a chronic, relapsing, inflammatory disease process affecting the apocrine gland that primarily involves the axilla, the inguinal region, or both. [41] The condition results from occlusion of the apocrine ducts by keratinous debris, which leads to ductal dilation, inflammation, and rupture into the SQ area. Secondary bacterial infection ensues, leading to abscess formation and scarring. This chronic recurring process leads to draining fistulous tracts, which involve large areas and are not amenable to simple incision-and-drainage procedures. [42]

Genetic factors may play some role in hidradenitis suppurativa. Family history is often significant in these patients. Fitzsimmons has proposed a single dominant gene transmission. [43] Blacks appear to have an increased incidence compared with whites. Although certain groups appear to be predisposed to this condition, the precipitating factor for this process is unclear. Because apocrine glands become active during puberty, it is rare to find hidradenitis suppurativa in the pediatric population. [41] Women are affected more frequently than men, for uncertain reasons. Shaving and depilation have frequently been suggested as causes of this discrepancy; however, this theory was not supported in a study that compared the frequency of these behaviors in patients with hidradenitis suppurativa and a group of controls. [44] Obesity is associated with an increased incidence of the disease. [45] Excessive dermal folds provide dark, wet, and warm areas, which are ideal for the proliferation of the bacteria that are needed for this infectious process. Antiperspirants and deodorants may decrease wetness and bacterial overgrowth, but they have been known to produce inflammatory responses, which could exacerbate the disease process.

The bacteriology of acute abscess formation in hidradenitis suppurativa reflects organisms seen in other soft tissue abscesses. *Staphylococcus* is the most commonly isolated organism, with *E. coli* and beta-hemolytic *Streptococcus* being other important pathogens. In the perineal region, enteric flora are often found. Many of these abscesses have multiple isolates, and anaerobic bacteria are frequently found.

Hidradenitis suppurativa begins as a single inflammatory event involving an apocrine gland, which progresses to frank suppuration and at this stage is no different from a simple furuncle. The clinical entity is distinguishable only in its chronic scarring phase. By then the lesion exhibits multiple foci coupled with areas of induration and inflammation that are in various stages of healing. Progression of the process reveals coalesced areas of firm, raised violaceous dermis. The lesion is usually markedly tender. This disease classically

involves the axilla and perineal or inguinal region, although multiple sites are often involved.

Initial outpatient management usually involves intervention in an acute suppurative lesion. Any fluctuant area requires drainage as described in the section on general abscess management. In cases of extensive cellulitis, a broad-spectrum, antistaphylococcal antibiotic should be used. Unfortunately, hidradenitis suppurativa is often not readily cured with localized incision and drainage. The chronic nature of the disease produces multiple areas of inflammation and SQ fistulous tracts that induce routine recurrences. The patient must be informed of this rather unfavorable prognosis and should be referred to a dermatologist or surgeon for long-term care.

Milder forms of the disease are initially treated with conservative measures. Many different approaches have been tried, with numerous case reports and case series noted in the literature. Unfortunately, few controlled studies have been performed. Patients are often counseled to lose weight, refrain from shaving, stop using deodorants, and improve personal hygiene. The benefits of these efforts are unknown. Oral antistaphylococcal antibiotics are most commonly used, with varying results. [46] There have been reports of success with topical clindamycin, [47] isotretinoin,  and laser therapy, [50] but these treatments have not been studied in a controlled setting and require further investigation. Dermal infection results from breakdown of the normal host defense mechanism, which occurs with irritation, traumatic injury, or inflammation, coupled with the availability of concentrated opportunistic bacteria. Therefore, the physician must institute therapies that will decrease bacterial availability without causing further injury to the affected dermis.

Advanced stages of the disease are routinely managed with wide or local excision and primary or delayed closure.  Despite this radical approach, recurrences do occur. [54] Patients must be counseled about the likelihood of recurrence before the procedure.

**Breast Abscess**

*Postpartum mastitis* occurs in 1 to 3% of nursing mothers within the first 2 to 6 weeks after delivery. The infection is usually precipitated by milk stasis following weaning or missed feedings. The cause is usually bacterial invasion through a cracked or abraded nipple by *S. aureus* or streptococci originating from the nursing child. Manifestations are redness, heat, pain, fever, and chills. Treatment consists of antistaphylococcal antibiotics, continued breast emptying with a breast pump, and application of heat. It is important to encourage continued breast emptying to promote drainage. Nursing can be continued with the noninfected breast, although passage of the antibiotics through the breast milk may result in some infant diarrhea. Cellulitis may progress to frank abscess formation. These patients may be quite ill and may appear toxic. Abscess formation complicating postpartum mastitis usually necessitates OR management, because the area is extremely tender, and adequate local anesthesia is difficult to obtain. Strict adherence to nipple hygiene to avoid cracks or inflammation is helpful in prophylaxis.

Surprisingly, most breast abscesses occur in women who are not in the puerperium, and have been termed *nonpuerperal breast abscesses*. [55] Scholefield and coworkers reviewed 72 breast abscesses over a 10-year period and noted that only 8.5% of

patients were in the puerperium. [56] These lesions have been classified anatomically.

*Peripherally located abscesses* are most commonly caused by *Staphylococcus* and respond well to traditional conservative incision and drainage. [56] Superficial abscesses in the SQ tissue may be drained under local anesthesia by means of an incision that radiates from the nipple (Fig. 40-7 (Figure Not Available) *A*).

*Periareolar abscesses* exhibit a more troublesome profile. The microflora often include multiple organisms, and anaerobic bacteria are important isolated pathogens. These infections may be the result of occluded and inflamed mammary ducts. Chronic disease may lead to ductal ectasia that provides a nidus for infection. The deeper and more extensive

**Figure 40-7** (Figure Not Available) *A,* A superficial breast abscess may be drained with a linear incision that radiates from the nipple. *B* and *C,* Diagrams of intramammary abscess *(B)* and retromammary abscess *(C).* Both require drainage under general anesthesia. The abscess itself may not be fully appreciated if it is deep seated, and the mistaken diagnosis of cellulitis may be made. (Redrawn from Wolcott MW: Ferguson's Surgery of the Ambulatory Patient. 5th ed. Philadelphia, JB Lippincott, 1974. Reprinted with permission.)

intramammary abscess appears as a generally swollen, tender breast (Fig. 40-7 (Figure Not Available) *B*). Fluctuance is not always obvious, since the abscess is located in the mammary tissue itself. Maier and colleagues reported that more than half of their series of 96 patients had nipple inversion due to chronic scarring. [59] These intramammary infections are complex and require incision and drainage under general anesthesia. Dixon has reported successful treatment with repeated aspiration in both lactating [60] and nonlactating patients. [61] This approach is probably best left to surgeons able to see such patients on a regular scheduled basis to track the progress of abscess resolution.

A *retromammary abscess* lies in the undersurface of the breast between the breast and the chest wall (Fig. 40-7 (Figure Not Available) *C*). Fluctuance may be difficult to appreciate because of the depth of the infection. Drainage under general anesthesia is required.

Recurrent abscesses are a common, troublesome complication, occurring in up to 38% of primary periareolar abscesses treated with standard incision, drainage, and antibiotics. [62] These cases require total excision of the involved area and necessitate the care of a general surgeon and further intraoperative management. It may be difficult to diagnose a breast abscess in the early stages, when cellulitis predominates. In equivocal cases antibiotics may be curative, but when pus is present, incision and drainage must be performed.

**Bartholin Gland Abscess**

The Bartholin glands (vestibular glands) are secretory organs located at the 5 and 7 o'clock positions on each side of the vestibule of the vagina. Asymptomatic cysts frequently occur from duct blockage and retention of secretions. Chronic low-grade inflammation from gonococcal infections has been implicated as an etiologic factor in

cyst formation, but occasionally frank abscess formation results. Such patients present with swollen and tender labia and a fluctuant, grape-sized mass that may be palpated between the thumb and the index finger. *Neisseria gonorrhoeae* organisms are infrequently cultured from the abscess cavity, and various anaerobes, especially *Bacteroides* species and other colonic bacteria, are usually found. It is reasonable to take cervical and anal cultures for gonorrhea from women with Bartholin gland abscesses because of the association of these infections with venereal disease, but one need not routinely treat patients for gonorrhea.

Word [63] has described an effective treatment of Bartholin gland abscess with a single-barreled, sealed-stopper, balloon-tipped catheter that may obviate the need for marsupialization (Fig. 40-8 (Figure Not Available) *A* and *B*). In his original description, Word reports only 2 recurrences in 72 lesions, both of which were successfully treated with a second catheter; no patient required marsupialization. The procedure involves fistulization of the duct cavity by a catheter, which acts as a foreign body. While not a standard incision-and-drainage procedure, the technique permits continued drainage of the Bartholin gland.

Following a small incision into the mucosa, the scalpel or a hemostat is used to puncture the abscess cavity proper (Fig. 40-9) . It is helpful to stabilize the abscess with the thumb and forefinger to ensure entrance into the abscess. Care is required to make a stab incision only large enough to accommodate the catheter and small enough to prohibit the inflated balloon from being extruded. Once the abscess has been entered (signaled by the free flow of pus), the deflated balloon is placed in the abscess cavity. Using a 25-ga needle to minimize the hole in the stopper, the balloon is then filled with 2 to 4 mL of water (not air). Persistent pain indicates that too much fluid has been used. The device is left in place for 6 to 8 weeks to allow for fistula formation, so follow-up is required. If the catheter falls out prematurely, it should be quickly replaced to fulfill the 6 to 8 weeks needed for fistulization. This is an interesting technique that even allows for sexual intercourse with the catheter in place.

The initial treatment of a Bartholin gland abscess may also include simple incision and drainage. The abscess is packed for 24 to 48 hours, and sitz baths are started after the first revisit. Broad-spectrum antibiotics are helpful if there is significant cellulitis or actual abscess formation has not yet occurred, but these agents are not required following routine incision and drainage.

It is preferable to make the drainage incision on the mucosal surface rather than on the skin surface. The incision is made over the medial surface of the introitus on a line parallel to the posterior margin of the hymenal ring. The abscess cavity is slightly deeper than most cutaneous abscesses, and one must be certain to enter the actual abscess cavity to achieve complete drainage. This is most easily accomplished if one inserts a hemostat through the mucosal incision and spreads the tips of the instrument in the deeper soft tissue. If the abscess recurs, more definitive therapy in the form of marsupialization or complete excision of the gland may be required, but these procedures are not performed initially. Because recurrence is common with simple incision and drainage, some authorities suggest definitive surgery routinely following the

first infection, whereas others prefer to wait until a recurrence is documented.

**Pilonidal Abscess**

Pilonidal sinuses are common malformations that occur in the sacrococcygeal area. The etiology of the sinus formation is unclear, but the malformation may occur during embryogenesis. Pilonidal cyst formation is thought to be secondary

to blockage of a pilonidal sinus. The result of pilonidal sinus obstruction is repeated soft tissue infection, followed by drainage and partial resolution with eventual reaccumulation. The blockage is most commonly the result of hairs in the region, and the lesion may in part be a foreign body (hair) granuloma. Although pilonidal sinuses are present from birth, they usually are not manifested clinically until adolescence or the early adult years, and pilonidal abscess formation most commonly affects young (often white) adults. The sinuses and cysts are lined with stratified squamous epithelium and may contain wads of hair and debris when excised. When cultured, pilonidal abscesses generally yield mixed fecal flora with a preponderance of anaerobes. [64]

The patient with a pilonidal abscess will seek care for back pain and local tenderness. On physical examination the area is indurated, but frank abscess formation may not be appreciated. One will usually see barely perceptible dimples or tiny openings at the rostral end of the gluteal crease (Fig. 40-10) (Figure Not Available) . A hair or a slight discharge may be noticed at the opening. One may find a more caudal cyst or abscess, possibly with a palpable sinus tract connecting the two. The sinus and cyst may be chronically draining, or they may become infected as the size increases and blockage occurs. [65]

Treatment of the acutely infected cyst is the same as previously discussed for any fluctuant abscess; all hair and pus should be removed, and the lesion should be packed. Antibiotic therapy is not usually required. The abscess cavity may become quite large, necessitating a rather lengthy incision to ensure complete drainage. It may take many weeks for the initial incision to heal. The area may be repacked at 2- to 4-day intervals as an outpatient procedure, although some prefer to discontinue packing after the first week.  Because simple incision and drainage are often not curative, secondary removal of both the cyst and the sinus should be planned after the inflammatory process has resolved. The elective surgical procedure should be complete and should involve all of the possible arborizations of the sinus.

Recurrence is occasionally prevented by simple incision and drainage, especially if the incision is wide, and adequate drainage is obtained. More commonly, recurrence can be expected unless excision of the sinus tract is performed. Small abscesses may be incised and drained as an outpatient procedure performed under local anesthesia, but the disease process is often extensive, and general anesthesia may be required to complete drainage. One is often surprised by the extent of the cyst cavity and the volume of pus that is encountered when the area is probed during initial incision; because of the degree of these abscesses, only localized infection lends itself to outpatient therapy. A method of catheter drainage for pilonidal abscesses has been described [37] [38] in which a flared-end Pezzer catheter is used for extended periods in

the abscess cavity. The catheter allows the patient more freedom from local care and provides continual drainage (Fig. 40-11) (Figure Not Available) .

## Perirectal Abscesses

Perirectal infections can range from minor irritations to fatal illnesses. Successful management depends on early recognition of the disease process and adequate surgical therapy. Because of the morbidity and mortality associated with inadequate treatment of these conditions, patients with all but the most localized abscesses should be promptly admitted to the hospital for evaluation and treatment under general or spinal anesthesia.

It is important to understand the anatomy of the anal canal and the rectum in order to appreciate the pathophysiology of these abscesses and their treatment (Fig. 40-12) (Figure Not Available) . The mucosa of the anal canal is loosely attached to the muscle wall. At the dentate line, where columnar epithelium gives way to squamous epithelium, there are vertical folds of tissue, called the rectal *columns of Morgagni,* which are connected at their lower ends by small semilunar folds called *anal valves.* Under these valves are invaginations termed *anal crypts.* Within these crypts are collections of ducts from anal glands. These glands are believed to be responsible for the genesis of most, if not all, perirectal abscesses. These glands often pass through the internal sphincter but do not penetrate the external sphincter.

The muscular anatomy divides the perirectal area into compartments that may house an abscess, depending on the direction of spread of the foci of the infection (Fig. 40-13) (Figure Not Available) . [65] [66] The circular fibers of the intestinal coat thicken at the rectum-anus junction to become the internal anal sphincter. The muscle fibers of the levator ani fuse with those of the outer longitudinal fibers of the intestinal coat as it passes through the pelvic floor. These conjoined fibers then are connected by fibrous tissue to the external sphincter system, which consists of three circular muscle groups.

### Pathophysiology

As described previously, the anal glands are mucus-secreting structures that terminate in the area between the internal and external sphincters. It is believed that most perirectal infections begin in the *intersphincteric* space secondary to blockage and subsequent infection of the anal glands. Normal host defense mechanisms then break down, followed by invasion and overgrowth by bowel flora. [67]

If the infection spreads across the external sphincter laterally, an *ischiorectal abscess* is formed. If the infection dissects rostrally, it may continue between the internal and external sphincters, causing a *high intramuscular abscess.* The infection may also dissect through the external sphincter over the levator ani to form a *pelvirectal abscess.* [66]

When infection of an anal crypt extends by way of the perianal lymphatics and continues between the mucous membrane and the anal muscles, a *perianal abscess* forms at the anal orifice. The perianal abscess is the most common variety of perirectal infection.

The abscess lies immediately beneath the skin in the perianal region at the lowermost part of the anal canal. It is separated from the ischiorectal space by a fascial septum that extends from the external sphincter and is continuous with the SQ tissue of the buttocks. The infection may be small and localized or very large, with a wall of necrotic tissue and a surrounding zone of cellulitis. [39] Perianal abscesses may be associated with a fistula in ano. The *fistula in ano* is an inflammatory tract with an external opening in the skin of the perianal area and an internal opening in the mucosa of the anal canal. The fistula in ano is usually formed after partial resolution of a perianal abscess, and its presence is suggested by recurrence of these abscesses with intermittent drainage. The external opening of the fissure is usually a red elevated piece of granulation tissue that may have purulent or serosanguineous drainage on compression. Many times the tract may be palpated as a cord. Patients with anal fistulas should be referred for definitive surgical excision.

*Ischiorectal abscesses* are fairly common. They are bounded superiorly by the levator ani, inferiorly by the fascia over the perianal space, medially by the anal sphincter muscles, and laterally by the obturator internus muscle. These abscesses may commonly be bilateral, and if so, the 2 cavities communicate by way of a deep postanal space to form a "horseshoe" abscess.

*Intersphincteric abscesses* are less common. They are bounded by the internal and external sphincters and may

extend rostrally into the rectum, thereby separating the circular and longitudinal muscle layers.

The *pelvirectal,* or *supralevator, abscess* lies above the levator ani muscle in proximity to the rectal wall and remains extraperitoneal. The etiology of this abscess is controversial. Kovalcik and colleagues [67] suggest that supralevator abscesses are primarily an extension of an intra-abdominal process, such as diverticulitis or pelvic inflammatory disease. Read and Abcarian [68] evaluated 404 patients with perirectal abscesses in a prospective study and found that of the 36 supralevator abscesses, none was caused by an intra-abdominal or pelvic pathologic condition. They determined that supralevator abscesses were most commonly associated with ischiorectal abscesses and suggested that these conditions may be an extension of ischiorectal abscesses through the floor of the levator ani. Nonetheless, they found rare isolated pelvirectal abscesses without intra-abdominal, pelvic, ischiorectal, or perianal infection.

Causes of perirectal abscesses other than the so-called cryptoglandular process have been documented but are fairly rare. It is believed that hemorrhoids, anorectal surgery, episiotomies, or local trauma may cause abscess formation by altering local anatomy and thus destroying natural tissue barriers to infections.

**Epidemiology**

Anorectal abscesses occur most commonly in healthy adults and are more frequent in males (>2:1 ratio).  These abscesses commonly appear during the fourth decade of life. Possible predisposing medical conditions are diabetes mellitus, inflammatory bowel

disease, and other immunocompromised states. Many patients (30%) have a history of previous perirectal abscess, and 75% of anorectal abscesses occur in the same location as the prior abscesses. [67] Of perirectal abscesses, usually >45% are perianal, 20% are ischiorectal, 12% are intersphincteric, and 7% are pelvirectal. [68]

**Physical and Laboratory Findings**

The diagnosis of a perianal abscess is generally not difficult. The throbbing pain in the perianal region is acute and is aggravated by sitting, coughing, sneezing, and straining. There is swelling, induration, and tenderness, and a small area of cellulitis is present in proximity to the anus. Rectal examination of the patient with a perianal abscess reveals that most of the tenderness and induration is below the level of the anal ring.

Patients with *ischiorectal abscesses* present with fever, chills, and malaise, but at first there is less pain than with the perianal abscess. Initially on physical examination, one will see an asymmetry of the perianal tissue, and later erythema and induration become apparent. Digital examination reveals a large, tense, tender swelling along the anal canal that extends above the anorectal ring. If both ischiorectal spaces are involved, the findings are bilateral.

Patients with *intersphincteric abscesses* usually present with dull, aching pain in the rectum rather than in the perianal region. No external aberrations of the perianal tissues are noted, but tenderness may be present. On digital examination one frequently palpates a soft, tender, sausage-shaped mass above the anorectal ring; if the mass has already ruptured, the patient may give a history of passage of purulent material during defecation.

Diagnosis of *pelvirectal abscesses* may be very difficult. Usually fever, chills, and malaise are present, but because the abscess is so deep seated, few or no signs or symptoms are present in the perianal region. Rectal or vaginal examination may reveal a tender swelling that is adherent to the rectal mucosa above the anorectal ring.

Laboratory findings usually do not aid in the diagnosis. Kovalcik and coworkers [67] found that <50% of their patients had a white blood cell count >$10.0 \times 10^9$ /L. Cultures of perirectal abscesses usually show mixed infections involving anaerobic bacteria, most commonly *Bacteroides fragilis* and gram-negative enteric bacilli.

**Treatment**

Successful management of perirectal abscesses depends on adequate surgical drainage. Complications from these infections may necessitate multiple surgical procedures, prolong hospital stay, and result in sepsis and death. Bevans and associates [69] retrospectively studied the charts of 184 patients who were surgically treated over a 10-year period. These patients were evaluated primarily to identify the factors that contributed to morbidity and mortality. Initial drainage was performed under local anesthesia in 38% of the patients and under spinal or general anesthesia in 62%. The authors identified 3 key factors in excessive morbidity and mortality: (1) a delay in

diagnosis and treatment, (2) inadequate initial examination or treatment, and (3) associated systemic disease. It was their belief that the only way to examine effectively and drain adequately all but the most superficial perirectal abscesses was under spinal or general anesthesia. This assessment was supported by evidence of an increased incidence of recurrence and of sepsis and death in patients treated with local anesthesia. Drainage under local anesthesia generally does not allow drainage of all hidden loculations. In addition, local anesthesia is not adequate for treatment of associated pathologic conditions.

Small, well-defined perianal abscesses are the only perirectal infections that lend themselves to outpatient therapy. The result of incision and drainage is almost immediate relief of pain and rapid resolution of infection. Indications for inpatient drainage are failure to obtain adequate anesthesia, systemic toxicity, extension of the abscess beyond a localized area, or recurrence of a perianal abscess. Recurrence may be caused by the presence of a *fistula in ano.*

A perianal abscess is drained through a single linear incision over the most fluctuant portion of the abscess in a manner previously described for other cutaneous abscesses. It is extremely painful to probe a perianal abscess and to break up loculations, and liberal analgesia is advised. The patient may begin sitz baths at home 24 hours following surgery. Packing is replaced at 48-hour intervals until the infection has cleared and granulation tissue has appeared. This usually occurs within 4 to 6 days. Antibiotics are generally not required. All other perirectal abscesses require hospitalization for definitive therapy.

Use of Pezzer catheters in anorectal abscesses has been described as an alternative to traditional incision and packing. Kyle and Isbister reported a series of 91 patients treated in this manner. They found equivalent rates of subsequent fistula surgery, less need for general anesthesia, and a shorter postoperative hospital stay when compared with patients treated with traditional incision and packing. [70] Beck and colleagues reported successful use of catheter drainage in 55 patients with ischiorectal abscess. [71] Due to the complexity of

ischiorectal abscesses, this technique is probably best left to the surgeon providing ongoing care.

Perirectal abscesses are currently recognized as a fairly common cause of fever in the granulocytopenic patient. These abscesses have a different bacteriologic profile: *Pseudomonas aeruginosa* organisms are isolated most frequently. These patients present later because pain develops later in the course, and fever may be the first manifestation. Therefore, any patients who are granulocytopenic with vague anorectal complaints, especially those with fever, should be examined carefully for perirectal abscesses. Any abscess that is found should be drained immediately under appropriate anesthesia, and extensive IV antibiotic coverage should be initiated.

**Infected Sebaceous Cyst**

A common entity that appears as a cutaneous abscess is the infected sebaceous cyst.

Sebaceous cysts may occur throughout the body and result from obstruction of sebaceous gland ducts. The cyst becomes filled with a thick, cheesy sebaceous material, and the contents frequently become infected. Sebaceous cysts may be quite large and may persist for many years before they become infected. When infected, they clinically appear as tender, fluctuant SQ masses, often with overlying erythema.

The initial treatment of an infected sebaceous cyst is simple incision and drainage. The thick sebaceous material must be expressed, since it is too thick to drain spontaneously (Fig. 40-14 *A*). An important difference exists between infected sebaceous cysts and other abscesses. *A sebaceous cyst has a definite pearly white capsule that must be excised to prevent recurrence* (Fig. 40-14 *B* and *C*). Traditionally, in the presence of significant inflammation, it is preferable to drain the infection initially and remove the shiny capsule on the first follow-up visit or a later visit, when it may be more easily identified. Alternatively, the entire cyst can be removed at the time of initial incision. At the time of capsule removal, the edges are grasped with clamps or hemostats, and the entire capsule is removed by sharp dissection with a scalpel or scissors. Following excision of the capsule, the area is treated in the same manner as a healing abscess cavity. Simple drainage without excision of the capsule often leads to recurrence.

Kitamura and associates reported a randomized study of 71 patients treated with either traditional incision and drainage or primary resection of the cyst, followed by irrigation and wound closure. In this study, the patients treated with primary resection had faster healing, fewer days of pain, and less scarring. [72]

**Paronychia**

A paronychia is an infection localized to the area around the nail root (Fig. 40-15) (Figure Not Available) . Paronychias are common infections probably caused by frequent trauma to the delicate skin around the fingernail and the cuticle. When a minor infection begins, the nail itself may act like a foreign body. Usually the infectious process is limited to the area above the nail base and underneath the eponychium (cuticle), but occasionally it may spread to include tissue under the nail as well, forming a subungual abscess. Lymphadenitis and lymphadenopathy are usually not seen. Generally, a paronychia is a mixed bacterial infection. *Staphylococcus* is commonly cultured from these lesions; however, anaerobes and numerous gram-negative organisms may be isolated. [73] Paronychias in children are often caused by anaerobes, and it is believed that this is the result of finger sucking and nail biting. Occasionally, a group A beta-hemolytic infection will develop in a paronychia if a child with a streptococcal pharyngitis puts his or her fingers in the mouth. [74]

A paronychia appears as a swelling and tenderness of the soft tissue along the base or the side of a fingernail (Fig. 40-16) . Pain, often around a hangnail, usually prompts a visit to the ED. The infection begins as a cellulitis and may form a frank abscess. If the nailbed is mobile, the infectious process has extended under the nail, and a more extensive drainage procedure should be performed. If soft tissue swelling is present without fluctuance, remission may be obtained from frequent hot soaks (6 to 8 times a day) and a short course of oral antibiotics (3 to 4 days). [73] Incision will be of no value at this early cellulitic phase. If a significant cellulitis is present, a broad-spectrum

antistaphylococcal antibiotic (cephalosporin or semisynthetic penicillin) may be tried. However, plain penicillin or erythromycin is often sufficient for limited inflammation. The digit should be splinted and elevated. [75] [76] One should never rely solely on antibiotic therapy once frank pus has formed.

**Technique**

When a definite abscess has formed, drainage is usually quickly curative. A number of invasive operative approaches have been suggested, but actual skin incision or removal of the nail is rarely required, and *neither procedure should be the initial form of treatment.* One can invariably obtain adequate drainage by simply lifting the skin edge off the nail to allow the pus to drain. This is usually curative, because a paronychia is not a cutaneous abscess per se, but rather a collection of pus in the potential space between the cuticle and proximal fingernail. Drainage may be accomplished without anesthesia in selected patients but frequently requires a digital nerve block. After softening the eponychium by soaking, a No. 11 blade, scissors, or an 18-ga needle is advanced *parallel to the nail* and under the eponychium at the site of maximal swelling (Fig. 40-17) . [76] [77] Pus rapidly escapes, with immediate relief of pain. A tourniquet placed at the base of the finger may limit bleeding and aid the physician in determining the exact extent of the infection during the drainage procedure.

If more than a tiny pocket of pus is present, one should fan the knife tip or needle or spread the scissors under the eponychium, keeping the instrument parallel to the plane of the fingernail. When a large amount of pus is drained, a small piece of packing gauze is slipped under the eponychium for 24 hours to provide continual drainage. Cultures are generally not indicated. Antibiotics are frequently prescribed, although they are not essential if drainage is complete or if the surrounding area of cellulitis is minimal. An alternative to systemic antibiotics is to keep the operative site bathed in antibiotic ointment. After anesthesia has worn off, the patient may be started on frequent soaks in warm tap water at home. In most cases the patient may easily remove the packing. At 24 to 36 hours, the finger is soaked in hot water and the gauze pulled out; a repeat visit to a physician is not required if healing is progressing. Once the packing is removed, the area is covered with a dry, absorbent dressing. An antibiotic ointment may be applied to the site for a few days. The benefit of antibiotic ointments in reducing infection is unproved, but instructing the patient concerning the use of the ointment may prompt soaking. In addition, the ointment helps to keep the bandage from sticking.

If the infection has produced purulence beneath the nail (subungual abscess), a portion of the nail must be removed or the nail trephined to ensure complete drainage. As an alternative to nail removal, a hole may be placed in the proximal nail with a hot paper clip. A large opening or multiple holes are required to ensure continued drainage. Most commonly the proximal portion of the nail is involved. This may be treated by bluntly elevating the eponychium to expose the proximal edge of the nail. The proximal one third of the nail is then elevated from the nailbed and resected with a scissors. The distal two thirds of the nail is left in place to act as a physiologic dressing and to decrease postoperative pain (Fig. 40-18) . If purulence is found below the lateral edge of the nail, the affected part may be gently elevated and excised longitudinally. [78] Care must be exercised during this procedure to avoid damage to the nail matrix. A wick of gauze

should be placed beneath the eponychium for 48 hours to ensure continued drainage.

Most paronychia resolve in a few days, and 1 to 2 postoperative visits should be scheduled to evaluate healing and reinforce home care. For compliant patients with a small paronychia, home care alone may suffice after the initial drainage. A well-known complication of even a properly drained paronychia is osteomyelitis of the distal phalanx. Clinical infection lasting longer than a few weeks should prompt evaluation for this complication.

Patients occasionally present to the ED complaining of a chronic, indolent infection of the paronychium. These seldom respond to ED intervention. Frank purulence is seldom present, and conservative treatments are often unsatisfactory. Many etiologies have been described for this frustrating condition, including fungal, bacterial, viral, and psoriatic conditions. Treatment modalities are varied, and controlled studies evaluating the various techniques are lacking. Meticulous hand care, oral and topical antimicrobial medications, and occasionally aggressive surgical intervention have been suggested. These patients should be referred to a dermatologist or hand surgeon because of the prolonged treatment required.

**Herpetic Whitlow**

Herpetic whitlow is an infection of the distal phalanx caused by the herpes simplex virus. Digital inoculation occurs through a discontinuity of the skin. [78] Health care providers and patients with other herpes infections are most commonly infected. The entity is recognized by the presence of herpetic vesicles, a burning or pruritic sensation, absence of frank pus, slow response to treatment, and a tendency to recur. In questionable cases, viral cultures can be obtained. Herpetic lesions are generally quite painful but are self-limited and resolve in 2 to 3 weeks. Surgical intervention is contraindicated, as this may potentiate a secondary bacterial infection and delay healing.  Treatment is symptomatic, consisting of splinting, elevation, and analgesia as needed. Antibiotics effective against herpes infections (acyclovir, famciclovir, and others) probably shorten the course of the disease if given early. Consideration must be given to preventing spread of the infection to other individuals. Although

an occlusive dressing may lessen the chance for viral transmission, any health care provider with this entity should refrain from patient contact until all lesions have crusted over and viral shedding has stopped.

**Felon**

A felon is an infection of the pulp of the distal finger (Fig. 40-19) . The usual cause is trauma with secondary invasion by bacteria. A felon may develop in the presence of a foreign body, such as a thorn or a splinter, but often a precipitating trauma cannot be identified. An important anatomic characteristic of this area is that there are many fibrous septa extending from the volar skin of the fat pad to the periosteum of the phalanx; these subdivide and compartmentalize the pulp area. When an infection occurs in the pulp, these same structures make it a closed space infection. The septa limit swelling, delay pointing of the abscess, and inhibit drainage after incomplete surgical

decompression. Pressure may increase in the closed space, initiating an ischemic process that compounds the infection. The infection can progress readily to osteomyelitis of the distal phalanx. Although the septa may facilitate an infection in the pulp, they provide a barrier that protects the joint space and the tendon sheath by limiting the proximal spread of infection.

The offending organisms are usually *Staphylococcus* or *Streptococcus,* although mixed infections and gram-negative infection may occur. A felon is one of the few soft tissue infections in which a culture may be helpful, since osteomyelitis and prolonged infection may occur. An initial culture may aid in the subsequent choice of antibiotics for complicated infections.

The patient developing a felon will describe gradual onset of pain and tenderness of the fingertip. In a few days the pain may be constant and throbbing and gradually becomes severe. In the initial stages, physical examination may be quite unimpressive, because the fibrous septa limit swelling in the closed pulp space. As the infection progresses, swelling and redness may become obvious. Occasionally one may elicit point tenderness, but frequently the entire pulp space is extremely tender. The patient characteristically arrives with the hand elevated over the head because pain is so intense in the dependent position. Cessation of pain indicates extensive necrosis and nerve degeneration.

Proper treatment of a well-developed felon consists of early and complete incision and drainage. [78] Antibiotics alone are not curative once suppuration has occurred. Delaying surgery may result in permanent disability and deformity. Most surgeons routinely administer broad-spectrum antibiotics to patients for 5 to 7 days following surgical incision.

**Technique**

The surgery can usually be performed as an outpatient procedure using a digital nerve block. A long-acting solution (bupivacaine) will prolong anesthesia. A tourniquet (1.25 cm Penrose drain) should be used to allow digital incision in a bloodless field.

Surgical drainage must be carefully performed to avoid injury to digital nerves, vessels, and flexor tendon mechanisms. Most commonly, a felon can be successfully managed with a limited procedure, but many surgical options have been advocated, none of which has been proven superior for all circumstances. [78] The preferred initial treatment is a simple longitudinal incision made over the area of greatest fluctuance, [87] which may occur laterally or along the volar surface (Fig. 40-20) . A potential drawback to an incision in the middle of the fat pad is the production of a scar in a very sensitive and commonly traumatized area. The incision must not extend to the distal interphalangeal crease because of the danger of injuring the flexor tendon mechanism. The SQ tissue is bluntly dissected using a hemostat to provide adequate drainage. A gauze pack may be placed in the wound for 24 to 48 hours to ensure continued drainage.

Recurrent or more severe infections may require a more aggressive approach. The following traditional incisions have a greater propensity for complications such as

sloughing of tissue and postoperative fat pad anesthesia or instability, [87] although they may provide for more complete drainage.

The hockey-stick incision is a well-accepted drainage procedure (Fig. 40-21) (Figure Not Available) . This incision is advantageous if the infection points to 1 side of the finger. The incision begins in the midline of the tip of the fat pad, just under the distal edge of the fingernail. It is extended to the lateral tip of the finger and proximally along the side of the distal phalanx (at the junction of the volar and dorsal skin markings) to 3 to 5 mm distal to the distal interphalangeal joint. The tip of the knife blade is inserted just under the bone to a depth corresponding to the opposite edge of the distal phalanx-- slightly more than halfway across the volar surface of the finger. A hemostat is inserted into the incision and is spread in the plane of the fingernail (perpendicular to the septa) to break open remaining septa and loculations. Necrotic tissue or any foreign matter is excised under direct vision, and the wound is irrigated. A small gauze pack is placed in the incision. Because the incision may produce partial numbness of the fingertip by associated digital nerve injury, the incision

**Figure 40-21** (Figure Not Available) Hockey-stick incision for the drainage of a felon. *Note:* An incision on the ulnar side of the index, middle, and ring fingers is appropriate. The little finger is best incised on the radial side. The site of the incision on the thumb is also preferably on the radial side, but this may depend on the occupation of the patient. *(From Chase RA: Atlas of Hand Surgery. Philadelphia, WB Saunders, 1973. Reproduced with permission.)*

should *not* be made on the radial aspect of the index finger or the ulnar aspect of the thumb or little finger.

An acceptable alternative to the hockey-stick, or median, incision is the through-and-through incision (Fig. 40-22) . This is basically a hockey stick-type incision (without the curved distal portion of the "hockey stick") that is carried through to the opposite side of the finger. A hemostat is used to break up loculations, and a rubber drain (Penrose) is placed through the incision for continual drainage. The through-and-through incision is probably the easiest procedure for most felons.

The fishmouth, or horseshoe, incision is basically 2 hockey-stick incisions that meet at the tip of the finger. A gauze pack is placed between the flaps and should be removed in a few days. This is a rather radical procedure but allows complete visualization and debridement of necrotic tissue (Fig. 40-23) . Some physicians advise against this incision, because it is extensive and may take a long time to heal. In addition, it produces a sizable scar and an unstable finger pulp. The fishmouth incision may be used if more conservative incisions are not successful, but it is not recommended for use initially.

No matter which incision is made, it must not be carried proximal to the closed pulp space because of the danger of entrance into the tendon sheath or the joint capsule. The patient should be rechecked in 2 to 3 days. A snug dressing, splinting and elevation, and adequate opioid analgesics are prerequisites for a successful outcome and a happy patient.

On the first postoperative visit, a digital block may again be performed and any packing

removed. The incision is irrigated copiously with saline, and any additional necrotic tissue is removed. At this time, the drain may be replaced for 24 to 48 hours if there is continued drainage, but usually it can be removed and a dressing reapplied. Soaking may be advised. At the first revisit, the sensitivities of the bacterial cultures are checked, and a decision to continue or change antibiotics is made. Most felons are empirically treated with antibiotics for at least 5 days. A broad-spectrum cephalosporin is a reasonable choice, pending cultures (if done).

A few additional points should be emphasized at this time. Frank pus may be encountered during incision, but usually only a few drops are expressed. One more often drains a combination of necrotic tissue and interstitial fluid. A careful search for a foreign body should be made even if the history is not known. Some physicians advocate radiographic evaluation for retained foreign bodies and a baseline evaluation of the bone for subsequent evaluation of osteomyelitis at the initial visit. Other physicians will reserve radiographs for wounds not showing significant improvement in 5 to 7 days. Evidence of osteomyelitis, however, may not be found radiographically for several weeks after the appearance of the lesion. More radical incision and drainage may be required in persistent infections. Following adequate drainage, osteomyelitis may respond surprisingly well to outpatient antibiotic therapy with almost complete regeneration of bone if incision and drainage have been adequate. Persistent cases may require IV antibiotics.

*Resistant fingertip infections are not uncommon.* Difficult or persistent cases require evaluation and care by a hand surgeon. In these cases, early consultation is advisable to avert catastrophic complications such as loss of function or amputation.

## Subungual Hematoma

Subungual hematoma is an injury that is frequently seen in the ED. Any digit may be affected. The hematoma often results from hitting the fingertip with a hammer or slamming it in a door. The main concern of the patient is relief of the terrible throbbing pain that accompanies the condition as the pressure of the hematoma increases. Pain relief can be accomplished quickly with nail trephination. Trephination may be performed with a large paper clip that has been heated until it is red hot. The instrument is applied to burn a hole at the base of the nail (Fig. 40-24) . Blood rapidly exits, and the blackened nail regains its normal color (Fig. 40-25) . The blood usually remains fluid for 24 to 36 hours and is easily expressed with slight pressure. Care should be taken to make multiple holes or a single hole that is large enough to allow continued drainage. An oversized paper clip is the simplest apparatus. Although a portable hot-wire electrocautery unit is available and is frequently recommended, it is difficult to obtain an adequate drainage hole without adapting the instrument and its use. One can modify the electrocautery device to burn a larger hole by "fattening" the end of the wire loop and rotating the device slowly as the nail is penetrated or by removing a small rectangle of nail with the cautery device. In addition to being convenient, the cautery device is desirable because the wire stays hotter longer, thus enhancing nail penetration. In the stoic patient, no anesthesia may be necessary, but a digital block affords painless trephination, and its routine use is suggested with the anxious patient.

The majority of subungual hematomas are painful but minor injuries. Complicated cases involve fractures of the distal phalanx. When the fingertip is unstable or the mechanism of injury suggests a significant distal phalanx fracture, a radiograph should be obtained. If a significant fracture is present, the digit should be splinted. A distal phalangeal fracture with a subungual hematoma is technically an open (compound) fracture. Such injuries usually heal without problems, although osteomyelitis of the tuft is a theoretical complication. The value of routine antibiotic prophylaxis in such cases is unproved, and their use is not standard in minor cases but may be of value in significant crush injuries. The presence of an underlying fracture does not contraindicate nail trephination for fear of changing closed fracture into an open one. It is difficult to predict the fate of the fingernail following drainage of a subungual hematoma. Some patients with subungual hematomas will lose the nail, but if the nail root or nailbed is not significantly disrupted and the nail remains implanted, a normal-appearing nail is the usual final result. If the nailbed is significantly lacerated or the edges of the nail are unstable, the nail should be removed and the nailbed repaired (see further discussion in Chapter 37) . Patients should be informed of possible future cosmetic problems.

# Chapter 41 - Burn Care Procedures

**Thomas J. Krisanda, Courtney A. Bethel**

Each year in the United States some 2 million people suffer a burn-related injury. Typical victims are children <5 years of age or young adults who are exposed to fire or hot or corrosive substances. [1] Fortunately, 95% of these burns are classified as minor and are amenable to outpatient management, with most patients completing their treatment course within 2 weeks.

The classification of burns is based on 3 criteria [2] : depth of skin injury, percentage of body surface area involved, and source of injury (thermal, chemical, electrical, or radiation). The *seriousness* of a burn injury is determined by the characteristics and temperature of the burning agent, the duration of exposure, the location of injury, the presence of associated injuries, and the age and general health of the victim.

The American Burn Association defines *minor* burns as uncomplicated partial-thickness burns of <10% of the total body surface area (TBSA) in children or the elderly or <15% TBSA in adults, or full-thickness burns of <2% TBSA. [3] *Moderate or major* burns include injuries that involve a greater TBSA, as well as burns to areas of specialized function, such as the face, hands, feet, or perineum. More serious burns also include those due to a high-voltage electrical injury or those with associated inhalation injuries or other major trauma.

The TBSA burned may be estimated in a number of ways. In adults, the "rule of nines" is a useful rule of thumb, but the formula is only a guide and *must be modified for children* who have proportionately larger heads and smaller legs (Fig. 41-1) . The Lund and Browder charts are another (more precise) guide to estimating the percentage of TBSA burned (Fig. 41-2) . For smaller or multiple burns, one can rapidly estimate the TBSA burned by using the area of the patient's palm as approximately 1.25% TBSA.

Throughout the course of history, physicians have experimented with burn therapies to relieve pain and promote healing. Before the latter half of the 19th century, the treatment of burns had little scientific basis. Accepted treatments consisted primarily of bleeding and purging the patient and placing substances on the wound that tended to promote infection or were toxic. Fortunately, the pathophysiology of burn injury and burn healing has gradually been elucidated, and the care of burns has evolved into its present form.

## WOUND EVALUATION

Emergency physicians should be aware that the depth of a burn wound cannot always be determined accurately on clinical grounds alone at the time of presentation and that burn injury is a dynamic process that may change over time, particularly during the 24 to 48 hours after the burning process has been arrested. It is common, for example, for a seemingly minor or superficial burn to appear deeper on the second or third return visit. This phenomenon is not a continuation of the burning process but is considered to be a

pathophysiologic event related to tissue edema, dermal ischemia, or desiccation. [4]

*First-degree burns* involve the epidermis only. The skin is reddened but is intact and not blistered. This injury ranges from mildly irritating or even pruritic to exquisitely painful. Minor edema may be noted. Causes include ultraviolet light (as in sunburn) and brief thermal "flash" burns. First-degree burns frequently blister within 24 to 36 hours, so the patient should be instructed appropriately. Often the skin begins to flake or peel within 5 to 10 days, but healing eventually occurs with no scarring.

*Second-degree burns* involve the entire epidermis and extend into the dermis to include sweat glands and hair follicles. *Superficial partial-thickness burns* involve only the papillary dermis. These burns are pink, moist, and extremely painful. Blisters may be present or the skin may slough. The burn blanches with pressure, and mild to moderate edema is common. Hair follicles are often noted to be intact. This is the most common depth of minor burn seen in the emergency department (ED). The usual causes are scalds, contact with hot objects, or exposure to chemicals. Barring infection or repeated trauma, these burns heal completely without scarring in about 2 weeks.

*Deep partial-thickness burns* extend into the reticular dermis and appear as mottled white or pink. There is obvious edema and sloughing of the skin, and any blisters are usually ruptured. Blanching is absent. These burns are generally not painful initially, but pressure can be perceived. Within a few days, however, these burns can become exquisitely painful. This type of burn can easily be converted to a full-thickness injury by further trauma or infection. Partial-thickness burns heal by re-epithelialization from dermal appendages, including hair follicles, sebaceous glands, and sweat glands.

In *full-thickness burns,* coagulation necrosis extends into the subcutaneous (SQ) tissues. These burns may appear a variety of colors but are usually dry, pearly white, or charred. They are initially painless, with a leathery texture. Marked edema and decreased elasticity may necessitate escharotomy when circulation is compromised. Exposure of the skin to temperatures in excess of 77 °C for more than 3 to 4 seconds generally causes a full-thickness injury. Although initially painless, in a few days these deep burns can become painful. Chemical burns often produce full-thickness injuries affecting a small or scattered surface area. Flame burns produce full-thickness injuries in <2 seconds if the temperature of the flame exceeds 500 °C. Generally, any burn wound that is not re-epithelialized or does not possess dense epidermal budding by 14 days post-burn should be considered a full-thickness injury. [4]

*Fourth-degree burns* extend deeply into SQ tissue, muscle, fascia, or bone. These burns are characteristically caused by contact with molten metal, flame, or high-voltage electricity.

## HISTOPATHOLOGY OF BURNS

One thermal wound theory describes 3 zones of injury in burns [5] :

1. *Zone of coagulation:* dead, avascular tissue that must be debrided.

2. *Zone of stasis:* injured tissue in which blood flow is impaired. Desiccation, infection, or mechanical trauma may lead to cell death.
3. *Zone of hyperemia:* minimally injured, inflamed tissue that forms the border of the wound. The hyperemia usually resolves within 7 to 10 days but may be mistaken for cellulitis.Histologically, full-thickness burns are characterized by confluent vascular thrombosis involving arterioles, venules, and capillaries. Edema due to loss of microvascular integrity results not only from the effects of direct thermal injury, but also from the release of vasoactive mediators. The increase in vascular permeability is linked to complement activation and histamine release. Histamine increases the catalytic activity of the enzyme xanthine oxidase, with resultant production of hydrogen peroxide and hydroxyl radicals. These by-products increase the damage to dermal vascular endothelial cells and result in progressive vascular permeability. [6] The cellular debris and denatured proteins of the eschar provide a substrate for the proliferation of microorganisms. The devitalized tissue (eschar) sloughs spontaneously, usually as a result of the proteolytic effect of bacterial enzymes. The greater the degree of wound bacteriostasis, the greater the delay in sloughing.Partial-thickness burns result in incomplete vascular thrombosis, usually limited to the upper dermis. The dermal circulation is gradually restored, usually over several days, resulting in a significant interval of relative ischemia. The eschar in deep partial-thickness burns is thinner than in a full-thickness burn and sloughs as a result of re-epithelialization rather than bacterial proteolysis.

## OUTPATIENT VS INPATIENT CARE

One of the first steps in minor burn care is to select patients for whom outpatient care is appropriate. Candidates for outpatient treatment are generally adults and children who meet the minor burn criteria detailed earlier. Persons who have deep burns of the hands, face, feet, neck, or perineum; burns resulting from abuse or attempted suicide; burns involving other significant trauma or inhalation injuries; or electrical burns should generally be managed as *inpatients*.

Poor candidates for outpatient care of even minor burns include those who have concomitant medical problems such as diabetes mellitus, peripheral vascular disease, congestive heart failure, and end-stage renal disease; patients who are using steroids or other immunosuppressive agents; patients who are very young or very old; those who are mentally retarded; alcoholics; those who are malnourished; and any individual with a suspect or unacceptable home support system. Inpatient treatment should be considered under these circumstances even though the burn might be considered "minor" by TBSA formulas.

## PROCEDURE

### Initial Care of the Minor Burn Victim

Prompt cooling of the burned part is an almost instinctive response and is one of the oldest recorded burn treatments, having been recommended by Galen (129 to 199 A.D.)

and Rhazes (852 to 923 A.D.). [4] Room-temperature tap water irrigation, immersion, or compresses (20 to 25 °C) are optimal in obtaining pain relief and providing some measure of protection for burned tissues without the problems of hypothermia that iced solutions can cause. [7] [8] First-aid telephone advice from the emergency physician includes immediately immersing the wound in room-temperature tap water. *Packing the wound in ice must be avoided*.

Emergency medical services (EMS) personnel should have a clear understanding of proper management of the minor burn victim. The benefits and details of early cooling should be understood and implemented. The victim should first be removed from danger. All involved clothing and jewelry, along with any gross debris, should be removed from the burned area. Chemical burns to the skin or eyes require prolonged tap water irrigation. The burn should be otherwise covered with a moist, sterile dressing-- nonmentholated shaving cream makes an excellent temporary covering for out-of-hospital use if a dressing is not available. [9] Home remedies such as butter, grease, or petrolatum should be avoided.

In the ED, the burned area should be immediately immersed in room temperature water or covered with gauze pads soaked in room-temperature water (Fig. 41-3) . The gauze must be kept cool and moist to provide continued pain relief; the patient will quickly let the physician know when additional cooling is required. Burns should be cooled or immersed until supplemental analgesia is effective and dressings are applied. Many physicians use sterile saline for cooling, but it has no proven benefit over tap water, even when the skin is broken. Immersion of burned tissue in ice or ice water should be avoided, because ice immersion increases pain and risks frostbite injury or systemic hypothermia.

The potential benefits of burn cooling are listed in Table 41-1 . With the exception of pain relief and removal of debris, the benefits of burn cooling are experienced only if the burn is cooled promptly, within the first 3 minutes after injury. [10]

Prompt burn cooling arrests cell death processes by inhibiting the release of toxic substances by the dying cells. If cooling is not performed, continued cell death results as oxygen free radicals are released, which can cause a "chain reaction" of cell membrane injury, arachidonic acid release, and increased local ischemia. [11]

The threshold temperature for cutaneous pain sensation is approximately 43 °C. The prompt alteration in capillary permeability and resultant edema induced by heat have been attributed to histamine release from stimulated or damaged mast cells. The tissue threshold for this phenomenon has been estimated at 52 °C. Cooling produces prompt and complete, but reversible, inhibition of histamine release. Tissue edema also results from damage to the membrane sodium pump, with resultant influx of sodium into tissue and loss of protein into the interstitial spaces. Unfortunately, this contribution to tissue edema is less responsive to local cooling. [1]

Minor burns are considered tetanus prone, and tetanus toxoid should be administered if the patient is unsure of his or her tetanus immunization status or when it has been more than 5 years since the last immunization. Nonimmunized patients should receive human

tetanus immune globulin, 250 units IM, along with tetanus toxoid, along with a booster injection of toxoid in about 3 weeks.

| TABLE 41-1 -- Advantage of Prompt Burn Cooling |
|---|
| Reduction or cessation of pain |
| Elimination of local hyperthermia |
| Inhibition of postburn tissue destruction |
| Decreased edema |
| Reduced metabolism and toxin production |

## Definitive Care of the Minor Burn

Few areas in medicine are fraught with as much mysticism, personal bias, and unscientific dogma as the care of the minor burn wound. Many physicians are rigidly committed to a specific ritual or approach merely because "it is the way it's done" in a specific institution or because the practitioner has had success with a particular therapy in the past. In reality, the plethora of successful regimens attests to the fact that almost any noninjurious approach results in a favorable outcome. Many misconceptions probably arise because the issues associated with major thermal injury are often erroneously extrapolated to the minor burn wound.

Minor burns are *not* associated with immunosuppression, hypermetabolism, or increased susceptibility to infection. [12] Many complications seen in minor burn care result from *overtreatment* of the injury rather than undertreatment. Examples include too-vigorous dressing changes that may peel off newly formed skin and secondary infections or pseudomembrane formation that results from topical or systemic antibiotic use.

**Burn Dressings**

**Open burn care.**

Burns are cared for using 2 general methods: open or closed. In the *open* method, a burn wound dressing is not used. The area is left open to the air and is washed 2 to 3 times per day, followed by application of a topical agent. This is the preferred method for managing burns of the face and neck and is an excellent way to manage hand burns, because it allows continuous inspection and range-of-motion exercises. The open method is impractical in young active persons, in children, or in other individuals in whom wound contamination is likely. Although many burns may be treated with this method, many patients prefer a dressing over a wound for cosmetic reasons.

**Simple closed dressing.**

The *closed* burn treatment method involves a dressing, of which there are various types. This is the method of choice for managing most minor burns treated in the ED. Wound preparation and basic bandaging should include the following steps (Fig. 41-4) :

1. The hair in the burn itself or around the wound should *not* be shaved. The burn is washed gently with a clean cloth or gauze pads and a mild non-alcohol-based soap or detergent (e.g., Ivory, Dreft, Hibiclens) and then flushed with normal saline. Adequate wound cleansing prevents further bacterial contamination, promotes early epithelialization in partial-thickness injuries, and aids the development of good granulation tissue in full-thickness injuries. [13]
2. Obviously sloughed skin should be debrided. This may be accomplished with scissors and forceps, but an expeditious and effective (and often painless) method is to use a dry 10-cm × 10-cm gauze pad (Fig. 41-5) to quickly debride loose skin. Meticulous and time-consuming instrument debridement is often quite stressful to the patient. Analgesia should be provided for any painful debridement.
3. Intact blisters are left alone in the absence of infection. Open blisters are debrided (Table 41-2) . All sloughed skin and blisters are debrided if infection is present.
4. A fine mesh gauze or a commercial nonadherent gauze such as Adaptic or Aquaphor is applied to the dry burn wound.
5. The burn is covered with loose gauze fluffs. If fingers and toes are included in the dressing, *the web spaces are padded and the digits are individually wrapped and separated with strips of gauze*. Failure to individually wrap fingers and toes may result in further injury (see Figure 41-4 *D*).
6. The entire dressing is wrapped snugly (but not tightly) with an absorbent, slightly elastic material such as Kerlix.
7. Antibiotic creams or ointments may be used as an option with this dressing. The topical antibiotic may be applied to the burned skin directly or impregnated into the gauze after step 3.Burn dressings should enhance healing. *The most important characteristic of a dressing is that it is capable of controlling fluid balance*. To accelerate healing, a burn dressing should be designed to keep the wound surface moist but avoid pooling of fluids. [14] The best material for this purpose is a generous amount of *simple dry gauze* applied over a nonadherent dressing or topical preparation. The outer dressing layer should be porous to permit the

evaporation of water from the absorbent dressing material.

**Biologic dressings.**

Biologic dressings are natural tissues, including skin, that consist of collagen sheets containing elastin and lipid. They are not routinely used in emergency care of minor wounds. Benefits of biologic dressings

---

**TABLE 41-2** -- General Approach to Blisters in Minor Burns [*]

If treated less than 48 hours after the burn:

  1. Leave all intact blisters alone.

  2. If blisters have ruptured, treat them as dead skin and debride them completely.

  3. Needle aspiration is generally not advised but may be used to decompress large burn blisters that appear ready to burst.

On follow-up, or more than 48 to 72 hours after the burn:

  1. Debride large (>5 cm in diameter) intact blisters and all blisters that have ruptured. Large, firm blisters of the palms and soles may be left intact longer. Do not aspirate blisters.

  2. Do not debride small or spotty blisters until they break, or until 5 to 7 days after the burn.

Five to 7 days after the burn:

| |
|---|
| 1. Debride all blisters completely . |
| *Note*: Intact blisters provide significant pain relief. Be prepared for an exacerbation of pain immediately after debridement. |
| Prophylactic analgesia is recommended. |

* All blisters and burned skin are debrided in the presence of infection.

include a reduction in surface bacterial colonization, diminished fluid and heat loss, prevention of further wound contamination, and prevention of damage to newly developed granulation tissue. Examples of biologic dressings include cadaveric human skin and commercially available porcine xenograft or collagen sheets.

**Synthetic dressings.**

Synthetic dressings are manufactured in various forms. Film-type dressings have a homogeneous structure and are usually polymers. Because these dressings are nonpermeable, there have been problems with retention of wound exudate. Some second-generation dressings have been developed that address these problems. These include the following:

1. *Tegaderm* (3M Inc., St Paul, Minn) pouch dressings consist of a thin polyurethane film coated with a water-resistant adhesive. The film is perforated to allow exudate fluid to pass into a pouch. An intact dressing is impermeable to liquids and bacteria. Although this dressing has been used on skin graft donor sites, it has not been studied as a primary burn dressing (C. D. Jones, written communication, 1993).
2. *Vigilon* (Bard, Inc., Berkeley Heights, NJ) is a hydrogel formulation of water and polyethylene oxide reinforced with a polyethylene support webbing. It is clear, conformable, and nonadherent and may be used along with topical antibiotic agents. Wound exudate is transmissible through the dressing and is absorbed by an overlying gauze dressing.
3. *DuoDerm* (Convatec, Inc, Arlington Heights, Ill) is a synthetic hydrocolloid dressing. The outer layer, consisting of polyurethane foam, is impermeable to gases, vapor, and fluids. The wound side of the dressing is a hydrocolloid polymer complex that adheres to the surrounding healthy skin via an approved acrylic adhesive. [15] Wound exudate converts the hydrocolloid complex into a gel, which creates a beneficial moist environment. The dressing may be left in place for up to 5 days. [16] DuoDerm is opaque and hence does not allow for wound inspection. It also has no antibacterial properties and should not be used on infected burns. [16]

4. *Biobrane* (Winthrop Pharmaceuticals, New York) is a

   biosynthetic, bilaminar membrane dressing that consists of a knitted nylon fabric mechanically bonded to a thin Silastic semipermeable membrane. Cross-linked collagen polypeptides coat the membrane and prohibit both desiccation and collection of serum. Biobrane is available with a variety of pore sizes to regulate wound transudation; these pores allow drainage of the exudate, which is absorbed by an overlying gauze dressing. Biobrane is generally well tolerated and permits the transmission of antibacterial activity from topical antimicrobial agents; the dressing itself has no inherent antibacterial activity. As long as the wound remains clean and there is no seroma, the collagen side of the dressing adheres to the burn surface, effectively sealing it. [17] Any fluid that accumulates beneath the fabric requires prompt removal by aspiration. Unfortunately, Biobrane often separates from the wound before healing is complete; these patients require complete removal of the dressing with alternative topical therapy. [18]

5. *Op-Site* (Smith and Nephew Medical, Massillon, Ohio) is a less than optimal membrane dressing, because fluid collections frequently occur beneath the barrier. Because this dressing is clear, however, it allows for ready wound inspection.

6. *Omniderm* (Jobskin, Jobst Institute, Inc., Toledo, Ohio) is a transparent polyurethane membrane that can be cut to any required shape or contour. [19] It provides an effective barrier dressing but is permeable to oxygen and antibiotic molecules. Omniderm is initially applied dry, but when wet, it becomes pliable and adherent. Pain relief is often remarkable, and the transparent dressing allows ready wound inspection without removal. Fluid accumulation may occur, usually within 1 to 2 days, and is treated with removal and reapplication of a new dressing, which is ultimately left in place for 5 to 10 days. The Omniderm dressing may be covered with flexible net tubular gauze to discourage tampering, especially in children.

7. *Sildimac* (Marion Laboratories, Kansas City, Kan) is an elastic, flexible, conformable sheet consisting of polyethylene glycol 400, poly-2-hydroxyethyl methacrylate, and dimethyl sulfoxide that contains a sustained-release delivery system for silver sulfadiazine. Sildimac is an opaque sheet that adheres to dry surfaces and conforms to body contours. The primary advantage of Sildimac lies in its ability to decrease the frequency of dressing changes while providing sustained-release antimicrobial activity that persists for up to 7 days, although dressing changes are recommended every 4 days. Thus far, Sildimac has been studied only in the management of small, full-thickness burns, but its efficacy seems comparable to twice-daily wound cleansing with application of 1% silver sulfadiazine cream. [20]

**Application of biologic and synthetic dressings.**

All of these dressings are best used on fresh *partial-thickness burns*; placement over a contaminated wound often results in nonadherence and infection. Biologic and synthetic dressings are readily available. These dressings provide immediate relief of pain and are the only dressing method that may actually promote faster wound healing. [2] In

addition, these dressings have the potential for a reduced number of dressing changes.

*Biologic dressings*, such as porcine xenograft, are applied as follows: The dressing is reconstituted as instructed by the manufacturer and is carefully "fit" to the wound. All fluid and air under the graft are pressed out. The dressing is secured with edge tapes and covered with coarse mesh gauze, and then the dressing is wrapped with absorbent gauze and a semi-elastic wrap. With healing, the biologic dressing dries, curls, and separates from the healed edge underneath. The loose edges should be trimmed frequently, any blisters debrided, and fluid accumulations drained.

*Synthetic dressings* are applied after standard wound cleansing. Blisters must be debrided before application of the synthetic dressings. The thicker membranes are cut to size and applied to the burn surface. The edge of the membrane should extend 2 to 3 cm beyond the burn onto the surrounding unburned tissue. Membranes such as DuoDerm can be wrapped about burned digits to permit continued function with limited motion during healing. [16]

Should fluid collect between the debrided burn and the dressing, one attempt at aspiration should be performed. If the fluid reaccumulates beneath the DuoDerm or Biobrane membranes after aspiration, the nonadherent portion of the fabric should be removed. For the first 5 to 8 days, the dressing should be checked at least every other day for adherence. After epithelialization is complete, the dressing begins to separate spontaneously and may be peeled away. [11]

If infection develops under a synthetic or biologic dressing, the dressing should be removed, the wound cultured, and treatment with topical (and possibly oral) antimicrobials instituted. Xenografts should not be used on superficial partial-thickness injuries, because the xenograft tissue may become incorporated into the healing wound in as many as 35% of patients so treated. [21]

Occasionally, application of a permanent dressing--or skin autograft--is appropriately performed in the outpatient setting. Appropriate patient selection is crucial; patients should be healthy and well motivated, with small (<2% TBSA), noncosmetic deep partial-thickness or full-thickness burns. These burns may initially be treated as outlined previously, with the use of a topical antimicrobial agent recommended. The wound should be reevaluated for excision and grafting within 4 days.

## Specific Clinical Issues in Minor Burn Care

### Analgesia

Pain is a much feared feature of any burn injury. Pain relief by the appropriate and judicious use of opioids is of paramount importance in the initial care of burn patients. Analgesia should be provided *before* extensive examination or debridement is performed. Inadequate analgesia is probably the most common ED error in the treatment of burn injuries. This error is most common in children.

Even with minor burns, patients often present with significant pain. Reassurance is important, but adequate analgesia is critical for gaining the patient's cooperation and ensuring comfort. Cooling may dramatically relieve pain temporarily, but parenteral opioids (meperidine, 1 to 2 mg/kg, or morphine, 0.1 to 0.2 mg/kg) are usually required, especially if painful procedures such as debridement and dressing changes are planned. We prefer to use IV opioids (occasionally supplemented with a short-acting benzodiazepine such

as midazolam) for all painful procedures. For complicated debridement or dressing changes, adequate analgesia and sedation (see Chapter 35) is strongly advocated.

Intramuscular (IM) medications following the procedure are helpful for prolonged analgesia. Regional or nerve block anesthesia is an excellent alternative when practical, and when feasible, nitrous oxide analgesia may be used. Oral opioids are inappropriate for initial treatment of significant pain but can be used for continued outpatient analgesia. Local anesthetics may be injected in small quantities when appropriate, such as for the debridement of a deep ulcer or other small burn. Topical analgesics have no role in burn care. A properly designed dressing will do much toward preventing further discomfort after release home; however, home burn care and dressing changes may be quite painful. For this reason an adequate supply of an oral opioid analgesic should be provided, and responsibility should be encouraged in analgesic use.

**Edema**

Minor burns lead to immediate inflammation mediated by the release of histamine and bradykinins, causing localized derangements in vascular permeability, with resultant burn wound edema. This edema is harmful in several ways. First, the increase in interstitial fluid increases the diffusion distance of oxygen from the capillaries to the cells, increasing hypoxia in an already ischemic wound. Second, the edema may produce untoward hemodynamic effects by a purely mechanical mechanism: compression of vessels in muscular compartments. Third, edema has been associated with the inactivation of streptococcicidal skin fatty acids, thus predisposing the patient to burn cellulitis. [22]

The successful management of burn edema hinges on *immobilization* and *elevation*. Most patients are unfamiliar with the medical definition of elevation and are not aware of or convinced of its value. Patient education in this regard is critical; however, certain burns (e.g., burns in dependent body areas) are prone to edema, despite everyone's best intentions. It is for this reason that lower extremity burns in general, and foot burns in particular, are prone to problems.

**Use of Topical Antimicrobials**

Minor burns result in insignificant impairment of normal host immunologic defenses, and burn wound infection is usually not a significant problem. Topical antimicrobials are often used; however, some believe these agents may actually impair wound healing. [23]

Although the procedure is of unproven value, many physicians routinely use antibiotic creams or ointments on even the most minor of burns.

Topical antimicrobials were designed for the prevention and care of burn wound sepsis or wound infection, and there is no convincing evidence that their use alters the course of first-degree burns and superficial partial-thickness injuries. As noted, the burn dressing is the key factor in minimizing complications in all burns. Nonetheless, topical antimicrobials are often soothing to minor burns, and their daily use prompts the patient to look at the wound, assess healing, perform prescribed dressing changes, or otherwise become personally involved in his or her care. Keep in mind that if a topical antimicrobial is used, its effectiveness is decreased in the presence of proteinaceous exudate, necessitating twice-daily dressing changes if the antimicrobial benefit of topical therapy is to be realized. In reality, once-daily dressing changes are more practical and are commonly prescribed, and there are no data to indicate that this regimen is inferior.

All full-thickness burns should receive topical antimicrobial therapy, because the eschar and burn exudate are potentially good bacterial culture media, and deep escharotic or subescharotic infections may not be easily detected until further damage is done. All deep partial-thickness injuries likewise benefit from the application of a topical antimicrobial. In deep partial-thickness injuries, re-epithelialization occurs from a few remaining deep epidermal appendages whose protection is important. Clinical studies and culture results support the hypothesis that surface destruction of dermal islands by bacterial enzymes and catabolic processes has the potential to convert a deep partial-thickness injury to a full-thickness injury. [24]

Initial topical therapy is prophylactic. [5] A burn wound infection that develops despite this therapy mandates a change to a different agent. Topical therapy, if chosen, should cover the usual bacteria responsible for burn wound infections (see later discussion of minor burn infections). Although topical agents are an important part of a burn treatment program, they are not substitutes for good local wound care or a careful program of management. Their successful use may prevent the conversion of deep thermal burns to deeper injury and allow better wound healing for earlier (and more successful) skin grafting.

Criteria for choosing a specific topical agent include in vitro and clinical efficacy, toxicity (absorption), superinfection rate, ease and flexibility of use, cost, patient acceptance, and side effects. Note that there are no firm scientific data that convincingly support the use of any specific topical antimicrobial.

**Specific topical agents**

**Silver sulfadiazine (Silvadene).**

This poorly soluble compound is synthesized by reacting silver nitrate with sodium sulfadiazine. It is the most commonly used topical agent and is well tolerated by most patients. It has virtually no systemic effects and moderate eschar penetration, and it is

painless on application.

Silver sulfadiazine is available as a "micronized" mixture with a water-soluble white cream base in a 1% concentration that provides 30 mEq/L of elemental silver. It does not stain clothes, is nonirritating to mucous membranes, and is easily washed off with water. It may be used on the face, but such use may be cosmetically undesirable for open treatment. Its broad gram-positive and gram-negative antimicrobial spectrum includes beta-hemolytic streptococci, *Staphylococcus aureus* and *Staphylococcus epidermidis*, *Pseudomonas* spp., *Proteus* spp., *Klebsiella* spp., Enterobacteriaceae spp., *Escherichia coli*, *Candida albicans*, and possibly *Herpesvirus hominis*.

Silver sulfadiazine often interacts with wound exudate to form a pseudomembrane over partial-thickness injuries. The pseudomembrane is often difficult and painful to remove. Except for term pregnancy and in newborns (i.e., due to possible induction of kernicterus), there are no absolute contraindications to the use of silver sulfadiazine. Allergy and irritation are unusual, although there is a potential cross-sensitivity between silver sulfadiazine and other sulfonamides.

**Mafenide acetate (Sulfamylon).**

Mafenide is another broad-spectrum antimicrobial that has excellent activity against *Pseudomonas* and other gram-negative organisms. It

has excellent eschar penetration but is quite painful when applied to sensate burns. Since mafenide is a carbonic anhydrase inhibitor, application to large surface wounds may result in a metabolic acidosis. This agent has been supplanted by other topical antimicrobials in minor burn care; its use should be limited to more serious burn wounds with or at high risk for invasive infection.

**Silver nitrate.**

Silver nitrate is an effective topical antimicrobial, but its popularity has decreased since the 1970s. It is painless on application, has a broad spectrum of antibacterial activity, and has no known bacterial resistance. It is available commercially as a 0.5% solution, but this is often clinically unavailable, necessitating custom compounding. Silver nitrate has several significant disadvantages: It is cumbersome to apply and tends to stain clothing and bandages; it has relatively poor tissue penetration; and it causes leaching of sodium, potassium, and chloride from the wound. Silver nitrate also requires increased nursing care in order to maintain moist bandages.

Silver sulfadiazine, mafenide acetate, and silver nitrate all tend to delay epithelialization and may actually slow burn wound healing. For this reason, these potent agents should be reserved for use in the treatment of extensive, infected, or full-thickness burns. [4]

**Broad-spectrum antibiotic ointments.**

Many nonprescription topical antimicrobials are used for minor burn therapy. Included

are bacitracin zinc ointment, polymyxin B-bacitracin (Polysporin), triple-antibiotic ointments such as polymyxin B-neomycin-bacitracin (Neosporin), and nitrofurazone (Furacin). These are all soothing, cosmetically acceptable for open treatment (such as on the face), and are effective antiseptics. Some researchers caution against agents containing neomycin because of a potential for sensitization.

### Povidone-iodine.

Despite the long record of safety and clinical efficacy this agent enjoys when used on *normal skin and mucous membranes*, its routine use on burns is not widely accepted. It is available commercially for topical use compounded in a water-miscible cream base. This combination is strongly acidic (pH 2.40), and its use may result in significant systemic absorption of iodine. In short, other safer and more readily available topical antimicrobials should be used routinely.

### Gentamicin sulfate.

Gentamicin sulfate is available for topical use as a 0.1% cream or ointment. It is readily absorbed when applied to open wounds and may result in the typical aminoglycoside-induced nephrotoxicity or ototoxicity. Burn specialists eschew its use for fear of the development of resistant strains of *Pseudomonas*.

### Chlorhexidine.

Chlorhexidine gluconate is a skin antiseptic that is widely available commercially in Hibiclens solution. Its prophylactic antibacterial effectiveness is comparable to silver sulfadiazine, but toxicity and absorption characteristics are not fully documented (Dr. Christine Ney, Zeneca Pharmaceuticals, personal communication, 1997). Chlorhexidine may be used as a skin or burn cleanser, but *it is not recommended as a topical agent under dressings*.

Chlorhexidine phosphanilate differs from other salts of chlorhexidine in that the phosphanilate moiety confers broad gram-negative antibacterial activity on the molecule that is not cross-resistant with silver sulfadiazine. [25]

### Aloe vera cream.

Aloe vera cream is commercially available in a 50% concentration with a preservative. It exhibits antibacterial activity against at least 4 common burn wound pathogens: *Pseudomonas aeruginosa*, *Enterobacter aerogenes*, *S. aureus*, and *Klebsiella pneumoniae*. Heck and colleagues compared a commercial aloe vera cream with silver sulfadiazine in 18 patients with minor burns. [26] Healing times were found to be similar, and there was no increase in wound colonization in the aloe vera group as compared with the patients treated with silver sulfadiazine. Aloe vera cream may be a viable, inexpensive option for open, home care of minor burns or skin graft donor sites.

# FOLLOW-UP CARE OF MINOR BURNS

The specifics of outpatient follow-up of minor burns are controversial and often based on physician preference and personal bias rather than on firm scientific data. Follow-up should be individualized for each patient and should be based on the reliability of the patient, the extent of the injury, the frequency and complexity of the dressing changes, and the amount of discomfort anticipated during a dressing change.

If a topical antibiotic agent is used, the dressing should be changed once or twice a day with reapplication of the antibiotic ointment. The wound should be rechecked by a physician after 2 to 3 days and periodically thereafter, depending on compliance, healing, and other social issues. If a dry dressing is opted for, follow-up every 3 to 5 days is usually adequate. The purpose of any burn dressing changes or home care regimen is defeated if the patient cannot afford the material or is not instructed in the specifics of burn care. Many EDs supply burn dressing material on patient release. (A complete pack includes antibiotic cream, gauze pads [fluffs], an absorbent gauze roll, a sterile tongue blade to apply cream, and tape.) Writing a prescription and merely stating that the dressing should be changed daily is often futile.

Daily home care can be performed by the patient with help from a family member or visiting nurse (Table 41-3) . The dressing may be removed each day and gently washed with a clean cloth or a gauze pad, tap water, and a bland soap. Sterile saline and expensive prescription soaps are not required. A tub or shower is an ideal place to gently wash off burn cream. The affected area may be put through a

| **TABLE 41-3** -- How to Change a Burn Dressing at Home: Patient Instructions |
| --- |
| 1. Take pain medicine ½ hour before dressing change if you find dressing changes a painful procedure. |
| 2. If the burn is on the hand or foot or other area difficult to reach, have someone else help you. |
| 3. Have all material available. Gloves may be worn. |

| |
|---|
| 4. Remove the dressing and rinse off all burn cream or ointment with tap water, under a shower, or in the bathtub. The area can be gently washed with mild soap and a clean cloth or gauze pads. |
| 5. Look at the burn and assess the healing, blistering, and amount of swelling. Note any signs of infection. |
| 6. Gently exercise the area through range of motion. |
| 7. Apply the burn ointment with a sterile tongue blade. |
| 8. Cover the cream with fluffed-up gauze. |
| 9. Wrap the area in bulky gauze. |
| 10. Repeat this dressing change daily. |

gentle range of motion during dressing changes. After the burn is cleaned, it is inspected by the patient. The patient is instructed to return if signs or symptoms of infection, significant blistering, or skin slough develop. Following complete removal of the old cream, a new layer is applied with a sterile tongue blade and covered with absorbent gauze.

If the undermost fine mesh gauze of a dry dressing is dry and the coagulum is sealed to the gauze, the patient should simply reapply the overlying gauze dressing. If the wound is macerated, the fine mesh gauze should be removed and the wound cleaned and redressed. The patient is instructed *not to remove a dry adherent fine mesh gauze from the underlying crust*. When epithelialization is complete, the crust will separate, and the gauze can be removed at that time. Dryness in healing skin may be treated with mild emollients such as Nivea (Beiersdorf, Inc., Norwalk, Conn) or Vaseline Intensive Care lotion (Chesebrough Ponds, Inc., Greenwich, Conn). Natural skin lubrication mechanisms usually return by 6 to 8 weeks. [12] Excessive sun exposure should be avoided during wound maturation, as this may lead to hyperpigmentation; a

commercially available sun block should be used. Exposure of the recently healed burned area to an otherwise minor trauma (chemicals, heat, sun) may result in an exaggerated skin response. Pruritus may be treated with oral antihistamines or a topical moisturizing cream.

Deep partial-thickness burns, along with small third-degree burns, may be initially managed in the outpatient setting. Topical antimicrobials are recommended; mafenide (Sulfamylon) cream may be useful in the care of small, full-thickness burns because it is capable of penetrating the entire eschar and is present in subjacent tissues at bactericidal levels. [13]

**Outpatient Physical Therapy for Burn Care**

When the hospital's outpatient physical therapy department is equipped to treat minor burns, it is prudent to consider this option. Many centers make available daily burn treatment, consisting of dressing changes, whirlpool debridement, and range-of-motion exercises. When patients are unable to handle their own burns at home, this can be an invaluable adjunct. An additional advantage is that medically trained personnel evaluate the burn daily, thereby decreasing physician visits and enabling identification of problems prior to the development of serious complications. Generally all that is required from the physician is to write a prescription for "burn care and dressing changes" and set up the appointment.

**Burn Healing**

Follow-up care will in part be guided by expectations of burn healing and observed healing. The following discussion is intended to serve as a general guide. However, burn healing is different from that of other wounds. [2] The timing is often variable, but it is proportional to burn depth. The inflammatory phase lasts 3 to 7 days (at times longer) and, if the burn is severe enough, is accompanied by the release of histamine and bradykinins, along with complement degradation. This degradation of complement may lead to immunologic, coagulation, and metabolic aberrations.

Within 1 to 3 weeks, neovascularization of the burn occurs, accompanied by fibroblast migration. Macrophages begin to replace the tissue neutrophils. Collagen production begins, but the molecules are often laid down in random fashion, leading to a scar. Re-epithelialization follows, but the presence of necrotic tissue and eschar impedes all aspects of wound healing. The amount of scar tissue produced is directly related to healing time. Burns requiring <16 days to heal generally do not scar excessively. [2]

Healing in superficial partial-thickness burns occurs within 10 to 14 days. After healing, the new epithelial layer tends to dry easily and crack. Using bland, lanolin-containing creams for 4 to 8 weeks following healing alleviates this problem. Deep partial-thickness burns heal by re-epithelialization from the wound edge and from residual dermal elements. Healing is slow and often unsatisfactory, frequently taking longer than 3 weeks, producing an unstable epithelium that is prone to hypertrophic scarring and contractures. This is a particular problem in burns that extend across joints. Burns that take longer than 2 to 3 weeks to heal are prone to infection; hence, topical

antimicrobials should be used. Because these burns often heal in complicated fashion, they should be considered for referral to expedite early excision, grafting, and physical therapy.

Full-thickness burns can heal only by contraction and epithelialization at the wound edge. Burns larger than 2 to 3 cm must be excised and grafted.

Cosmetic and functional recovery follows complete epithelialization of a partial-thickness injury or successful skin grafting of a full-thickness burn. The ultimate goal is to prevent scar thickening; achieve and maintain optimal range of motion; and prevent secondary environmental damage to the skin, particularly from sun exposure. [13] Nonscented skin lotions may be used after epithelialization to keep the burn scar soft. Compression dressings are especially helpful in preventing scar thickening. Repeated evaluations are important, because burn contractures can occur up to 12 months after the injury. Nighttime splinting is useful in maintaining full extension of joints.

## SPECIAL MINOR BURN CARE CIRCUMSTANCES

### Blisters

The management of blisters in minor burns is controversial. Management arguments are generally theoretic or emotional; the ultimate outcome of a minor burn is rarely determined by how one deals with blisters. Intact blisters do offer a physiologic dressing that rarely becomes infected; however, most large blisters spontaneously rupture after 3 to 5 days and eventually require debridement. When the integrity of the blister is breached, the fluid becomes a potential culture medium. Clinical choices include debridement, aspiration, or simply leaving the blister intact.

Some studies suggest that intact burn blisters may allow for reversal of capillary stasis and less tissue necrosis. [2] Madden and colleagues have shown that burn exudate (as contained within intact blisters) is beneficial for the stimulation of epidermal cell proliferation. [27]

Swain and colleagues demonstrated that the density of wound colonization with microorganisms was much lower in minor burns with blisters left intact. [28] They also found that 37% of patients with aspirated blisters experienced a

reduction in pain versus none of those whose blisters were unroofed. Other investigators believe that undressed wounds with debrided blisters have additional necrosis secondary to desiccation, which can convert a partial-thickness burn to a full-thickness injury. [3] Finally, intact blisters clearly provide some pain relief, as evidenced by a sudden increase in pain immediately following debridement. Increased pain should be anticipated and analgesia offered as appropriate when debridement is necessary. We suggest the guidelines in Table 41-2 as a general approach to burn blisters.

**Minor Burn Infections**

There are bacteria on the skin at all times--normal skin usually harbors nonvirulent pathogens such as *S. epidermidis* and diphtheroids. Therefore, all burns are *contaminated* but not necessarily *infected*. Thermal trauma results in a coagulative necrosis. Burn wounds therefore contain a variable amount of necrotic tissue, which, if infected, acts much as an undrained abscess, preventing access of antibiotics and host defensive factors.

The microbial flora of outpatient burns varies with time after the burn. Shortly after injury, the burn becomes colonized with gram-positive bacteria such as *S. aureus* and *S. epidermidis*. After this period of time there is a gradual shift toward inclusion of gram-negative organisms, 80% of which originate from the patients' own gastrointestinal tract. [4] Common organisms seen on days 1 to 3 include *S. epidermidis*, beta-hemolytic streptococci, *Bacillus subtilis*, *S. aureus*, enterococci, *Mima polymorpha*, *Enterobacter* spp., *Acinetobacter* spp, and *C. albicans*. One week after the burn, these organisms may be seen along with *E. coli*, *P. aeruginosa*, *Serratia marcescens*, *K. pneumoniae,* and *Proteus vulgaris*.

Anaerobic colonization of burn wounds is rare unless there is much devitalized tissue, as occurs in a high-voltage electrical injury. [29] For this reason, routine anaerobic cultures are generally unnecessary in an assessment of infective organisms that produce minor infections.

Most superficial wounds that have been treated properly do not get infected. However, it is sometimes difficult to differentiate wound infection from the normal healing process, as both involve pain, edema, and erythema. Early (days 1 to 5) burn infections are generally caused by gram-positive cocci, especially beta-hemolytic streptococci. Streptococcal cellulitis is characterized by marked, spreading erythema extending outward from the wound margins. Despite the plethora of organisms and the presence of some gram-negative pathogens noted in superficial burn cultures, first-line treatment in the normal host is oral penicillin, 1 to 2 g/day.

Effective topical treatment at the time of initial burn care and subsequent dressing changes is meant to delay bacterial colonization, maintain the wound bacterial density at low levels, and produce a less diverse wound flora. Because outpatient management of burns should be attempted only when the risk of infection is minimal, the use of systemic antibiotics is generally unnecessary for minor burns even in the setting of delayed treatment, diabetes, and steroid use. [30] Parenteral antibiotics in the management of minor burns have been recommended for patients undergoing an autograft procedure and for patients with a sore throat or other known streptococcal infection in whom penicillin is otherwise indicated. [31]

In minor burn care, wound cultures are not required or recommended. It is useless, for example, to culture blister fluid in the patient who presents for emergency care immediately after a thermal injury. Cultures are necessary only when overt infection develops, especially when this occurs while a topical or systemic antibiotic is being

used. Cultures may also be of benefit when the infected wound is old, when hygiene is poor, or when there are old abrasions nearby. [32] Swab surface cultures are generally eschewed. Although they may adequately reflect wound flora, falsely sterile cultures are relatively frequent. These cultures do not reflect deep burn flora and give no quantitative information.

Sterile wound biopsy for culture is most satisfactory for the assessment of intraescharotic, subescharotic, or invasive infections and allows for quantification of bacterial flora. If a wound culture is taken, it should be obtained from the deepest or worst-appearing area of the burn.

Surface bacterial densities >10 [5] /cm [2] or tissue bacterial densities >10 [5] /g correlate with invasive infection. Surface colonization may be treated with an alternative topical agent, but truly invasive infection warrants the administration of systemic antibiotics. Generally, the infectious process resolves in 24 to 48 hours.

## Foot Burns

Despite their relatively small surface area, foot burns tend to heal poorly, usually due to excessive edema; therefore they are formally categorized as major burns. Foot burns are the most common burn category to fail outpatient therapy and subsequently require admission and inpatient care. Zachary and coworkers reported on a series of 104 patients with foot burns. [33] No patient admitted on the day of injury developed burn cellulitis; in contrast, 27% of delayed admission patients had cellulitis. Their study also noted a higher incidence of hypertrophic scarring and need for skin grafting in the delayed admission group. Overall, fewer days of hospitalization were required for the initially admitted group.

Specific problems in the care of foot burns include pain, wound drainage, difficulty in changing dressings without help, inability of even motivated patients to comply with requirements for elevation, and prolonged convalescence. Hospital admission allows for splinting, intensive local burn care, physical therapy, and bed rest with elevation, which minimizes edema. For these reasons, initial admission for all but the most minor of foot burns is advised.

## Hand Burns

Because of their functional importance, hand burns can be a devastating injury, despite involvement of a relatively small TBSA. Hand function is critical, regardless of whether the patient is dealing with loss of use during healing, later limitation by scar contractures, a long-term appearance change, or loss due to amputation. [34]

As with other burns, the depth and extent of the burn determine the severity of the injury. The entire surface of one hand represents only 2.5% TBSA, yet even small burns can cause a disproportionate functional loss. Deep partial- or full-thickness hand burns, even if quite small, often warrant referral for early excision and grafting in order to limit scarring and maintain function. The skin on the dorsum of the hand is thinner than that

on the palm and is more susceptible to burn injury, but must remain flexible to allow

for finger motion. Any exposed tendon or bone, such as may be seen with an electrical burn, constitutes a true fourth-degree injury, which requires either flap closure or amputation in order to heal the wound. Many of the issues complicating outpatient management of foot burns are relevant to the care of hand burns. After initial burn cooling, the wound should be gently cleansed with mild soap. Any loose skin or ruptured blisters should be gently debrided, rinsed, patted dry, and covered with a topical antimicrobial agent and a nonadherent, bulky gauze dressing. The fingers should be carefully separated and bandaged individually. Small, intact blisters that do not interfere with hand function should be left intact to serve as a biologic dressing. Elevation of the hand is very important in the first few days after a burn injury in order to minimize edema. Deep partial- or full-thickness burns to the dorsum of the hand should be splinted after bandaging to avoid the development of contractures or a boutonniere deformity.

Hospital admission should be considered for all hand burns, particularly full-thickness injuries and circumferential burns involving the digits. If outpatient treatment is attempted, the patient must be given comprehensive instructions and should have the resources available to perform twice-daily dressing changes and range-of-motion exercises of the fingers and wrist during these dressing changes. An initial follow-up visit should be arranged in 48 to 72 hours, but the patient should be encouraged to return if there is development of a burn cellulitis, worsening pain, fever, or lymphangitis. Ideally, the patient should be seen twice in the first week after injury and once a week after that until the burn is healed.

**Facial Burns**

Facial burns commonly result from unexpected ignition flash burns (e.g., from a stove or charcoal grill) or from car radiator accidents. Facial burns often result in significant edema, which may compromise vision. Concurrent corneal injury can lead to the development of purulent conjunctivitis, with the risks of corneal ulceration and perforation. Fluorescein staining and slit-lamp examination should be used to confirm the diagnosis of suspected corneal injury. The treatment of a corneal injury involves irrigation, topical ophthalmic antibiotic ointment, and consideration of eye patching (see Chapter 67) . Facial burns are otherwise treated with an open technique. Patients are instructed to wash the face 2 to 3 times a day with a mild soap and then apply a thin layer of antibiotic ointment, such as bacitracin zinc. Neck burns are treated similarly. All patients presenting with head or neck burns should be carefully evaluated for a concomitant inhalation injury. Such patients may present with direct evidence of injury, such as oral burns, blisters, soot, or hyperemia, or with indirect evidence, such as dyspnea, wheezing, arterial hypoxemia, or an elevated carboxyhemoglobin level. The definitive diagnostic test for inhalation injury is fiberoptic bronchoscopy. [35] Inpatient care should be considered for all patients with significant facial burns.

Corneal contact burns, as from accidental contact with a curling iron, often present rather dramatically, with opacified, "heaped-up" corneal epithelium. Despite their appearance, the end result is usually excellent. Treatment is the same as for a corneal

abrasion. [36]

## Abuse of Children and Elderly Individuals

Recognition of the possibility of deliberate abuse by burning in the pediatric and geriatric populations is essential. In addition, children under 2 years old have a thinner dermis and a less well developed immune system than do adults. Elderly patients (>65 years old) likewise tolerate burns poorly, and these 2 populations are the most prone to abuse, often by family members. For these reasons, both groups of patients often require inpatient care. [8]

The majority of abused children are 18 to 36 months old, and for unknown reasons the majority are male. [23] Immersion burns are a common type of abuse. These are characterized by circumferential, sharply demarcated burns of the hands, feet, buttocks, and perineum. Cigarette burns and burns from hot objects such as irons should be obvious. Contact burns on "nonexploring" parts of the child also warrant suspicion.

## Burns in Pregnancy

There is little information in the literature concerning the special problems of the pregnant burn victim. Ying-bei and Ying-jie reported on 24 pregnant burn patients representing a wide range of burn severity. [37] Complications of the burn injuries included abortion and premature labor, although all patients in this series with burns covering <20% TBSA did well and delivered living full-term babies.

As the resistance of pregnant women to infection is lower than that of nonpregnant women, control of burn wound infection is paramount. Gestational age appears to have no direct bearing on prognosis. Silver sulfadiazine cream should be avoided near term because of the potential for kernicterus.

# SPECIFIC BURNING AGENTS

## Hot Tar Burns

Asphalts are products of the residues of coal tar commonly used in roofing and road repair. These products are kept heated to approximately 450 °F. When spilled onto the skin, the tar cools rapidly, but the retained heat is sufficient to produce a partial-thickness burn. Fortunately, full-thickness burns are unusual. Cooled tar is nonirritating and does not promote infection. When cooled tar is physically removed, the adherent skin is usually avulsed (Fig. 41-6) . Careless removal of the tar may inflict further damage on burned tissues. Agents such as alcohol, acetone, kerosene, or gasoline have been used to remove the tar, but these are flammable and may cause additional skin damage or toxic response secondary to absorption.

*There is no great need to meticulously remove all tar at the first visit.* Obviously devitalized skin can be debrided, but adherent tar should be emulsified or dissolved rather than manually removed (Fig. 41-7) . Polyoxyethylene sorbitan (Tween 80 or

polysorbate 80) is the water-soluble, nontoxic emulsifying agent found in Neosporin and several other topical antibiotic ointments. It is a complex mixture of ethers, esters, and sorbitol anhydrides that possesses excellent hydrophilic and lyophilic characteristics when used as a nonionic, surface-active emulsifying agent. With persistence, most tar may be removed (emulsified) on the initial visit.

Another household product (De-Solv-It multi-use solvent) also appears promising for topical ED use. [38] The De-Solv-It product has a surface-active moiety that wets the chemical's surface and emulsifies tar and asphalt. Since the latter product is itself a petroleum-based solvent, it should be applied only briefly, and the operator should wear gloves and protective eyewear during application. It should be used only for external exposure to tar or asphalt.

Many physicians prefer instead to emulsify the tar on an outpatient basis. A generous layer of polysorbate-based ointment can be applied under a bulky absorbent gauze dressing. The patient is then released home, and the residual is easily washed off after 24 to 36 hours (Fig. 41-8) . Once the residual tar is removed, the wound is treated like any other burn.

Shur-Clens, a nontoxic, nonionic detergent, also works well for tar burn wound cleansing, as do mineral oil; petrolatum; and Medisol (Orange-Sol, Inc, Chandler, Ariz), a petroleum-citrus product.

**Chemical Burns**

Chemical burns usually occur in the workplace, and the offending substance is usually well known. More than 25,000 chemicals currently in use are capable of burning the skin or mucous membranes. Commonly used chemical agents capable of producing skin burns are shown in Table 41-4 .

Injury is caused by a chemical reaction, rather than a thermal burn. [39] Reactions are classified as oxidizing, reducing, corrosive, desiccant, or vesicant or as protoplasmic poisoning. The injury to skin continues until the chemical agent is physically removed or exhausts its inherent destructive capacity. The degree of injury is based on chemical strength,

| **TABLE 41-4** -- Commonly Used Acids and Alkalis | |
|---|---|
| **Acids** | **Alkalis** |
| Picric | Sodium hydroxide |

| | |
|---|---|
| Tungstic | Ammonium hydroxide |
| Sulfosalicylic | Lithium hydroxide |
| Tannic | Barium hydroxide |
| Trichloroacetic | Calcium hydroxide |
| Cresylic | Sodium hypochlorite |
| Acetic | |
| Formic | |
| Sulfuric | |
| Hydrochloric | |
| Hydrofluoric | |
| Chromic | |

concentration, and quantity; duration of contact; location of contact; extent of tissue penetration; and mechanism of action.

Immediate flushing with water is recommended for all chemical burns, with the exception of those caused by alkali metals. Flushing serves to cleanse the wound of

unreacted surface chemical, dilute the chemical already in contact with tissue, and restore lost tissue water. Leonard and colleagues clearly demonstrated that patients receiving immediate copious water irrigation for chemical burns showed less full-thickness burn injury and a 50% reduction in time of hospital stay. [40]

**Acid and Alkali Burns**

Alkalies cause saponification and liquefactive necrosis of body fats. Alkaline burns are *penetrating* and cause much tissue destruction. With acid burns, tissue coagulation produces a thick eschar that limits the penetration of the agent. Desiccant acids, such as sulfuric acid, create an exothermic reaction with tissue water and can cause both chemical and thermal injury. With extensive immersion injuries, acids may be systemically absorbed, leading to systemic acidosis and coagulation abnormalities.

Chemical burns may be excruciatingly painful for long periods of time. Discomfort can be out of proportion to what one might expect from the depth or extent of the burn.

The emergency care team should remove all potentially contaminated clothing. Any dry (anhydrous) chemical should be brushed off the patient's skin. The involved skin should be irrigated with large amounts of water under low pressure. Any remaining particulate matter should be carefully debrided during irrigation.

Strong alkali burns may require irrigation for 1 to 2 hours before the tissue pH returns to normal. Some recommend that *after extensive irrigation*, if the burn continues to feel "slippery" or tissue pH has not returned to normal, chemical neutralization may be helpful. [41] [42] Given that any heat of neutralization will be carried away with the irrigation solution, [43] prompt irrigation with a dilute acid (e.g., vinegar; 2% acetic acid) may hasten neutralization and patient comfort.

**Wet Cement Burns**

Portland cement is an alkaline substance whose major constituent is calcium oxide (64%), combined with oxides of silicon, aluminum, magnesium, sulfur, iron, and potassium. There is considerable variability in the calcium oxide content of different grades of cement, with concrete having less and fine-textured masonry cement having more. [40] The addition of water exothermically converts the calcium oxide to calcium hydroxide, a strongly corrosive alkali with a pH of 11 to 13. As the cement hardens, the calcium hydroxide reacts with ambient carbon dioxide and becomes inactive.

Both the heat and the $Ca(OH)_2$ produced in this exothermic reaction can result in significant burns. Because of its low solubility and consequent low ionic strength, a long exposure to calcium hydroxide is required to produce injury. This usually occurs when a worker spills concrete into his or her boots or kneels in it for a prolonged period. The burn wound and the resultant protein denaturation of tissues produces a thick, tenacious, ulcerated eschar. Concrete burns are insidious and progressive. What may appear initially as a patchy, superficial burn may in several days become a full-thickness injury requiring excision and skin grafting. [44] The pain of these burns is often severe

and more intense than the appearance of the wound might suggest (Fig. 41-9) . Interestingly, many workers are not warned of the dangers of prolonged contact with cement, and because initial contact with cement is usually painless, exposure may not be realized until the damage is done.

Treatment is as follows: Any loose particulate cement or lime is brushed off, contaminated clothing is removed, the wound is copiously irrigated with tap water (the pH of the effluent is tested and irrigation continued if the effluent is still alkaline), compresses of dilute acetic acid (vinegar) may be applied to neutralize the remaining alkali and provide pain relief after irrigation, and antibiotic ointment is applied to the eschar during the early postburn period.

Sutilains ointment (Travase, Flint Pharmaceuticals, Deerfield, Ill) is often recommended because it contains proteolytic enzymes and helps speed eschar separation, but any common topical burn preparation is acceptable. The depth of burns from wet cement can be difficult to assess in the first several days. If it becomes apparent that the burns are full-thickness burns, early excision and skin grafting are recommended.

Cement burns should be differentiated from cement dermatitis, which is far more common. The latter is a contact sensitivity reaction, probably due to the chromates present in cement. The contact dermatitis can initially be treated as a superficial partial-thickness burn.

**Air Bag Keratitis**

Recently enacted safety legislation has mandated increased use of air bags to protect automobile occupants in the event of collision. The automobile air bag is a rubberized nylon bag that inflates on spark ignition of sodium azide, yielding nitrogen gas, ash, and a small amount of sodium hydroxide. If the air bag ruptures, the alkaline contents of the bag are dispersed as a fine, black powder that usually causes no problems unless the eyes are exposed. Patients present with clinical evidence of a chemical keratoconjunctivitis, including photophobia, tearing, redness, and decreased visual acuity. The tear pH is usually elevated, and there may be a small amount of particulate material in the fornices. [45]

The severity of an ocular alkaline burn is related to the duration of exposure and the concentration and pH of the chemical. For this reason, prompt, copious irrigation of the eyes with frequent assessment of tear pH is essential to prevent or minimize the injury (see Chapter 67) . A rising pH suggests that trapped particulate matter is releasing additional chemical. Corneal edema and conjunctival blanching are signs of serious injury and necessitate immediate ophthalmologic consultation.

**Hydrocarbon Burns**

Hydrocarbons are capable of causing severe contact injuries by virtue of their irritant, fat-dissolving, and dehydrating properties. Cutaneous absorption may cause even more dangerous systemic effects. Gasoline, the usual agent involved, is a complex mixture of

C4 to C11 alkane hydrocarbons and benzene; the hydrocarbons appear to be the major toxic agent. Lead poisoning caused either by absorption through intact skin or burns from "leaded" gasoline exposure have been previously reported but are currently quite rare, as unleaded gasoline has virtually replaced the leaded version for most purposes. [46]

Depth of injury is related to the duration of exposure and concentration of the chemical agent. Gasoline immersion injuries resemble scald burns and are usually partial thickness. [47] Occasionally, gasoline-injured skin exhibits a pinkish brown discoloration, possibly related to dye additives. A common source of exposure is a comatose patient from a motor vehicle crash who had been lying in a pool of gasoline.

The lungs are the usual site of systemic absorption and are often the only major route of excretion. The resultant high pulmonary concentrations may lead to pulmonary hemorrhages, atelectasis, and adult respiratory distress syndrome (ARDS). Treatment of hydrocarbon burns includes the following: removal of contaminated clothing, prolonged irrigation or soaking of the contaminated skin, early debridement in significant burns caused by lead-containing gasolines (to reduce systemic lead absorption), and use of topical antibiotic ointments.

### Phenol Injury

Phenol is a highly reactive aromatic acid alcohol that acts as a corrosive. Carbolic acid, an earlier term for phenol, was noted to have antiseptic properties and was used as such by Joseph Lister in performing the first antiseptic surgery. Hexylresorcinol, a phenol derivative, is in current use as a bactericidal agent. Phenols, in strong concentrations, cause considerable eschar formation, but skin absorption also occurs and can cause systemic effects such as central nervous system depression, hypotension, hemolysis, pulmonary edema, and death. Interestingly, phenol acts differently from other acids in that it penetrates deeper when in a dilute solution than when in a more concentrated form. [39] Therefore, irrigation with water is suboptimal for phenol burns, but because water commonly is readily available, it is frequently used for irrigation.

Full-strength polyethylene glycol (PG 300 or 400) is more effective than water alone in removing phenolic compounds and should be obtained and used after water irrigation has begun. Polyethylene glycol is nontoxic and nonirritating and may be used anywhere on the body. When immediately available, polyethylene glycol can be used to remove the surface chemical before water irrigation (and chemical dilution) is begun.

### Hydrofluoric Acid Injury

Hydrofluoric acid (HFA) is one of the strongest inorganic acids known; it has been widely used since its ability to dissolve silica was discovered in the late 17th century. [48] Currently, HFA is used in masonry restoration, glass etching, and semiconductor manufacturing; for control of fermentation in breweries; and in the production of plastics and fluorocarbons. It is also used as a catalyst in petroleum alkylating units. It is available in industry as a liquid in varying concentrations up to 70%. It is also found in

many home rust-removal products, and in aluminum brighteners and heavy-duty cleaners in concentrations of <10%.

Although HFA is quite corrosive, the hydrogen ion plays a relatively insignificant role in the pathophysiology of the burn injury. The accompanying fluoride ion is a protoplasmic poison that causes *liquefaction necrosis and is notorious for its ability to penetrate tissues and cause delayed pain and deep tissue injury.* This acid can penetrate through fingernails and cause nailbed injury. With home products, the unwary user does not realize that the substance is caustic until the skin (usually the hands and fingers) is exposed for a few minutes

to hours, at which time the burning begins and becomes progressively worse. At this point the damage is done, and the absorbed HFA cannot be washed off. With higher-strength industrial products, symptoms are almost immediate.

The initial corrosive burn is due to free hydrogen ions; secondary chemical burning is due to the tissue penetration of fluoride ions. Fluoride is capable of binding cellular calcium, resulting in cell death and liquefaction necrosis. The ionic shifts that result, particularly shifts of potassium, are believed to be responsible for the severe pain associated with HFA burns.

In high concentrations, the fluoride ions may penetrate to the bone and produce demineralization. Skin exposure to concentrated HFA involving as little as 2.5% TBSA can lead to systemic hypocalcemia and death from intractable cardiac arrhythmias; it has been calculated that exposure to 7 mL of anhydrous HFA (HFA gas) is capable of binding *all* of the free calcium in a 70-kg adult. [49] [50] If the hands are exposed, the acid characteristically penetrates the fingernails and injures the nailbed and cuticle area. As with most caustics, *the pain is generally out of proportion to the evident external physical injury.* HFA burns produce variable areas of blanching and erythema, but rarely are blisters or skin sloughing seen initially. Skin necrosis and cutaneous hemorrhage may be noted in a few days.

Immediate treatment should begin with copious irrigation with water. Another approach is to wash the area with a solution of iced magnesium sulfate (Epsom salts) or a 1:500 solution of a quaternary ammonium compound such as benzalkonium chloride (Zephiran) or benzethonium chloride (Hyamine 1622). Magnesium and calcium salts form an insoluble complex with fluoride ions, preventing further tissue diffusion. Unfortunately, topical preparations are often ineffective in limiting injury or controlling pain.

If there is no or only minimal visible evidence of skin injury and minimal pain, the burn may be dressed with topical calcium gluconate paste. This is not commercially available in the United States but is easily compounded in the pharmacy by mixing 3.5 to 7.0 g of pulverized calcium gluconate with 5 oz of a water-soluble lubricant such as K-Y jelly. This will form a thick paste with a calcium gluconate concentration of 2.5 to 5.0%. Some have suggested dimethyl sulfoxide (DMSO) as a vehicle to aid in skin penetration of the calcium. Plastic wrap (e.g., Saran wrap) is used over a standard dry burn dressing to cover the calcium paste on the limbs; a vinyl or rubber glove is used over the paste

when used on the hands. The wound should be completely redressed and the paste reapplied every 6 hours for the first 24 hours. A digital or regional nerve block with long-acting bupivacaine is an excellent way to provide prolonged pain relief if the hands are involved. Otherwise, oral opioids are generally required. If bullae or vesicles have formed, these should be debrided to decrease the amount of fluoride present, and the wound should then be treated as any partial-thickness burn. Burns with HFA of <10% strength will heal spontaneously, usually without significant tissue loss, but pain and sensitivity of the fingertips may persist for 7 to 10 days. In addition, the fingernails may become loose.

The presence of significant skin injury or intense pain implies penetration of the skin by fluoride ions. This scenario is particularly common with exposure to HFA solutions in concentrations of 20%, but tissue injury can occur with prolonged exposure to less concentrated products.

Initial treatment of a more concentrated exposure begins as described above and includes immediate debridement of necrotic tissue to remove as much fluoride ion as possible. Following this, a 10% solution of calcium gluconate *(note: avoid calcium chloride for tissue injections)* is injected intradermally and subcuticularly with a 30-ga needle about the exposed area, using about 0.5 mL/cm [2] of burn. Pain relief should be almost immediate if this therapy is adequate. Since the degree of pain is a measure of the effectiveness of treatment, the use of anesthetics, especially by local infiltration, may be deleted if the burn is on the arm or leg. HFA can penetrate fingernails without damaging them. Soft tissue can be injected without prior anesthesia, but if the fingertips or nailbeds are involved, they may be injected after a digital nerve block has been performed (Fig. 41-10) . Before anesthesia and prior to injecting calcium, the patient can outline the affected areas with a pen to ensure accurate injection of the antidote (see Fig. 41-10 *B*). Although some investigators recommend that the fingernails be removed routinely, we strongly advise *against* this unless the nails are very loose or there is obvious necrosis of the nailbed. Fingers are best injected with a 25- or 27-ga needle (a tuberculin syringe works well). [51] Nails frequently become loose in a few days, but often they return to normal and do not require removal, particularly when lower concentration nonindustrial products are involved.

Although calcium gluconate infiltration is effective, the technique has certain limitations. Injections are painful, and the calcium gluconate solution itself causes a burning sensation. Because of the volume restrictions, not enough calcium may be delivered to bind all the free fluoride ions present. For example, 0.5 mL of 10% calcium gluconate contains 4.2 mg (0.235 mEq) of elemental calcium, which will neutralize only 0.025 mL of 20% HFA.

Several authorities have advocated intra-arterial calcium infusions in the treatment of serious HFA burns of the extremities. [49] [52] Although very effective, this technique is not recommended for burns secondary to dilute HFA (i.e., concentrations <10%), because morbidity is usually quite mild. When using this technique, 10 mL of 10% calcium gluconate are diluted in 50 mL of a 5% dextrose-and-water solution. The dilute solution is given by a slow infusion into an arterial catheter. If only the radial 3 digits are involved, only the radial artery need be cannulated. Otherwise, a percutaneous catheter

is inserted into the brachial artery, and *the solution is slowly infused over 4 hours.* At this point, the catheter is left in place, and the patient is observed. If pain returns at any time over the next 4 hours, the infusion is repeated. If the patient is pain free over the 4-hour observation period, the burn is dressed, and the patient is released home.

Advantages of the intra-arterial method are elimination of the need for painful SQ injections and avoidance of the volume limitations of the SQ route while providing substantially more calcium to neutralize the fluoride. Both the chloride and gluconate salts of calcium have been used intra-arterially. A volume of 10 mL of 10% calcium gluconate contains 4.7 mEq of calcium, whereas the same volume of 10% calcium chloride contains 14 mEq of calcium. Thus, intra-arterial calcium chloride may provide nearly 3 times as much neutralizing calcium equivalent as the gluconate salt; however, because calcium chloride can irritate tissues, it is not recommended for SQ use. Disadvantages of intra-arterial calcium therapy include the possibility of local arterial spasm (which can be treated with vasodilators such as phentolamine or removal of the catheter), local arterial injury or thrombus, and the long duration of treatment required.

HFA burns to the eye are a potentially devastating injury that deserves special mention. Ocular exposure to liquid or gaseous HFA will result in severe pain, tearing, conjunctival inflammation, and corneal opacification or erosion. Complications include decreased visual acuity, globe perforation, uveitis, glaucoma, conjunctival scarring, lid deformities, and keratitis sicca. Optimal therapy for ocular HFA burns, other than initial irrigation, is unknown. Irrigation may be performed with water, isotonic saline, or magnesium chloride. [53] Anecdotal reports have described promising results using a 1% solution of calcium gluconate in normal saline, with instillation of 2 to 3 drops into the injured eye every 3 hours for 2 days. Topical antibiotics and cycloplegics, along with light pressure patching, are also recommended. The use of topical steroids has been advocated by some in order to lessen corneal fibroblast formation, but other attempted therapies such as subconjunctival injections of calcium gluconate and ocular irrigation with quaternary ammonium compounds have been associated with additional injury. [54]

**Chromic Acid Injury**

Chromium compounds are used extensively in industry, mainly in metallic electroplating. Chromic acid is commonly used in concentrated solutions containing up to 25% sulfuric acid. It causes sufficient skin damage to allow absorption of the toxic chromium ion if intensive irrigation is not undertaken immediately. Heated (60 to 80 °C) chromic acid makes the problem of chromium absorption much worse.

Dichromate salts containing hexavalent chromium are the most readily absorbed and the most toxic because they can cross cell membranes. The mortality rate from these burns is very high if the burn exceeds 10% TBSA. Chromium absorption leads to diarrhea, gastrointestinal bleeding, hemolysis, hepatic and renal damage, coma, encephalopathy, seizures, and disseminated intravascular coagulation.

Treatment includes immediate excision of the burned tissues to lessen the total body dichromate burden. Wounds should be washed with a 1% sodium phosphate or sulfate

solution and dressed with bandages soaked in 5% sodium

thiosulfate solution. These actions reduce the hexavalent chromium ion to the less well absorbed trivalent form. [55]

Chelation therapy with ethylenediaminetetra-acetic acid (EDTA) should be instituted, and IV sodium thiosulfate and ascorbic acid given. Hemodialysis, peritoneal dialysis, or exchange transfusion may be indicated.

**Phosphorus Burns**

White phosphorus is a translucent, waxy substance that ignites spontaneously on contact with air. For this reason, it is usually stored under water. It is used primarily in fireworks, insecticides and rodenticides, and military weapons.

Phosphorus causes both thermal burns from the flaming pieces and acid burns, which result from the oxidation of phosphorus to phosphoric acid. The burns classically emit a white vapor with a characteristic garlic odor. [56]

These burns are treated first with immersion in water, followed by debridement of any gross debris. The wound is then washed with a 1% copper sulfate solution, which reacts with the residual phosphorus to form copper phosphate; the latter appears as black granules and allows for easy debridement. Following debridement, the residual copper is removed by a thorough water rinse, and the wound is dressed and treated as any other burn.

**Elemental Alkali Metal Burns**

The commonly encountered alkali metals (sodium, lithium, and potassium) are highly reactive with water and with water vapor in air, producing their respective hydroxide with liberation of hydrogen gas. Therefore, water should never be used for extinguishing or debridement of the metal. A class D fire extinguisher or plain sand may be used for smothering the fire, followed by application of mineral oil or cooking oil to isolate the metal from water and allow safe debridement. The burn is then treated as an alkali burn.

Magnesium burns in a less intense fashion but otherwise acts as do other alkali metals. These burns may be particularly injurious, however, because if all of the metallic debris is not removed, the small ulcers that form will slowly enlarge until they become quite extensive.

# EMERGENCY ESCHAROTOMY

Full-thickness burns result in an eschar that is inelastic and may become restrictive. During fluid resuscitation and as a direct result of transcapillary extravasation of fluid from thermal injury, intracellular and interstitial edema progresses. As the soft tissues become edematous and pressure rises under the unyielding eschar, first venous and

then lymphatic, capillary, and ultimately arterial flow to the underlying and distal unburned tissue may be compromised. Full-thickness and extensive partial-thickness circumferential extremity burns are most likely to impede peripheral blood flow. Circumferential chest burns may restrict chest wall movement, impairing ventilation, and circumferential neck burns may result in tracheal obstruction. In such cases immediate escharotomy may be indicated.

On occasion, because of high-volume fluid resuscitation, noncircumferential and even unburned limbs may develop progressive tissue edema and ischemia requiring surgical decompression to prevent complications of nerve or muscle damage. Early elevation of the limb and active range-of-motion exercises every 15 minutes may minimize tissue edema, but once signs and symptoms of vascular impairment are present, the clinician must act quickly to prevent tissue hypoxia and cellular death. This pathophysiology may manifest itself within a time frame that mandates that the emergency physician must intervene. The reluctance of non-burn specialists to perform an adequate escharotomy is illustrated by the report of Brown and colleagues, [57] who found that 44% of pediatric burn cases were inadequately decompressed prior to arrival at a referral burn unit.

**Indications**

The indications for escharotomy are based on clinical examination, compartment pressure, or both. A high index of suspicion and a low threshold for intervention are essential for a successful outcome. Skin temperature and palpation of pulses are unreliable and imprecise indicators of adequacy of circulation because of peripheral vasoconstriction and local edema. The patient with circulatory embarrassment significant enough to warrant escharotomy may complain of deep aching pain, progressive loss of sensation, or paresthesias, but these parameters are difficult to quantitate in the severely burned, sedated, or mechanically ventilated patient. In the awake and cooperative patient, alteration in vibratory sensory testing with a 256-cps tuning fork may be a reliable indicator of increased compartment pressure. [58] However, gross motor activity and peripheral pulses may remain intact despite severe underlying muscle ischemia. In the series by Brown and colleagues, [57] peripheral pulses were present in 74% of the limbs that required decompression. Serial assessment of capillary refill, arterial flow (with the use of Doppler technique), arterial oxygen (with peripheral oximetry ), and compartmental pressures (using the technique described in Chapter 58) are sensitive indicators to monitor. Muscle compartments with pressures in excess of 30 mm Hg should be decompressed. Measurements should be taken before and after escharotomy to ensure adequate decompression.

In the patient with absent distal arterial flow (as determined by Doppler ultrasonic flowmeter) but an otherwise adequate blood pressure, immediate escharotomy is indicated. Bardakjian and colleagues suggest that an oxygen saturation below 95% in the distal extremity as demonstrated by pulse oximetry (in the absence of systemic hypoxia) also is a reliable indicator of the need for emergency escharotomy. [60]

**Technique of Escharotomy**

Because full-thickness burns are insensible to pain and involve coagulation of

superficial vessels, no general or local anesthesia is necessary, although sedation is highly recommended in the awake patient. A properly executed escharotomy releases the eschar to the depth of the SQ fat only. This results in minimal bleeding, which can be controlled by local pressure or electrocautery. These incisions, although limb- or life-saving, are potential sources of infection for the burn patient and should be treated as part of the burn wound. The wounds should be loosely packed with sterile gauze impregnated with an appropriate topical antimicrobial such as silver sulfadiazene cream. Fasciotomy, which involves a deeper incision, may be needed for thermal or electrical burns.

### Limbs

Under sterile conditions the lateral and medial aspects of the involved extremity are incised with a scalpel or electrocautery 1 cm proximal to the burned area, extending to 1 cm distal to the involved area of constricting burn (Fig. 41-11) (Figure Not Available) . The incision is carried through the full thickness of skin only and results in immediate separation of the constricting eschar to expose SQ fat. Because joints are areas of tight skin adherence and potential vascular impingement, incisions should cross these structures (Fig. 41-12) (Figure Not Available) . Care must be taken to avoid vital structures, such as the ulnar nerve at the elbow, the radial nerve at the wrist, the superficial peroneal nerve near the fibular head, and the posterior tibial artery at the ankle. The incision should extend to the great toe medially and the little toe laterally in circumferential burns of the feet, and to the thenar and hypothenar aspects of the hands (see Figs. 41-11 (Figure Not Available) and 41-12) (Figure Not Available) . Improvement in color, sensation, Doppler flow signal strength, and oximetry values indicate adequate release.

### Chest

Full-thickness circumferential chest or upper abdominal burns may impair respiration. For release of this eschar, the incision should extend from the clavicle to the costal margin in the anterior axillary line bilaterally and may be joined by transverse incisions, resulting in a chevron-shaped escharotomy.

### Neck

Neck escharotomy should be performed laterally and posteriorly to avoid the carotid and jugular vessels.

### Penis

Penile escharotomy is performed midlaterally to avoid the dorsal vein.

## Complications

Complications of escharotomy include bleeding, infection, and damage to underlying structures. Complications of inadequate decompression include muscle necrosis, nerve

injury (such as foot drop), and even amputation of the limb. Systemic complications of inadequate decompression include myoglobinuria and renal failure, hyperkalemia, and metabolic acidosis

## CONCLUSION

Patients with circumferential or nearly circumferential burns should be evaluated for deep tissue ischemia. Emergency physicians should not hesitate to perform an escharotomy prior to transfer of the patient to the burn center if there is evidence of reduced perfusion.

# Chapter 42 - Esophageal Foreign Bodies

**Steven A. Pace, Gary P. Young**

The presentation of a patient with an esophageal foreign body (FB) can range from the adult with dysphagia and a history of acute ingestion to the child with signs mimicking acute epiglottitis or chronic respiratory symptoms such as asthma. Four groups of patients are prone to have FBs lodge in the esophagus: patients with esophageal pathology; older patients with dental prostheses who ingest poorly chewed food (although this belief has been questioned [13] ); small children with oral retentive behavior; and adults with functional or organic impairment, including suicidal, psychotic, mentally retarded, incarcerated, and alcoholic patients. The type of esophageal FB usually depends on the patient's age and underlying condition--for example, coins in children, fish bones in adults, food boluses in the elderly, and almost anything in impaired adults.

The esophagus has three normal anatomic sites of narrowing where impactions are most common: the cricopharyngeus muscle, the crossing point of the aorta, and the lower esophageal sphincter (LES). In addition, pathologic strictures may develop elsewhere because of intrinsic lesions. Most impactions occur at the cricopharyngeus muscle, and patients are better able to localize the site of an esophageal impaction when the FB is above the cricopharyngeus. [14]

*Impacted esophageal FBs must be removed or dislodged.* Although the urgency of removal depends on many factors, FBs cannot be left in the esophagus for prolonged periods to dissolve or "pass by themselves." This approach courts disaster. After acute airway compromise, [15] esophageal perforation is the most serious complication of esophageal FBs. [16] Blunt objects lodged for any extended period of time can cause pressure necrosis and esophageal perforation. Small button batteries should be removed immediately; they are of special concern, because they can cause perforation in as little as 18 hours. [17] Although 80 to 90% of esophageal FBs cause transient symptoms and pass spontaneously, often within minutes, [18] approximately 10 to 20% require endoscopic removal, and approximately 1% require surgery. [19] In-hospital observation of small, round FBs or food boluses while awaiting spontaneous passage should be limited to no longer than 24 hours. [4] Conners [20] reported that 9 of 15 children with distal esophageal coins spontaneously passed the coins without intervention. Button batteries and sharp, angulated, or large FBs (2 cm in diameter or longer than 5 cm) are best removed immediately. This chapter focuses on the diagnosis and management of esophageal FBs.

## BACKGROUND

Both the diagnostic workup and the treatment of esophageal FBs are procedure oriented. In 1937, Jackson and Jackson published a classic monograph on the management of FBs of the upper airway and esophagus that was based on the use of the rigid endoscope in 3266 patients. [21] In 1945, Richardson reported the use of papain (a technique currently discouraged) in the successful treatment of meat obstructions of

the esophagus. [22] In 1966, Bigler reported the use of the Foley catheter to extract blunt radiopaque FBs from the upper gastrointestinal tract. [23] More recently, medications and solutions have been used to relax the LES. [18] Although otolaryngologists and thoracic surgeons continue to use the rigid esophagoscope for FB extraction, gastroenterologists began to use the flexible endoscope during the 1970s. [19]

To prevent morbidity and mortality from esophageal FBs, the emergency physician must first rapidly diagnose the presence and site of an FB, often with radiographic assistance. [24] Then the emergency physician must choose the best method of removal, often with the assistance of an otolaryngologist, a gastroenterologist, or both. [19] The anesthesiologist, thoracic surgeon, and operating room crew may also be called on to assist in the definitive management of these patients.

Cohen analyzed the reasons for the failure to diagnose esophageal FBs. [25] Among the explanations for retained FBs being missed are (1) the absence of a positive history, (2) the omission of the possibility of an FB from diagnostic consideration, and (3) the absence of positive radiologic findings. The last may be due to inadequate x-ray studies or to the characteristics of the FB itself. Although complications may occur with attempts to remove esophageal FBs, [16] long-term complications are more common with retained esophageal FBs. Children are more prone than adults to such complications.

## RADIOLOGY OF ESOPHAGEAL FOREIGN BODIES

### Background

The radiographic examination of the patient suspected of harboring an esophageal FB is usually straightforward and

accurate, assuming proper patient selection. Chaikhouni and colleagues [4] diagnosed esophageal FBs in 67 of 79 patients with standard chest radiographs only. Haglund and coworkers [24] reported a series of 264 patients with suspected esophageal FBs who were studied with plain film radiographs and contrast esophagograms. All patients received esophagoscopy, and only 1 of 195 patients with an FB detected by endoscopy had normal radiographic findings, although 43 patients (18%) had false-positive radiographic findings.

Some esophageal FBs are not so readily diagnosable by radiographs (e.g., radiolucent FBs). Food, wood, plastic, and aluminum cannot be seen on standard radiographs. Occult esophageal FBs can cause chronic respiratory symptoms. Children with previously unrecognized esophageal perforation caused by a pop-top from an aluminum can (which is radiolucent on plain radiographs) may present in this manner. [29] Unrecognized esophageal FBs may simulate asthma or epiglottitis, and symptoms may even improve when treatment is instituted for the wrong diagnosis. [30] Ros and Cetta has described the use of a metal detector to locate coins in children.

### Indications

Some investigators believe that the presence of ingested FB impaction is often overestimated by physicians, parents, and patients. Boothroyd and coworkers [33] found that if radiographic workups were limited to children with a clinical history suggestive of esophageal FB impaction or FB aspiration following a button battery ingestion, half of their pediatric patients would have been spared the expense of and exposure to x-rays. Whether patients (especially children) with a history of FB ingestion should routinely receive radiographs is a controversial issue. [ Whereas older patients may be able to localize the hypopharyngeal or esophageal FB by pain, pressure, or FB sensation, preschool children often cannot. Hence, *any preschool child, with or without symptoms, with a history of FB ingestion should be radiographed.*

It is axiomatic that a complete radiographic study include the areas from the "nose to the anus." In a small child this is often accomplished with a single radiograph taken with a large cassette. An ingested FB may end up in the nasopharynx of an infant and be missed if only a chest or abdominal film is obtained. Plain films of the neck are indicated in the patient with FB symptoms localized to the hypopharynx or upper chest, unless the hypopharyngeal FB can be visualized directly by the physician or there is a reliable history that a radiolucent FB (e.g., food without bones) was ingested. Plain films of the chest are indicated in the symptomatic patient who has ingested a radiopaque FB. Abdominal radiographs may also help locate a radiopaque FB, especially if the neck or chest films are negative and the physician or patient and family would be reassured by finding the FB. Plain radiographs may be necessary even after "removal" of an FB in an unreliable adult or a child who may have ingested more than one FB. Two ingested coins, for example, may overlap and appear as a single FB if only an anteroposterior film is obtained. Contrast esophagograms may be indicated if an FB ingestion or impaction is suggested by the history or examination but the plain films are negative or equivocal (e.g., radiolucent FBs).

Radiographic studies may also identify complications from esophageal FBs. For example, plain films may identify retropharyngeal air or pneumomediastinum caused by esophageal perforation, and contrast studies can demonstrate the site of perforation or obstruction. Conversely, if the patient recently ingested a poorly chewed meat bolus and is currently complaining of FB symptoms in the lower chest or epigastrium, it may be possible to relieve the distal esophageal impaction with or without benefit of prior x-ray contrast studies. Subsequently, these patients will require either contrast esophagograms or esophagoscopy to rule out any underlying pathology. If the FB has moved into the stomach or is found to be in the stomach, clinical follow-up (e.g., checking the stools of an infant) should be adequate to verify movement of the FB through the alimentary tract. Follow-up plain x-ray films are indicated after a reasonable period of time (e.g., 4 to 7 days, or sooner when symptoms ensue) if passage cannot be documented. Infrequently, there may also be indications for other radiographic studies, such as computed tomography (CT) and xerograms (described later). The following discussion presents an approach to the use of radiographic studies in patients with presumed esophageal FBs.

**Procedures**

The diagnostic methods to be discussed include plain film radiographs of the neck and chest, contrast esophagograms, CT, and xeroradiography. Soft tissue-intensity radiographs or xeroradiographs enhance the visualization of weakly radiopaque FBs.

Lateral and anteroposterior views of the neck are among the most commonly ordered films to evaluate the presence of FBs of the neck, because the majority of FBs are located at the level of the cricopharyngeus constrictor muscle. [24] Soft tissue technique should be routinely requested, and the technician should include the nasopharynx to the seventh cervical vertebra. (Note that FBs of the nose and those ingested and propelled into the nasopharynx are not uncommon and may be missed if only the neck is examined.) If slight hyperextension is performed, the clavicles are projected away from the lower esophagus. Having the patient phonate "Eeeeee" during the exposure traps air in the pharynx to enhance soft tissue landmarks and prevents swallowing, an activity that produces motion and blurs the anatomy. The upper esophagus, vallecula, and retropharyngeal area deserve special attention when reading the film.

Plain radiographs are somewhat difficult to interpret in the elderly. The physician must be cognizant of the anatomy and aware that soft tissues (ligaments, glands) and cartilage calcify as aging occurs. It is common to misidentify calcified structures as a possible FB (Fig. 42-1 (Figure Not Available) *A- C*).

Posteroanterior and lateral views of the chest are necessary for examination of the remainder of the esophagus. Both projections should be made, because small objects may be visible in only 1 plane, or multiple objects may be recognized in different x-ray planes. [30] Positive radiographic findings include the presence of radiopaque FBs, air-fluid levels, soft tissue swelling, intraesophageal air, subcutaneous (SQ) emphysema, or free air in the mediastinum. A chest radiograph may reveal infiltrates consistent with aspiration pneumonitis resulting from total esophageal obstruction or a tracheoesophageal fistula.

Contrast esophagograms can be used to pursue the suspicion of an FB in the absence of positive findings on plain x-ray films (Fig. 42-2) (Figure Not Available) . The type of FB, its precise location, the degree of obstruction or perforation, and possible underlying anatomic abnormalities can all be delineated by contrast studies. Several controversial choices arise at this point. Should a cotton pledget soaked in contrast agent be used initially? Which contrast agent should be used? Are fluoroscopy and radiologic consultation necessary? What is the risk of aspiration of contrast material or other complications?

A cotton ball or cotton pledget soaked in contrast can be swallowed, followed by x-ray exposures of the neck and chest during and after the swallow. [36] Total obstruction will impede the cotton, partial obstruction may do the same, and on occasion small pieces of cotton will lodge on irregular or sharp surfaces, thus indicating the presence of an FB. Advantages of this maneuver are that fluoroscopy is not necessary, and very little contrast material is required, which minimizes interference with follow-up endoscopy should it be required. However, the diagnostic accuracy of this technique is poor, [24] and

if negative, this technique does not definitely rule out an FB with incomplete obstruction.

The contrast agent of choice is debated among radiologists. In general, it is suggested that if a perforation is suspected, a water-soluble material (diatrizoate [Gastrografin]) should be used because it is less reactive on serosal surfaces than barium. Small perforations, however, may be missed with a diatrizoate study. In all other situations, barium compounds should be chosen, because imaging is better and tracheal aspiration of barium is less injurious.

Fluoroscopy and radiologic consultation are not always necessary. Allen [1] described excellent results in a series of 22 patients using barium solutions and spot films. The technique involves asking the patient to swallow the contrast solution, and after 4 to 5 swallows (approximately 50 mL), frontal, lateral, and perhaps oblique exposures are made. Should total obstruction be suspected despite the absence of an air-fluid level, the patient should be told to ingest only a small amount of barium initially to minimize the risk of aspiration. Positive findings include either positive or negative imaging of the FB in the column of barium and obstruction. In difficult cases, fluoroscopy performed by a radiologist presumably increases diagnostic accuracy and perhaps precludes the necessity for further diagnostic evaluation of possible underlying anatomic or motility disorders. Computed tomography (CT) is most useful in diagnosing suspected esophageal perforation in which the previously described modalities have been normal or inconclusive. The high resolution of CT may detect small FBs not apparent with plain films, barium swallow, or esophagoscopy. CT evaluation with soft tissue and bone windows may replace the barium swallow because of its better detection of thin, small, minimally calcified FBs that are often obscured by overlying tissues in the usual x-ray studies. Xeroradiography also may have some limited utility in this setting. The technique can also identify low-density materials such as aluminum or wood in the cervical esophagus.

In summary, assuming proper patient selection, the radiologic diagnosis of esophageal FBs can often be carried out successfully with plain radiographs alone. Contrast esophageal studies can delineate radiolucent objects and define the degree of obstruction. CT and xeroradiography have limited application in this setting.

## ESOPHAGEAL PHARMACOLOGIC MANEUVERS

Unsatisfactory pharmacologic alternatives for removing or resolving esophageal FB impactions include the use of diazepam, meperidine, and atropine. [43] These agents, alone or in combination, have success rates below 10%, which is no better than observation alone. [4] The pharmacologic alternatives described later (glucagon, nitroglycerin, nifedipine, and gas-forming agents [Table 42-1]) are most effective for distal esophageal food impactions. Most patients with this problem have underlying pathologic abnormality; a benign fibrotic stricture or Schatzki-type ring is more common than esophagitis with spasm, motility disturbances, or carcinoma. [18] If therapy with pharmacologic agents does not relieve the distal esophageal obstruction, the patient remains a candidate for other recommended therapy for the removal of an esophageal FB. In general, nonfood items (especially angulated, abrasive, or sharp FBs) should not be treated with pharmacologic modalities but instead should be removed by

esophagoscopy.

## Glucagon

**Pharmacology**

Glucagon is the prototype for the spasmolytic agents.  Its use has been advocated for enhancing the passage of esophageal FBs since 1977, and there have been several isolated reports of dramatic relief.  Glucagon relaxes esophageal smooth muscle and decreases LES pressure. However, it has no effect on the upper third of the esophagus, where striated muscle is present and some voluntary control is operative. It only minimally affects the middle third of the esophagus, although the successful use of glucagon to dislodge food in the middle third of the esophagus has been reported. Peristalsis is not affected by glucagon.

**Indications and Contraindications**

Glucagon is most useful for meat impactions at the LES that are suspected because of the patient's complaint of pain or "something stuck" in the lower chest or epigastrium. The clinical diagnosis is usually straightforward. Nevertheless, some clinicians recommend that the FB be localized first with contrast radiographs to establish that the impaction is at the LES and to serve as the baseline study for comparison following glucagon administration. [45] However, with a classic history and physical examination, most investigators agree that an initial contrast study can be omitted. Glucagon is successful in approximately 30 to 69% of lower esophageal obstructions, although reported case series generally involve small numbers.  Glucagon is not effective in upper and middle esophageal obstructions, and it is not yet recommended for use in children. Glucagon also is usually not effective in patients with fixed fibrotic strictures or rings at

| **TABLE 42-1** -- Recommended Pharmacologic Therapies for Esophageal Foreign Bodies | | | |
|---|---|---|---|
| **Class and Agents** | **Site of Action** | **Dose and Route** | **Adverse Effects** |
| Spasmolytics | | | |

| | | | |
|---|---|---|---|
| Glucagon | LES | 1-2 mg IV * | Nausea, vomiting, hyperglycemia, hypersensitivity |
| Nitroglycerin | Body and LES | 0.4-0.8 mg SL | Hypotension, tachycardia or bradycardia |
| Nifedipine | LES | 10 mg SL | Hypotension, tachycardia |
| Gas-forming agents | | | |
| Tartaric acid and sodium bicarbonate | Distal and proximal | 15 mL tartaric acid (18-20 g/100 mL) § | Vomiting, increased intraesophageal pressure |
| | | 15 mL sodium bicarbonate (10 g/100 mL) § | |
| Carbonated beverage | Distal and proximal | 100 mL PO | Vomiting, increased intraesophageal pressure |
| I V, intravenously; LES, lower esophageal sphincter; PO, per os; SL, sublingually. | | | |

* May be repeated once or used in conjunction with nitroglycerin.
1-2 inches of paste applied under an occlusive dressing may be an alternative.
A capsule is punctured, chewed, held in the mouth for 3 minutes, then swallowed.
§ Alternatively, dissolve 2 to 3 g tartaric acid and 2 to 3 g sodium bicarbonate in 30 mL water.

the gastroesophageal (GE) junction. [18] Glucagon is contraindicated if the patient has an insulinoma, a pheochromocytoma, Zollinger-Ellison syndrome, a hypersensitivity to glucagon, or a sharp esophageal FB.

**Use of Glucagon**

Some reports recommend a small test dose to check for hypersensitivity to glucagon. The therapeutic dose is 1 to 2 mg administered intravenously over 1 to 2 minutes in the sitting patient. The patient is given water orally after the injection of glucagon to stimulate normal esophageal peristalsis to help push the food through the relaxed LES into the stomach. [18] Glucagon has rapid onset and short duration of action. Gastrointestinal smooth muscle relaxes within 45 seconds, and the duration of action is about 25 minutes. If there are no results within 10 to 20 minutes, a second administration of 2 mg may be tried. Some clinicians combine nitroglycerin with glucagon therapy.

**Complications**

Glucagon is associated with a few minor side effects. If administered too rapidly, glucagon causes nausea and vomiting, so the adult patient must be alert and mobile enough to avoid aspiration. Occasionally the vomiting will dislodge the impacted food bolus. Theoretically, there is a risk of rupture of the obstructed esophagus during induced emesis, so slow injection is preferred. [18] The administration of glucagon is also associated with dizziness. Mild elevation of blood glucose levels is also common but is not of clinical concern. No fatalities have been reported.

**Further Evaluation and Therapy**

If glucagon should fail to produce symptom relief or resolve radiograph findings, its use does not preclude another method from being used subsequently. If the patient experiences symptom relief, follow-up contrast radiographs or esophagoscopy at that time or later, depending on the clinical concern for perforation or severe obstruction, should be obtained to rule out coexistent esophageal pathology. Esophageal pathology was demonstrated in all but 1 such patient in a study of 19 patients. [44]

## Nitroglycerin and Nifedipine

**Pharmacology**

Both sublingual nitroglycerin and nifedipine have been used in a manner similar to that of glucagon to relieve LES tone to allow the passage of a distal esophageal FB. Although these 2 agents have been used less than glucagon for the treatment of esophageal FBs, both agents are useful for the relief of chest pain associated with esophageal smooth muscle spasm [42] and may be administered concurrently with glucagon. Manometric and radiographic studies after the administration of nitroglycerin reveal abolition of repetitive high-pressure wave contractions characteristic of esophageal spasm, whereas nifedipine significantly reduces LES pressure without changing contraction amplitudes in the body of the esophagus. Thus, nitroglycerin may relieve partial or complete obstruction of the middle or lower esophagus secondary either to intrinsic esophageal disease or to simple FB impaction. However, nifedipine is

most likely to succeed when the bolus is lodged at the GE junction.

**Indications and Contraindications**

As is true for the clinical indications for the use of glucagon, any patient presenting with an impacted esophageal FB, especially a food bolus, may be a candidate for nitroglycerin and/or nifedipine. Also, similar to the mode of action of glucagon, neither of these agents is expected to relax a fixed fibrotic stricture or ring at the GE junction. [18] Nevertheless, because both agents have a relatively benign side effect profile, if the patient has no contraindication to their use, they may be tried with or without previous documentation of the distal esophageal obstruction by contrast study. Contraindications to their use include a history of allergic reactions, a sharp esophageal FB, and hypotension.

**Use and Complications**

Doses of 1 to 2 (0.4 mg) sublingual nitroglycerin tablets or 1 to 2 inches of nitroglycerin paste, or 10 mg of nifedipine (the capsule is punctured, chewed, held in the mouth, then swallowed) have been reported. A case report of the administration of a 10-mg dose of sublingual nifedipine to an elderly man with a 3-day history of a distal esophageal

FB led to an almost immediate but transient increase in chest pain and pressure followed by the relief of all symptoms. [43] It should be remembered that some patients with esophageal FBs may present with some degree of dehydration due to the inability to swallow liquids or their own saliva. These patients may be prone to hypotension from the vasodilation associated with the use of either agent. Ideally, rehydration should precede therapy with these agents.

**Further Evaluation and Therapy**

If treatment is successful, follow-up barium swallow or esophagoscopy should be performed to rule out underlying disease, as mentioned following the use of glucagon. Unsuccessful symptom relief should also be followed up with further study of the distal esophagus. [44] The use of either nitroglycerin or nifedipine does not contraindicate the use of any other method for the treatment of an esophageal FB.

# Gas-Forming Agents

**Pharmacology**

The use of gas-forming agents for the treatment of distal esophageal food impactions, especially for meat boluses, was first described in 1983. [53] The combination of tartaric acid solution followed immediately by a solution of sodium bicarbonate or even carbonated beverages has been reported. In theory, the use of this acid-base mixture or of a carbonated beverage may produce sufficient carbon dioxide to distend the esophagus, relax the LES, and push impacted food through the GE junction into the stomach. Radiologists use similar gas-forming solutions for upper gastrointestinal

contrast studies.

**Indications and Contraindications**

Gas-forming agents are indicated for the relief of distal esophageal food impactions, with or without prior FB confirmation by a contrast radiograph study. Although gas-forming agents are more likely to succeed with distal esophageal food impactions, they have also been used successfully in obstructions in the proximal esophagus. One author recommends that this modality be used first when a food bolus impaction is suspected at the GE junction, [16] because a fixed lower esophageal stricture or ring is more common than LES spasm. Concurrent administration of spasmolytic agents may improve the effectiveness of the gas-forming agents, which presumably distend the esophagus and "push" the food into the stomach.

**Use and Complications**

In one study, [53] patients were administered 15 mL of a solution of tartaric acid (18.7 g/100 mL), followed by 15 mL of a sodium bicarbonate solution (10 g/100 mL). In this study, Rice and coworkers achieved success in all 8 patients. In another study, [54] 1.5 to 3.0 g of tartaric acid and 2 to 3 g of sodium bicarbonate were each dissolved in about 15 mL of water. In this study, Zimmers and coworkers observed symptomatic relief in 17 of 26 cases (65%). A study involving the use of carbonated beverages in 28 patients found that 8 patients responded to the ingestion of barium sulfate suspension alone, and 16 of the remaining 20 patients (80%) responded to the ingestion of about 100 mL of a carbonated beverage. [55] Coca-Cola has been used with some success. [56] Most patients with esophageal FB impactions have been noted to retch after receiving gas-forming agents, which theoretically puts patients with FB impactions at risk for esophageal trauma. The only reported complication with the use of gas-forming agents for this indication was a mucosal tear of the esophagus (requiring surgical exploration to rule out mediastinitis) in a 66-year-old patient with an 18-hour-old esophageal impaction. [54] Because of this complication and the reduced effectiveness in prolonged FB impactions, the investigators recommended that gas-forming agents not be given to patients with impactions of more than 6 hours' duration or to patients with chest pain that might be indicative of an esophageal injury. [54]

**Further Evaluation and Therapy**

As with the use of glucagon, nitroglycerin, or nifedipine, even if administration of the gas-forming agent is successful, as judged by relief of symptoms, further evaluation is necessary for determining the underlying esophageal abnormality that potentially led to the FB impaction.

## Papain

Papain is *not* a recommended treatment for an esophageal FB. It is a proteolytic enzyme that has been touted for dissolving meat impactions. [57] Papain is available commercially as Adolph's meat tenderizer. This therapy has never been submitted to

clinical trial. In vitro studies suggest that the commercial preparation may have no intrinsic proteolytic activity. [58] Although it is harmless when in brief contact with the normal esophagus, if it is left too long in the obstructed esophagus, papain may begin to dissolve the abnormal esophageal mucosa underlying an FB.  This is likely to occur when the esophageal wall is ischemic owing to FB impaction, when esophageal injury results from small bony spicules in the FB, or when an underlying lesion is responsible for the obstruction. The subsequent rupture and leakage of the proteolytic enzymes result in a self-perpetuating mediastinitis, which can be followed by great vessel rupture. [54] Patients with esophageal FBs are at increased risk for aspiration, and pulmonary aspiration of papain results in acute hemorrhagic pulmonary edema. [55] In general, papain is not currently recommended because of the 2% mortality rate associated with its use and the fact that perforation has occurred in 2 of 90 cases reported in the literature.

## FOLEY CATHETER MANIPULATION OF ESOPHAGEAL FOREIGN BODIES

Some esophageal FBs can be safely managed with balloon-tipped catheters (e.g., Foley or Fogarty). Blunt esophageal FBs that are not believed to have resulted in total obstruction, perforation, or other esophageal injury may be amenable to Foley catheter manipulation. The classic patient for this technique is a small child who is brought to the hospital shortly after swallowing a coin documented by radiograph. FB extraction or dislodgment into the stomach with this technique are both therapeutic. An 80% or better success rate can be expected.  One serious complication and no deaths have been reported in >1000 cases in the literature.

Foley catheter extraction costs less than endoscopy under general anesthesia. [63] Emergency physicians, radiologists, otolaryngologists, and surgeons have all described this technique. In McGuirt's survey of otolaryngologists, thoracic surgeons, pediatric radiologists, and pediatric surgeons, [69] 50% of the pediatric specialists stated that they use the technique. However, few of the otolaryngologists and thoracic surgeons used this technique, instead preferring endoscopy. [69] Fluoroscopic assistance may be preferable, but it is not essential. Whether the procedure is performed in the emergency department or the radiology department, equipment and personnel capable of emergency pediatric airway management must be present.

### Background

In 1966, Bigler [72] first described the use of a Foley catheter for esophageal FB extraction. In 1972, Henry and Chamberlain [74] reported that 14 of 16 children were successfully treated with this technique. Campbell and Foley [65] reported a series of 100 children in which 91 FBs were extracted and 7 were dislodged into the stomach. No complications were reported. Nixon [71] reported the successful use of this technique in 12 of 15 patients with radiolucent FBs. The literature abounds with other reports of successful Foley catheter extractions.  The largest series reported includes 415 fluoroscopic Foley catheter extractions in children with an overall 91% success rate. [75] The success rate was 96% if the duration of impaction was 3 days or less, compared

with 50% if the duration was longer.

**Indications and Contraindications**

It is important that the patient not be combative during the procedure. Some researchers believe that the presence of an uncooperative patient precludes the use of a Foley catheter. [43] Many investigators use various combinations of topical anesthesia and parenteral sedation. Others report restraining children but not sedating them.

*Recently ingested smooth, blunt objects that are radiographically opaque are the only FBs suitable for balloon catheter extraction.* Coins are physically amenable to Foley manipulation, and their frequent presentation has resulted in numerous successful reports. Food boluses have also been extracted successfully, as have button batteries. [76] Even patients with preexisting esophageal disease have been treated with Foley catheter manipulation. [71] Recently ingested FBs carry little likelihood of causing pressure necrosis, perforation, or other significant injury. Recommendations for the maximum duration of impaction before treatment range from 24 hours to 2 weeks with inert objects, although most reports cite 48 to 72 hours as the upper limit. However, potentially reactive FBs such as button batteries have caused second-degree mucosal burns in as few as 4 hours [79] and esophageal perforations in less than 24 hours. [17] Animal studies have shown mucosal burns occurring within 1 hour and transmural penetration occurring in as little as 4 hours with button batteries. [17]

Radiographically opaque objects are most easily located by plain film radiograph. Radiolucent objects can be manipulated, [71] but uncertainties about location mandate contrast esophagograms. Fluoroscopy can be very helpful, and some believe the procedure should not be performed without it. Conversely, many practitioners describe manipulation by traction and tactile stimulation alone. Given the potential for airway obstruction, it is imperative to perform the procedure with airway equipment and suction available.

Contraindications to catheter removal of esophageal FBs include total esophageal obstruction, as manifested by an air-fluid level on plain radiograph or by contrast esophagography. The presence of a total obstruction prevents passage of the catheter tip distal to the FB. Esophageal perforation, as recognized by the typical symptoms and signs, requires immediate surgical consultation and precludes any blind esophageal manipulation. *Sharp, irregularly shaped FBs should not be removed with this technique*, because esophageal perforation or laceration can result, and the balloon may burst during the procedure.

**Equipment**

The equipment necessary is basic and present in most emergency departments (Table 42-2) . Although never reported, airway obstruction during the procedure is the most feared potential complication. Thus, the proper equipment and personnel capable of managing airway obstruction must be present, including suction devices. Clamps and forceps (bayonet and Magill) of various sizes should be available to extract the FB from the pharynx. Foley catheters ranging in size from No. 8 Fr with 3-mL balloons to No. 26

Fr with 30-mL balloons have been used.  In settings in which both children and adults may be treated, sizes ranging from 10 Fr to 16 Fr with 5- to 10-mL balloons should suffice. Child restraint devices (e.g., papoose board), topical anesthetics, and parenteral sedation may be used.

**Procedure**

Every patient should be appropriately coached concerning the procedure and cooperation. Young children must be gently restrained. Conscious sedation and nasopharyngeal topical anesthesia may be used, although some argue that the use of topical anesthesia and parenteral sedation depresses physiologic airway reflexes and increases the risk of aspiration. [73] The patient is then placed in a *head-down* Trendelenburg, lateral decubitus, or supine position during preparations. Some operators prefer to place the child prone during the procedure to facilitate oral expulsion of the FB.

Assuming the procedure is being performed in a young child, a 12 Fr Foley catheter may be the correct choice. After checking for symmetric balloon inflation, the catheter is most frequently inserted through the nose for patient comfort

| **TABLE 42-2** -- Equipment for Foley Catheter Extraction of Esophageal Foreign Bodies |
| --- |
| Standard resuscitation equipment for advanced airway management of children and adults |
| Foley catheters (10 to 16 Fr) |
| Topical anesthetics * |
| Parenteral sedatives * |
| Child restraint device * |
| Fluoroscope * |

* Optional equipment.

(Fig. 42-3) (Figure Not Available) . Some reports [65] argue that oral insertion is preferred to allow removal of the inflated catheter and FB with 1 smooth, uninterrupted pass. When using fluoroscopy, the catheter is visually passed distal to the FB. If performed without fluoroscopy, the distance from nose or mouth to FB is estimated, and the catheter is inserted accordingly. On occasion, the operator feels the catheter tip pass the object. The balloon is *slowly* filled with 3 to 5 mL of saline or contrast agent (if fluoroscopy is used). *Balloon inflation should be stopped if the patient complains of increased pain*; the catheter should be repositioned before an attempt at reinflation. Fluid is preferable to air, as it is less compressible. Overdistention of the balloon is undesirable; *in children no more than 5 mL of fluid should be used.*

The catheter is then withdrawn with steady, gentle traction. Contact with the object can be sensed as the friction of withdrawal increases. Significant impedance to traction requires termination of the attempt. Should the catheter slide by the object without dislodging it, the balloon can be enlarged and another attempt made. Often when the object reaches the hypopharynx, the balloon and gravity act in concert to fully externalize the FB, especially if the patient can be instructed to spit it out. The operator can also grasp the object with the fingers, forceps, or a clamp.

A follow-up radiograph may be necessary to exclude the possibility of multiple objects. [68] If fluoroscopy is not used and no FB is retrieved, another radiograph should be obtained, because 10 to 20% of the time, the FB will pass distally into the stomach. [10] This is considered therapeutic, because once a blunt FB has traversed the esophagus, only rarely will the FB not eventually pass per rectum. Such an occurrence has only been noted with objects longer than 6 to 7 cm. Multiple attempts should not be required if catheter size, placement, and balloon inflation are correct. Failed attempts are best followed by a change in one of the aforementioned parameters or repeat x-ray studies to confirm the continued presence of the esophageal FB before esophagoscopy.

**Complications**

No deaths have been reported. One esophageal rupture has occurred. [75] Nosebleeds, nasopharyngeal displacement of the FB with impaction, and a case of laryngospasm have been described. [80] Failure to move the object at all can be estimated to occur in approximately 15 to 20% of cases. Endoscopists who object to this technique argue that (1) an underlying disease or multiple FBs may be missed, (2) blind

manipulation may perforate the esophagus, (3) radiation exposure is prolonged when fluoroscopy is used, and (4) airway obstruction can occur. In the large series (415 catheter extractions) reported by Schunk, [75] minor complications occurred in 2% of the episodes, and major complications (i.e., esophageal rupture requiring surgical repair, mucosal laceration followed by 24 hours of fever, and 2 cases of transient respiratory distress) were noted in 1%.

**Disposition**

If the patient is asymptomatic and the extraction was successful, or if the FB is in the stomach, the patient may be released. If the FB is in the stomach, clinical follow-up should be adequate to verify movement of gastric FBs through the alimentary tract. Follow-up plain x-ray films are indicated after a reasonable period of time (e.g., 4 to 7 days, or sooner if symptoms ensue) if passage is not documented. Instructions should include warnings about gastrointestinal obstruction, perforation, and hemorrhage. Parents of children who swallow coins can be instructed to watch for any coins ("change in the stools"). Adults with esophageal FBs that have been removed successfully must be referred for evaluation of possible esophageal pathology. Should an FB remain lodged in the esophagus, immediate referral to an endoscopist is necessary.

## NASOGASTRIC TUBES

Several case reports describe the use of nasogastric (NG) tubes to manage esophageal FBs. Before insertion, the NG tube must be modified by cutting off the end of the tube at the level of the last side hole to allow maximal negative (suction) pressure to be exerted at the tube-FB interface. The initial technique is to place the tip of the catheter just proximal to a totally obstructing object (e.g., a food bolus) to aspirate oropharyngeal secretions. Then maximal suction may be applied directly to the esophageal FB itself in an attempt to lift it out proximally. Other modifications of this technique include the use of larger bore NG tubes or magnets and insufflation of air above the FB. Although gentle pushing of the FB through the GE junction is a tempting procedure to consider, *the technique of blindly pushing an impacted FB distally may injure the esophagus and is strongly discouraged*. This technique may be appropriate if performed under direct visualization during endoscopy (see later discussion).

Henry and Chamberlain [74] described the case of a steel ball obstructing the pylorus that was lifted into the esophagus with an NG tube and magnet, after which it was removed with a Foley catheter. Jaffe [80] used a similar method to remove button batteries from the stomachs of 6 patients. The batteries could not be extracted beyond the cricopharyngeus, and Foley catheters were used for ultimate removal. Chaikhouni and colleagues [4] reported a patient who had a needle removed from the lower esophagus under fluoroscopy with an NG tube and a magnet. Kozarek [81] described the use of a 34 Fr gastric evacuation tube and a syringe to successfully aspirate a meat bolus. Zalev [82] used air insufflation just above a meat impaction to negotiate the esophageal FB through the GE junction in 1 patient. Binder [2] described 1 patient whose dysphagia resolved after NG intubation. In summary, NG tubes may have limited usefulness in treating esophageal FBs.

## ESOPHAGOSCOPY

Esophagoscopy is the definitive diagnostic and therapeutic procedure for impacted esophageal FBs. [19] With esophagoscopy the physician can document the presence and location of the FB along with any underlying lesion, then remove the object and

reevaluate the esophagus after FB removal to rule out perforation or underlying pathology. Although it is not a procedure performed by the emergency physician, its proper role must be understood by the emergency physician (Table 42-3) . Esophagoscopy may be necessary even if a radiologic contrast study does not reveal complete obstruction, because x-ray studies are not always conclusive. Esophagoscopy may be necessary to rule out predisposing pathology or resultant perforation, even if the symptoms presumed to be due to an esophageal FB resolve.

## Indications and Contraindications

Endoscopy is the preferred method for removal of sharp or pointed objects such as bones, open safety pins, and razors. [83] [84] However, sharp, pointed FBs may require surgery. Toothpicks and bones require surgery more often than nails,

| | Flexible Endoscopy | Rigid Endoscopy | Laryngoscopy |
|---|---|---|---|
| **TABLE 42-3** -- Comparison of Rigid and Flexible Esophagoscopy and Laryngoscopy for Esophageal Foreign Bodies (FBs) | | | |
| Indication | Upper GI tract FB | Esophageal FB | Upper airway FB |
| Consultant | Gastroenterologist | Otolaryngologist | Otolaryngologist |
| | | Thoracic surgeon | Anesthesiologist |
| | | | Emergency physician |

| | Advantages Diagnostic | Diagnostic | Diagnostic |
|---|---|---|---|
| | Usually effective | Most effective | Usually effective |
| | More available | | Most available |
| | Local anesthesia | | Local anesthesia |
| | | | Airway management |
| Disadvantages | Requires patient cooperation | Requires general anesthesia | Requires patient cooperation |
| | Airway unprotected | | |
| Perforation | <0.1% | 0.5% | ? |
| Mortality | ? | <0.05% * | ? |

* Mortality is primarily related to the risk of general anesthesia.

needles, pins, blades, and dental prostheses. [83] In the case of sharp objects prone to causing esophageal perforation, IV antibiotics should be administered before any removal procedure. It is important to regard button batteries impacted in the esophagus as a true emergency because of their potential to cause perforation secondary to liquefaction necrosis.  Once button batteries pass the stomach, surgery may be necessary if serial radiographs indicate that the batteries have not continued to move or if they become symptomatic. In terms of the most common esophageal FBs, coins can cause pressure necrosis and perforate with prolonged impaction. Meat impactions may pass overnight, so endoscopy need not be performed immediately in these patients,

although it would be necessary eventually to assess for esophageal pathology.

### Equipment

Currently the flexible endoscope is more commonly used than the rigid esophagoscope for several reasons. [19] There are more gastroenterologists and others trained in the use of the flexible endoscope than in the use of the rigid endoscope. The latter remains the tool of surgeons, especially otolaryngologists and thoracic surgeons. Flexible esophagoscopy is easier to use and more mobile and has magnified visibility. Flexible endoscopic procedures are usually performed without general anesthesia, even in most children. [85] Because of this fact, Webb argued that flexible endoscopy is more cost-effective compared with either rigid endoscopy or surgery. [19] Berggreen and colleagues [86] reported a success rate of 96% using flexible endoscopy and 100% using rigid endoscopy in 192 patients with esophageal foreign bodies. Overall, rigid esophagoscopy had a higher complication rate (10% vs 5%, $P > .05$), but this trend did not reach statistical significance.

Rigid endoscopy is still favored by some clinicians, because it is superior to flexible endoscopy in containing and removing esophageal FBs. [19] Despite the advantages discussed, some reports contend that flexible endoscopy too often fails to remove the FB. Instead, the FB may become impacted, making subsequent extraction with the rigid endoscope more difficult or impossible. The main danger of flexible endoscopy that is not associated with the use of rigid endoscopy is the accidental loss of the FB into the airway during extraction in the unintubated patient. The requirement for general anesthesia and airway protection is both the main advantage and the drawback to rigid endoscopy.

## SPECIAL SITUATIONS

### Fish Bones in the Throat

It is common to evaluate patients who complain of sharp pain in the throat on swallowing after they have eaten fish. This symptom is perceived by the patient as evidence of a fish bone lodged in the throat.

Because an impacted fish bone can produce serious sequelae [87] if not removed, efforts should be undertaken in the emergency department to diagnose and treat retained bones. It is important to note that a bone is identified in only 20 to 30% of such patients, and symptoms may be due to minor mucosal trauma (abrasion, laceration) simulating the retained FB. Ngan and colleagues [88] described the outcomes for 358 patients with a history of fish bone ingestion: 117 bones were found, 21 on oral examination alone.

A plain radiograph (soft tissue technique, lateral neck projection) may identify some bones in the throat. However, even though cod, haddock, and halibut bones tend to be radiopaque, this technique is generally unreliable in ruling out retained bones, because many commonly eaten fish (e.g., mackerel, trout, salmon) have a radiolucent skeleton. [89] Evans and associates [90] reported 100 cases of proven fish bone impaction in which

lateral neck radiographs yielded a sensitivity of only 25%, a specificity of 86%, and a positive predictive value of 73%. These results suggest that plain radiographs are not useful in the diagnosis of fish bone impaction. Other radiographic techniques, such as swallowing a contrast-laden cotton ball, are also unreliable in identifying small bones.

Although pharyngoscopy is the most accurate method to diagnose a retained fish bone, *simply looking in the mouth and pharynx with the use of a tongue blade may identify many impacted bones.* Knight and Lesser reported a series of 71 patients who complained of the sensation of a fish bone in the throat; they noted that in 56 cases (79%), no bone was ultimately identified after careful follow-up. [91] Of significance, 14 of the 15 retained fish bones (93%) were visible on the initial examination of the oropharynx or hypopharynx. Derowe and Ophir reported 22 negative endoscopy examinations in 98 patients with a suspected esophageal FB; 20 of the 22 negative examinations were performed for suspected bones (mostly fish). [92] Most commonly these bones were instead found lodged in the base of the tongue, tonsil, or posterior pharyngeal wall (Fig. 42-4) . All were easily removed under direct vision. Saliva strands may mimic or obscure a bone, so care should be taken when extracting possible retained fish bones.

Although any patient persistently complaining of a retained fish bone ultimately should be referred for endoscopy (usually within 48 hours), many patients with the annoying symptoms do not have an esophageal FB. If a fish bone is present, however, many may be diagnosed and removed on the initial visit if one merely looks in the mouth and throat with a good light source and a tongue blade or with indirect laryngoscopy or with fiberoptic pharyngoscopy. Local anesthetic sprays may aid in the evaluation and removal of a bone, or they may be used for temporary relief of dysphagia. If a fish bone is not identified and the patient has no SQ emphysema, soft tissue swelling, hemoptysis, or inability to swallow, then referral for outpatient follow-up within 24 to 48 hours is acceptable. Note that retained fish bones do not "dissolve" and rarely pass spontaneously once lodged in the mucosa.

**Button Battery Ingestion**

Small disc batteries, such as those used in cameras, watches, and hearing aids, present a distinct type of esophageal FB because of the potential for serious morbidity and mortality. [93] Litovitz and Schmitz [94] reported on 2382 patients with battery ingestion, 2 of whom suffered serious esophageal injury. Batteries range in size from 7 to 25 mm and are radiopaque. Batteries appear as round densities, similar to an impacted coin, but some batteries may demonstrate a "double contour" configuration. [93] It is important to distinguish between a coin and a button battery, because button batteries require immediate removal (Fig. 42-5) (Figure Not Available) . The devices consist of 2 metal plates joined by a plastic seal. Internally the batteries consist of an electrolyte solution (usually concentrated sodium or potassium hydroxide) and a heavy metal, such as mercuric oxide, silver oxide, zinc, or lithium.

If ingested, these batteries often lodge in the esophagus. The mechanisms of injury include electrolyte leakage, injury from an electrical current, heavy metal toxicity, and pressure necrosis. Of particular concern is the development of a corrosive esophagitis

(occasionally fatal) or esophageal stricture or perforation as a result of caustic injury and prolonged mucosal pressure. Although relatively harmless in the stomach and intestines, batteries lodged in the esophagus should be considered an emergency situation, because even new batteries demonstrate corrosion, leakage, and mucosal necrosis within a few hours of contact with the esophagus.

Esophageal impaction mandates immediate removal. The Foley catheter technique may be successful, but it is less ideal than endoscopy. Endoscopy allows for direct esophageal evaluation and a more controlled extraction. In addition, the "invasive" nature of batteries may lead to rapid edema, making the catheter technique technically more difficult. Glucagon or gas-forming agents have no role in the extraction of button batteries. It is particularly dangerous to delay treatment in hopes that the FB will pass spontaneously.

A survey of British endoscopists and pediatricians revealed variable approaches to the child with a history of button battery ingestion. Forty-eight percent of 312 respondents indicated that under some circumstances batteries should be removed from the GI tract; 78%, 72%, and 48% extracted them from the esophagus, stomach, and duodenum, respectively, within 24 hours of ingestion. [95] However, *when in the stomach*, batteries do not require immediate removal. They may be followed radiographically to demonstrate passage, with little risk of gastrointestinal injury or heavy metal poisoning, even if the battery opens.  Some advise cathartics or metoclopramide (Reglan) if the battery is in the stomach. Ipecac is best avoided, as it has been reported to move a battery from the stomach to a more dangerous position in the esophagus.

**Childhood Coin Ingestion**

Coins are among the most commonly ingested objects in preschool-age children. In most cases the ingestion is quickly realized by a caretaker, and in the majority of cases, the coins pass uneventfully. [98] Rarely, an esophageal coin can be clandestine for many weeks or months, producing a variety of vague respiratory or gastrointestinal symptoms. Byard and colleagues described a case of supposed sudden infant death syndrome that at autopsy was found to be due to an obstructing esophageal coin. [99] Most coins pass from the esophagus to the stomach with only transient symptoms. The child may be in pain for a few minutes as the coin migrates, but on arrival in the emergency department, the child is often

asymptomatic. Coins that remain in the esophagus are likely to, but do not always, produce continued symptoms (e.g., drooling, pain, dysphagia, or refusal to eat or drink). Rarely, esophageal coins can produce airway distress by external compression of the trachea, simulating an asthmatic attack. Coins below the diaphragm are generally asymptomatic. Coins in the trachea produce immediate and obvious respiratory distress.

The decision regarding whether to obtain a radiograph during evaluation of the child with a possible coin ingestion is controversial. Some authors recommend that *asymptomatic* children not be radiographed. [35] However, because up to 44% of children with esophageal coins may be asymptomatic, it seems prudent to perform a

film on all children with a suggestive history. [100] In most cases a single film that includes the pharynx, esophagus, and stomach will suffice to prove or exclude an ingested coin. Previous mention has been made of the possibility of multiple coins stacked on top of each other simulating a single coin on an anteroposterior radiograph.

Once a coin's presence has been documented, a decision concerning removal must be made (Fig. 42-6) (Figure Not Available) . Schweich suggests that an impacted coin may be safely observed for 24 hours without risk in the asymptomatic or minimally symptomatic child. [101] Others recommend quick removal if the child is symptomatic, if the coin is in the upper third of the esophagus, or if the child has a history of gastrointestinal abnormalities. A few hours of observation in the child with minimal symptoms and an upper esophageal coin seems reasonable, as does a repeat film just prior to attempts at removal. Although coins in the upper and middle third of the esophagus are less likely to pass spontaneously, coins in the distal esophagus often pass spontaneously within a few hours.

Schweich suggests that all asymptomatic children, regardless of coin location, can be followed and re-radiographed in 24 hours. Anxious children and those in pain will benefit from the prudent use of sedation and analgesia. Conners and colleagues suggest that healthy children with coins *at the gastroesophageal junction* for <24 hours be allowed to eat and drink, with reevaluation for coin passage a few hours later. [20] Medical interventions (nitroglycerin, nifedipine, glucagon) may be appropriate at this juncture. If asymptomatic, reliable patients may be released home to return for follow-up evaluation. If the child fails the test of eating and time, he or she is then a candidate for a removal procedure. With this approach, attempts at removal may have to be further delayed until normal gastric emptying occurs.

About half of ingested coins are in the stomach at the time of first investigation, and such patients can be safely released home to allow for almost certain spontaneous passage with normal diet. Spontaneous passage of a coin from the stomach to the anus usually requires 5 to 7 days; there is no need for routine cathartics. Parents should be advised to check the stool for the coin and return for repeat radiographs if the coin is not found in 1 to 2 weeks. Most coins are passed unknowingly by the patient. Any abdominal discomfort or distention warrants reevaluation in the emergency department. If a follow-up radiograph demonstrates a persistent coin in the intestines for more than 3 to 4 weeks, an obstructive lesion may be present, and surgical referral is warranted.

## CONCLUSION

Esophageal FBs should be approached with great clinical respect because of their potential to produce serious injury or death. Esophageal FBs can be difficult to diagnose (e.g., in children and neurologically impaired adults), difficult to locate (e.g., when radiolucent or asymptomatic), and difficult to remove (e.g., requiring the services of radiology and specialty consultation).

# Chapter 43 - Nasogastric and Feeding Tube Placement

**Leonard E. Samuels**

A wide variety of patients require nasogastric intubation, including those with obstructed, preoperative, or potentially hemorrhaging upper gastrointestinal systems. Although the initial placement of feeding tubes is rarely the province of emergency physicians, patients with feeding tube complications frequently present to the emergency department (ED). Therefore, the emergency physician must be familiar with the different types of enteral tubes, as well as with their care, cleansing, and means of replacement.

## PROPERTIES OF NASOGASTRIC AND FEEDING TUBES

Enteral tubes are made of various materials. Polypropylene is the most common material used for Levin and Salem sump nasogastric tubes (Fig. 43-1) , but it is too rigid for long-term use for a feeding tube. Although they are less likely to kink than others, polypropylene tubes are more capable of creating a false passage during placement. Latex (rubber) tubes are moderately firm, require greater lubrication for passage, are relatively thick-walled, and induce a greater foreign body reaction than tubes of other commonly used materials. Latex, especially in latex balloons, deteriorates more rapidly than other materials. [1] Silicone tubes are thin-walled, quite pliable, and nonreactive. However, the walls of silicone tubes are less strong and may rupture if fluid is introduced into a kinked tube. [2] Polyurethane tubes are quite nonreactive and relatively durable. Rigidity varies from manufacturer to manufacturer, depending on tube thickness. A stylet may aid in the passage of polyurethane and silicone tubes, but its use increases their rigidity and capability of tissue dissection, especially in tubes that have a small distal end bulb. [3] Some feeding tubes have weights, usually made of tungsten, which are nontoxic if released into the gastrointestinal tract.

## NASOGASTRIC TUBE PLACEMENT

### Indications and Contraindications

The simplest nasogastric tube is the Levin tube, which has a single lumen and multiple distal "eyes." The advantage of the Levin tube is its relatively large internal diameter in proportion to its external diameter. The theoretical disadvantage is that a Levin tube should not be left hooked up to suction after the initial contents of the stomach have been drained, because the suction will cause the stomach to invaginate into the eyes of the tube, blocking future tube function and potentially causing injury to the stomach lining. Levin tubes are useful for diagnostic aspiration of stomach contents or for simple instillation of therapeutic agents. Tubes with lumens larger than 16 Fr are preferred for lavage procedures if large particulates are anticipated (see Chapter 45) .

In practice, the Levin tube is no more prone to bleeding complications than the Salem sump tube, provided that the *intermittent* suction setting for the Levin tube is 40 mm Hg.4 The Salem sump tube is preferred to the Levin tube for more chronic use as a

drainage device because of the presence of a separate channel (blue colored) that vents the distal main lumen to the atmosphere (Fig. 43-2) (Figure Not Available) . When extended gastric drainage is desired, Salem sump tubes should be placed on intermittent suction, even though the vent generally will prevent excessive vacuum at the tube tip. Note that potentially both intermittent suction and wall unit vacuum can exceed the venting capacity of the second lumen; for intermittent suction, the vacuum setting should be 120 mm Hg.4

The major indication for nasogastric intubation is to aspirate the stomach contents (see also Chapter 45) . Aspiration may be useful in the management of upper gastrointestinal bleeding, particularly to differentiate upper from lower gastrointestinal bleeding. Except when frankly bloody fluid is obtained, the sensitivity and specificity of aspiration to detect upper intestinal bleeding are not good. Use of Hemoccult or guaiac cards to detect bleeding in gastric aspirates is unreliable, because false-positive tests are obtained frequently. [5] Although variceal rupture has occurred during insertion of instruments into the esophagus, several studies suggest that nasogastric tube passage is generally safe, even in the presence of esophageal varices. [8] [9] Nasogastric suction is indicated in cases where vomiting is likely to be recurrent or dangerous, such as with a paralytic ileus or intestinal obstruction, or to relieve acute gastric dilation. The trauma patient may need a nasogastric tube as part of the evaluation for gastrointestinal injury or to decompress the stomach prior to surgery or peritoneal lavage. A radiopaque nasogastric tube may help delineate transdiaphragmatic hernia of the stomach after trauma.

Nasogastric tubes are contraindicated in a few groups of patients with special predispositions to injury from tube placement. Patients with facial fractures who have a cribriform plate injury may suffer intracranial penetration with a blindly placed nasal tube. [10] Patients who have esophageal strictures or a history of alkali ingestion, especially recent alkali ingestion, may suffer esophageal perforation. Comatose patients may vomit during or after nasogastric tube placement; their airways should be protected prior to placing the nasogastric tube. Gagging will decrease venous return and increase cervical and intracranial venous pressure, which may be clinically significant in selected trauma patients with cervical or intracranial bleeding and in the presence of significantly elevated intracranial pressure.

Despite their traditional use, *nasogastric tubes are not routinely required in patients with mild to moderate pancreatitis*. Extended irrigation of the stomach with water in a patient with upper gastrointestinal hemorrhage can lower serum potassium levels, [13] and animal studies suggest that cold water lavage can cause rather than control bleeding. [14] [15] No study has shown irrigation to be effective in the control of bleeding, and vigorous lavage with cold water may lower the body temperature. However, a nasogastric tube may be used to instill air into the stomach for documentation of a suspected gastric perforation by enhancing visualization of free air under the diaphragm on an upright chest film.

**Equipment**

Nasogastric passage of standard nasogastric tubes or feeding tubes can be messy and

may be accompanied by coughing, retching, sneezing, bleeding, and spilled water or stomach fluid. Both patient and physician should be gowned; cleanup may be reduced if the bib area is covered with a towel and a supply of tissue or washcloths is available. The physician requires gloves; the appropriate nasogastric or feeding tube; a penlight or other light source; a tongue blade; an emesis basin; a glass of water with drinking straw (or an extra syringe if the patient is uncooperative); water-soluble lubricant; a stethoscope; a safety pin; a rubber band; topical anesthetic jelly, spray, or ointment; and vasoconstrictor nasal spray or liquid. For standard nasogastric tube placement, a piston or bulb syringe (with a catheter slip-tip) should be available. Nasogastric feeding tubes should have a compatible 50- or 60-mL syringe (some are Luer compatible and others are slip-tip compatible).

Tape torn in 4-in. strips or a commercial nasogastric tube holder (e.g., Suction Tube Attachment Device, Hollister, Libertyville, Ill) should be handy for securing the tube after placement. Cotton-tipped applicators and tincture of benzoin may be helpful if the skin is greasy. Depending on the indication, other equipment may be needed (e.g., saline for irrigation or Magill forceps in an uncooperative or anesthetized

patient). Make sure the feeding tube is designed for duodenal passage if that is desired--such tubes are usually longer than regular feeding tubes.

An antireflux valve (Keith Antireflux Valve, Sherwood Medical Industries, St Louis) is a one-way valve used on the vent port of the Salem sump tubes. One study found the device to be cost-effective by reducing the need for gown and linen changes during inpatient nasogastric tube use. [17] Placement of this device in the ED should be considered if extended use is anticipated.

**Procedure**

Explain the procedure to the patient. If the patient is alert, the head of the bed should be raised so that the patient is in the high Fowler position or upright. A towel is placed over the patient's chest to protect the gown, and an emesis basin should be available on the patient's lap. [18] The tube (typically a 16 or 18 Fr sump) should be positioned so that insertion distance can be estimated, and this distance should be marked with tape or noted using the markers printed on the proximal tube. A simple method is to extend the tube from the xiphoid to the earlobe and then from there to the tip of the nose and *then add 15 cm* (6 in.) (Fig. 43-3) . [19] While other techniques of estimating appropriate nasogastric tube insertion distance using formulas may be more accurate than the nose-ear-xiphoid method, the formulas are difficult to memorize and calculate. [20] [21] It is a common error to fail to estimate the proper length of the tube prior to passage.

The nares should be checked for obstruction and the tube passed down the more patent naris. Patency can be assessed by direct visualization or by having the patient sniff while first one and then the other nostril is occluded. The selected naris and the distal 5 cm of the tube should then be lubricated with a liberal amount of water-soluble lubricant, viscous lidocaine, or lidocaine jelly. Lubrication of the naris can be facilitated using a syringe (without needle) filled with 5 to 10 mL of lubricant (Fig. 43-4) . Alternatively, the nostrils can be lubricated and anesthetized with a pair of cotton swabs soaked in the

desired anesthetic/lubricant. A 10% lidocaine spray also can provide excellent (and rapid) anesthesia of the nose. Additional topical pharyngeal anesthesia using benzocaine (Cetacaine) or lidocaine spray in the pharynx prior to the procedure will significantly reduce gagging and aid tube passage. The mucosa should be anesthetized *prior to tube passage*. It is insufficient to put anesthetic jelly on the tube and expect anesthesia during the tube's passage.

The tube is inserted into the naris along the floor of the nose and not toward the nasal bridge (Fig. 43-5) (i.e., directed posteriorly and not cephalad). Mild resistance may occur in the posterior nasopharynx, but only gentle pressure should be required to overcome this resistance. Bleeding or dissection into retropharyngeal tissue may occur if force is used, and it is better to try the other nostril or an oral tube if significant resistance is encountered. Once the tube passes into the oropharynx, a pause may help the patient regain composure and enhance the chances for cooperation with the rest of the procedure.  Have the patient, if he or she is alert and cooperative, sip water from a straw and swallow while the tube is advanced into and down the esophagus. This often helps ease passage of the tube. Flexing the neck also tends to direct the tube into the esophagus rather than the trachea. Excessive choking or gagging or any coughing, change in voice, or the appearance of condensation on the inner aspect of the tube should alert the clinician to the possibility of tracheal tube position, prompting withdrawal of the tube into the oropharynx. The tube may be inspected through the mouth to detect coiling or respiratory passage. A tube position lateral to the midline suggests correct position in the esophagus. [22] Once the tube is in the esophagus, it should be *advanced rapidly* to the previously determined depth. Slow passage of the tube prolongs discomfort and may precipitate more gagging. [18]

**Confirmation of Tube Placement**

Before the nasogastric tube is secured, nonradiographic confirmation of its successful placement should be obtained. All confirmation methods have some possibility of error, so it is wise, when in doubt, to use more than 1 method.

Insufflation of air into the nasogastric tube is simple and quick. The requirement of increased pressure to instill air or the absence of borborygmi with instillation should raise concern about tube malposition or kinking. If the patient immediately burps on air instillation, an esophageal tube position should be suspected. [23]

Unfortunately, passage of nasal tubes into the lungs is frequent, especially in comatose or demented patients, and insufflation is often insufficient to detect this type of malpositioning. The insufflation test also is unreliable in detecting whether the tube has advanced past the stomach into the small bowel. [26]

Aspiration of stomach contents, especially if pH tested, is more reliable. If the pH is less than 4, the nasoenteral tube has an approximately 95% chance of having its tip in the stomach. Furthermore, nonrespiratory placement is almost guaranteed. [31]

Aspirated fluid can occasionally be obtained from the lung or pleural space, but the pH should be 6.0 or higher. Approximately 2% of patients have an alkaline stomach pH. [33]

Causes include duodenal reflux, antacids, H2 blockers, or recent instillation of formula or medications.

Ask the patient, if he or she is awake and cooperative, to talk. If the patient can't speak, respiratory placement is likely. Note that with small-bore tubes, patients may still be able to speak despite tracheal placement. [34]

Once correct tube position is tentatively confirmed, the tube should be secured. If the tube is radiopaque, radiographic confirmation of tube position can then be obtained. If the patient requires abdominal or chest radiographs for other diagnostic purposes, it is best to place the nasogastric tube prior to obtaining the films.

**Securing the Tube**

The nasogastric tube is generally secured to the patient with tape attached to both tube and nose. A butterfly bandage (or tape on each side of the nose) that then coils around the nasogastric tube is a typical approach. The tube should be clean and possibly prepped with tincture of benzoin, as should the nose. If a tape should let go or require repositioning, both the tape and the tincture of benzoin must be replaced. It is wise to also secure the tube to the patient's gown, so that a tug on the tube will encounter this resistance before pulling on the material securing the tube to the patient's nose. A rubber band tied around the tube with a slip knot (Fig. 43-6 *A* and *B*) and pinned to the gown near the patient's shoulder is effective. It is critical to ensure that the tube is not secured in such a way that it presses on the medial or lateral nostril. Necrosis or bleeding can result if a tube is not secured correctly.

When a Salem sump is used, the blue pigtail must be kept above the level of the fluid in the patient's stomach, or stomach contents may leak back through the vent lumen. If a patient needs to ambulate with a sump tube in place, the blue pigtail can be fitted into the plastic connector at the end of the suction lumen, creating a closed loop that should not leak. For a Levin tube, a syringe can be attached to the lumen and taped to the patient's gown. Alternatively, a commercial antireflux valve may be attached.

**Placement Issues**

Nasogastric tube placement in the unconscious patient can be attempted using the same technique as in the conscious patient, omitting those steps requiring patient cooperation. If the patient is intubated, the balloon of the endotracheal tube should be deflated briefly to allow passage of the nasogastric tube. The nasogastric tube is easily misplaced into the pulmonary tree in the unconscious patient, a complication that may be missed during the procedure, because gag and cough reflexes may be suppressed and the patient cannot talk. In addition, the absence of swallowing may prevent successful passage of the tube. Several techniques may be used to successfully pass a nasogastric tube in a difficult unconscious patient.

The nasogastric tube may be placed initially through a naris into the oropharynx. The tip of the tube is then visualized with a laryngoscope, grasped with Magill forceps, and

pulled out of the mouth. An endotracheal tube with an internal diameter that is slightly larger than the external diameter of the nasogastric tube is selected and is slit along its lesser curvature from its proximal end to a point 3 cm from its distal end. The slit endotracheal tube (generally 8 mm ID [internal diameter]) is then passed through the mouth into the esophagus. [35] Alternatively, a 7-mm ID slit endotracheal tube may be passed directly through the nose into the esophagus. [36] Passage into the esophagus is facilitated by the stiffness of the larger endotracheal tube and does not require active swallowing. The tip of the nasogastric tube is then threaded into the endotracheal tube and advanced into the stomach (Fig. 43-7) (Figure Not Available) . The slit endotracheal tube is then removed from the esophagus. When the distal part of the endotracheal tube is visible, the unslit 3-cm distal part is slit with scissors. The endotracheal tube is removed, and the nasogastric tube remains in place. [37] Any slack tubing is then advanced with the forceps or pulled back nasally, depending on the final depth required for the nasogastric tube. The technique can also be performed by passing the slit endotracheal tube nasally, which also saves the trouble of orally advancing or nasally retracting any slack tubing.

In particularly passive or toothless patients, guiding the nasogastric tube with the fingers is occasionally successful. [38] Displacing the larynx forward by manually gripping and lifting the thyroid cartilage has aided tube insertion, [39] as has simple jaw elevation. A soft nasopharyngeal airway, well lubricated, is at times easier to pass nasally than the nasogastric tube, and then the lubricated nasogastric tube can be passed through it. In addition, it affords some protection to the nasal mucosa if multiple attempts to pass the nasogastric tube are necessary, or if it is particularly important to minimize bleeding or trauma. Cooling a nasogastric tube increases its rigidity, and coiling it can increase the tube curvature, both of which may help pass the tube. Note that a cooled tube is more likely to dissect into tissue.

Ultimately, if all other methods fail, a flexible fiberoptic bronchoscope or esophagoscope can be placed under direct vision into and through the esophagus. [40] A guide wire is threaded into the stomach. The nasogastric tube can be placed over the guide wire into the stomach; the guide wire is then removed.

### Complications

Complications of standard nasogastric tube placement are similar to problems noted with nasogastric feeding tube placement. The complications related to tube misplacement are discussed in that section. In addition, the physician placing the nasogastric tube in the patient with neck injuries should be cautious of potentiating cervical spine injuries with excessive motion during passage (especially in association with coughing and gagging in the awake patient). Furthermore, passage of a nasogastric tube in the awake patient with a penetrating neck wound may exacerbate hemorrhage, should coughing or gagging result. Particularly serious forms of tube misplacement are pulmonary placement (Fig. 43-8) (Figure Not Available) and intracranial placement (Fig. 43-9) (Figure Not Available) .

## NASOENTERIC FEEDING TUBES

**Indications and Contraindications**

The most common indication for feeding tube replacement in the ED is unintentional removal of a preexisting feeding tube. In 1 prospective study, 38% of tubes were removed unintentionally. Although some of these tubes had fallen out or been coughed out, more than half had been pulled out by the patient. [42] Tube rupture, deterioration, or clogging may also necessitate replacement. Management of the clogged or nonirrigating feeding tube is discussed in the section Clogged Feeding Tubes.

Enteric feeding tube replacement is contraindicated in the presence of vomiting, intestinal obstruction, severe ileus, upper gastrointestinal bleeding, distal enteric anastomoses, and conditions in which bowel rest is desired. [43] Occasionally, the patient whose feeding tube is displaced does not require nasoenteric feeding tube replacement because oral intake is adequate for nutritional needs. Patients suffering erosions or discomfort may be candidates for percutaneous endoscopic gastrostomy (PEG) placement.

**Choice of Feeding Tube Site**

Three major classes of enteral feeding tubes are currently in common use; classification is according to site of insertion. Tubes can enter through the nares, a cervical ostomy, or an abdominal ostomy. The mode of nutritional support is determined by the patient's physiologic requirements and degree of debilitation, the disease process, the anticipated duration of inadequate oral intake, and the facilities and equipment available to initiate and maintain delivery of nutritional support.

Tube feeding may be either a temporary or a permanent means of nutritional support and may have supplemental or complete responsibility for meeting the patient's nutritional needs. Enteral nutrition is less expensive and easier to use than total parenteral nutrition [45] and probably safer.  Enteral nutrition is indicated when oral intake is less than two thirds of the patient's requirement, despite a functioning gastrointestinal system. Nasoenteric feedings are appropriate when fewer than 4 weeks of feeding are required, [43] although in some cases a pharyngostomy may be preferred because it is less irritating and cosmetically easier to conceal. Cervical or abdominal ostomies are recommended when more than 4 weeks of feeding are necessary. [43] Oral ingestion may continue with gastrostomy tubes and may be possible with nasogastric and cervical ostomy tubes with a gauge of 12 or lower.

Enteral tubes may terminate in the stomach or the small intestine. Gastric feeding results in better digestion than intestinal feeding. Normally about 20% of gastric antral contents pass into the duodenum, with 80% refluxing back into the body of the stomach for further mixing. [20] If the feeding tube is placed in the antrum of the stomach or in the small bowel, enteral feeding solution passing into the small bowel may not be tolerated, resulting in diarrhea and paradoxical decreased nutrition.

The most common rationale for small intestinal feeding is to reduce regurgitation and

aspiration. Disagreement exists concerning the clinical significance and frequency of regurgitation-induced aspiration.  Nevertheless, the emergency physician should attempt to ensure that the terminal end of a replaced tube is in the same viscus as the original.

External inspection may or may not reveal where a feeding tube should terminate. A de Pezzer (mushroom) or Foley gastrostomy tube is designed only for intragastric termination. Some tubes have 2 lumina, 1 terminating in the stomach for decompression and the other in the small bowel for feeding. These can be confused with tubes that have 2 entrances to 1 lumen (1 for continuous feeding, the other for medications) and tubes that have a second lumen leading to an inflatable balloon. Review of the medical record or communication with the surgeon is often necessary when external inspection does not reveal the specific type of feeding tube or indicate where a feeding tube should terminate.

**Procedure**

Nasoenteric feeding tube replacement requires greater time and effort if the patient is uncooperative or has a physically obstructing lesion. Nasoenteric feeding tube migration into the duodenal bulb generally requires patient positioning in the right decubitus position for about an hour after successful intragastric passage. [50]

The physician should explain the procedure to the patient before tube passage. The patient's assistance with esophageal passage can be enlisted in 2 ways. First, the patient can assist with swallowing the tube. Second, the patient can vocalize when requested to confirm that the tube is not passing down the trachea. Many patients find it helpful to develop a signal to indicate to the physician when they need a temporary reprieve and rest. It is generally advisable to restrain the hands of demented, impaired, or otherwise uncooperative patients.

The nares should be prepared before passage with generous application of a lubricant and local anesthetic. A local vasoconstrictor will dilate the nasal passages slightly and help prevent bleeding. Viscous lidocaine or water-soluble jellies (e.g., Surgilube) are excellent lubricants. A small amount of lidocaine with epinephrine can be squirted from a syringe into the nares a few minutes before lubrication. Application of lidocaine with epinephrine, application of the lubricant, or both, can also be accomplished with soaked cotton-tipped applicators. Alternatively, viscous lidocaine can be introduced into the nose with a gloved finger. The physician should be prepared and garbed for the inevitable sneeze that this will provoke. Patients with a hyperactive gag reflex should gargle with viscous lidocaine or with benzocaine (Cetacaine) or lidocaine (Xylocaine) spray before the placement attempt.

The feeding tube stylet should be lubricated and inserted into the feeding tube before the insertion of the feeding tube into the nares. Tube stylets can be lubricated with water-soluble jelly. Dobbhoff, EntriFlex (Biosearch), and several other tubes have a preapplied lubricant that must be activated with a 5-mL flush of water. Frying pan lubricant sprays (e.g., Pam) also may work well on the stylet. Other types of lubricants may have greater potential for pulmonary complications or may damage the substance

of the tubes. *The stylet should never protrude beyond the end of the feeding tube*, because these stiff, small-diameter wires have the capacity to scratch the esophagus and can encourage the creation of a false passage. The stylet may lock into position on the tube at the proximal end and should be properly secured.

An upright patient position is more comfortable for the physician during nasogastric passage. The distal end of the feeding tube should be moistened or lubricated and then passed down the more patent of the 2 nasal passages. Most persons' nostrils are fairly symmetric, but in some cases, growths or old trauma may narrow the passage. The tube

should be inserted into the nostril and then directed toward the ear. A common mistake of the inexperienced practitioner is to continue directing the feeding tube in a cranial direction. If the first nostril is impassable, the opposite side may permit tube passage. In particularly difficult cases, it may be desirable to first pass a lubricated nasal airway (trumpet) and then pass the feeding tube through it, reducing nasal and nasopharynx stimulation.

The tube's passage into the nasopharynx can usually be detected by a lessening of resistance. One can then proceed with esophageal passage. Topical anesthesia of the pharynx may be required to ameliorate any excessive gagging that may occur at this point. Cough should warn of incipient respiratory placement of the tube. If the patient can vocalize, the tube has not yet passed through the vocal cords. The patient should bend the head forward if possible. This encourages the tube to pass into the esophagus and aids airway closure. If the patient is gagging or choking, it may be because the tube is beginning to coil in the oropharynx. Look in the mouth with a penlight to see if this is occurring. If the tube is coiled on entering the airway, temporarily pulling the tube back should relieve the problem. When pulling back, some resistance is again felt as the thicker end bulb of the tube begins to enter the posterior nasal passages; at that point, the tube has been pulled back far enough for a fresh try at esophageal intubation. The cooperative patient should swallow ice chips or take small sips of water with a straw to facilitate tube passage down the esophagus. In some cases it may be impossible to avoid coiling when the tube is advanced, unless the patient is swallowing. When the patient is uncooperative, the introduction of 5 to 15 mL of water into the mouth or into the proximal end of the feeding tube with a syringe may induce swallowing, facilitating tube passage. Although the patient may not swallow for several minutes, waiting for the swallow may mean the difference between a coiled or pulmonary tube placement and successful passage.

Another technique for passing soft feeding tubes involves attaching the distal end to a Salem sump tube by means of a gelatin cap (one half of a size 0 gelatin capsule, as used for a large medication capsule). Lubricate the joined tubes, avoiding lubrication of the soluble capsule. The Salem sump-soft tube complex is passed nasogastrically. Once the tubes are positioned in the stomach, both tubes are irrigated several times with 10 to 20 mL of water. After about 5 minutes the gelatin capsule should have dissolved, allowing independent removal of the Salem sump tube (Fig. 43-10) . [2]

**Placement Confirmation**

Auscultatory confirmation of tube placement can be misleading.  The proper technique for auscultation is to inject 20 mL of air into the tube rapidly while listening with a stethoscope in the left upper abdomen. Air insufflation should occur without resistance and without delay in borborygmi. If the sound is muffled, faint, or delayed, the physician may reinject another 20 mL of air and listen over the lower lung. If the sound is clearer, the tube may be in the lung. The injected air should be aspirated from the stomach after placement is confirmed so that the patient is less likely to burp or regurgitate.

Proper placement of nasogastric feeding tubes should be confirmed with a radiograph. Tubes should be secured with tape before taking the radiograph. Tincture of benzoin applied to the tube and the patient makes the tape stick better. Commercial tube fixation devices may also be used. The position before and after the radiograph is taken is more likely to be the same if the tube is secured before filming.

Gordon suggests that radiographs are not necessary to check nasogastric feeding tube placement if the following criteria are met in order. [2] The tube must be passed beyond the 50-cm mark in a normal-sized adult; palpation and visual inspection confirm that the tube is not coiled in the mouth or oropharynx; air insufflation occurs without resistance and without delay in epigastric borborygmi; 10 mL of water advances through the tube without difficulty; and some water can be retrieved with aspiration. Do *not* inject water into the tube if any of the preceding criteria suggest that placement may be in the lung or upper esophagus. A risk of delivering water into the airway exists. [43]

Aspirated pleural or pulmonary fluid contents can be mistaken for return of intragastric fluids. [3] In addition, radiographic confirmation of tube placement may be misleading. In viewing the radiograph, it is particularly important to study the area around the carina. An esophageal tube shows at most a mild change in course, whereas a tracheally placed tube usually deviates significantly as it travels into the right or left main stem bronchus. The end of a nasogastric tube may appear to be in the stomach yet be in the left lung behind and below the top of the diaphragm. [51] When a stylet has been used for passage, the stylet should be left in the feeding tube for the radiograph, because the tube's course is not always visible without it. The stylets of most tubes are designed to allow insufflation and aspiration while in place. Even when stomach entry is certain, the intestinal location may be misleading on radiograph: A nasoenteric tube may lie completely to the left of midline and yet have its tip in the duodenum, or it may have a position overlying the right abdomen yet not have entered the duodenum. A contrast study is necessary to ascertain duodenal position when pulmonary placement has been ruled out.

The radiograph should also be examined for the presence of mediastinal air and a pneumothorax, which may suggest pulmonary or esophageal puncture. An esophageal puncture should be evaluated with endoscopy and may require surgery, depending on the size of the rent.

The end bulb of most nasoduodenal tubes will pass into the duodenum after patient

positioning in the right decubitus position for an hour. Some researchers recommend pretreatment with metoclopramide to enhance gastric emptying. One investigator found that metoclopramide enhances duodenal passage of nasogastrically placed feeding tubes in diabetic, but not in nondiabetic, patients. [50] Gastric antral motility in diabetics is often impaired; metoclopramide helps restore normal synchronized activity in these patients but has little effect on emptying in subjects who have normal antral function. The usual dose of metoclopramide is 10 mg administered IV. Also, 3 mg/kg of erythromycin lactobionate given IV over 1 hour works similarly and may be effective even if metoclopramide fails. [55] Endoscopy or fluoroscopy may be necessary if positioning and metoclopramide are not successful. [56]

## Complications

Pulmonary intubation is an uncommon but well-known and potentially fatal complication of nasal feeding tube insertion (see Fig. 43-8) (Figure Not Available) . Coughing and respiratory distress are the most common symptoms of respiratory passage of a nasogastric tube, but there may be relatively few symptoms in a demented or comatose patient. [57] Decreased mentation and an absent cough reflex are predisposing factors for unrecognized nasopulmonary intubation with nasogastric tubes. [3] A small end bulb, e.g., 2.7 mm diameter, can slip past a tracheal high-volume, low-pressure cuff and pass easily to the lung periphery.

To prevent nasopulmonary feeding tube intubation, one may choose to use the wire stylet only for initial tube passage. This will prevent pulmonary parenchymal penetration but not tracheobronchial intubation. It also will make passage of tubes positioned in the esophagus more difficult. [3] Esophageal entry of the feeding tube can be checked with the use of a laryngoscope. The metal stylet can be left in the tube during esophageal tube passage when an esophageal position has been confirmed by laryngoscopy. [59] A stylet should never be reinserted into a tube already in the patient. The stylet may puncture the tube at a kink or exit the side holes and puncture the esophagus. [60]

A pneumothorax may result when a nasogastric tube dissects into or is withdrawn from the pulmonary parenchyma. [30] Bloody aspirate from a tube should heighten awareness of tissue damage.

A clogged or nonfunctional nasogastric tube may prove difficult to remove. Fluoroscopy may allow careful insertion of a guide wire or stylet into an in situ tube to facilitate removal. Fluoroscopy also may identify the mechanical problem interfering with the removal. Bent-double segments are probably the most common; knots are uncommon but do occur. Excessive force should not be used in the removal of a nasogastric tube, because serious injury to the patient may result.

Premature removal of the nasogastric tube is the most frequent complication of feeding tube use. To help prevent removal by the uncooperative patient, the nasogastric tube may be secured to a loop anchor passed in the same naris. The anchor works by aversive stimulation of the soft palate and nose with distraction of the nasogastric tube, rather than by mechanical stabilization of the tube.

Sax and Bower recommend a technique for creating a separate nasogastric tube anchor. [61] A soft weighted nasoenteric tube is cut approximately 12 in. from the top. A heavy (2-0) silk suture is passed through the tube to exit the side hole. The guide wire is inserted with care, as it must not protrude from the cut end. The patient should be sedated if uncooperative. The tube is inserted through the anesthetized naris into the nasopharynx, grasped with Magill forceps, and pulled to exit from the mouth (Fig. 43-11 A). The excess tube is trimmed without cutting the silk suture. A closed loop is made by tying the silk suture in front of the nose. The loop must be slack enough that it does not apply continuous pressure to the nose or palate at rest. The nasal feeding tube is passed through the same nostril and secured to the loop (see Fig. 43-11 B). This anchor is simpler to construct and more comfortable than anchors passed through the opposite nostril. [61]

Complications of properly placed nasoenteric tubes include nasopharyngeal erosions, esophageal reflux, tracheoesophageal fistulas, gagging, rupture of esophageal varices, and otitis media. [43] One survey of nasogastrically fed patients found that the most distressing features of nasogastric feeding tube use were deprivation of tasting, drinking, and chewing of food; soreness of the nose; rhinitis; esophagitis; mouth breathing; and the sight of other patients who were eating. [62]

Checking feeding tolerance is difficult with small-gauge feeding tubes. Aspiration of tubes to check for residual is not recommended with tubes of 9 Fr or smaller. Aspiration is likely to clog the tubes, because they collapse under pressure and because relatively small particles can occlude the tube. For the same reasons the residual is likely to be inaccurate. [46]

**Patient Instructions**

Patient instructions should include a proscription against aspiration in small tubes. To maintain catheter patency, small tubes should be flushed with 20 to 30 mL of tap water at least 2 to 3 times daily and after administration of medication.  Water is a more effective irrigant than cranberry juice. [63] Medications should be in liquid form or be completely dissolved, or they may clog the tube. Methods of dealing with a clogged tube are discussed below.

The tube should be anchored to the nose and face in such a way that it is not in contact with the skin at the nasal opening. This reduces tube discomfort and prevents necrosis of the alae, nares, and distal septum. Patients who exhibit a tendency to pull on their tubes need adequate restraints. Patients receiving tube feedings should have their heads elevated to at least 30 degrees above the horizontal.

# PHARYNGOSTOMY AND ESOPHAGOSTOMY FEEDING TUBES

### Indications

Cervical pharyngostomy and cervical esophagostomy both have been developed

relatively recently. Cervical esophagostomy was first described by Klopp in 1951, [65] and cervical pharyngostomy was described in 1967 by Shumrick. [66] Cervical esophagostomies are generally performed at the time of cervical or maxillofacial operations. Malignant growths of the proximal esophagus, head, or neck are the primary indications for esophagostomy. Cervical esophagostomies may eventually evolve a permanent sinus, allowing the feeding tube to be removed between meals.

As with an esophagostomy, a pharyngostomy may be either a simple ostomy or a permanent tract formed by suturing pharyngeal mucosa to the skin. [66] [67] [68] The more common version is a simple opening that closes very rapidly (within a few hours) if it is not stented with a tube (Fig. 43-12) . Pharyngostomy is a simple procedure and does not require general anesthesia. It is useful when nasogastric feeding tubes are contraindicated or when prolonged tube feeding is anticipated. [44] Patients with traumatic or congenital anomalies of the maxillofacial region who are undergoing an operation and patients with impaired swallowing from neuromuscular disorders are potential candidates for cervical pharyngostomy. [68] A pharyngostomy is also indicated to bypass obstructing lesions for feeding, to assist healing after head or neck surgery for malignancy, and to feed the unconscious patient. A pharyngostomy can also be used for gastric decompression if it is required for more than 3 days. [67] Cervical pharyngostomy tubes produce only mild discomfort. Patients do not usually complain about pharyngostomy tubes, as they do about a nasogastric tube. The additional comfort and ease of productive coughing are especially important in critically ill and elderly patients.

**Technique of Tube Passage**

The feeding tubes commonly used for esophagostomy and pharyngostomy feeding are the same as those used for nasogastric feeding. Polyurethane feeding tubes are the most frequently used. The large (>3 g) bolus weights on some feeding tubes may be inconvenient to pass through the ostomy. Tubes that are approximately 90 cm (3 feet) long are appropriate for gastric feeding; tubes that are longer--108 to 112 cm (43 or 45 in.)--are used for duodenal feeding.

The feeding tube replacement technique is the same for pharyngostomy and esophagostomy. The outside of the tube tip can be lubricated with a small amount of water-soluble lubricant jelly. Mineral oil, which irritates the airways if aspirated, should never be used. The tip of the tube is inserted into the ostomy and directed caudally to ensure that it enters the esophagus and does not pass upward into the nasopharynx or mouth (Fig. 43-13) . The patient may be able to assist by attempting to swallow. The length of feeding tube required varies depending on the position of the ostomy and is several centimeters longer than the distance from ostomy to xiphoid. For duodenal feeding, the tube should be advanced about 20 cm beyond the distance from ostomy to xyphoid.  If the feeding tube persistently exits the mouth during attempts at passage instead of passing down the esophagus, the following 2 techniques may prove useful. After insertion of the feeding tube a short distance into the ostomy, a flashlight is used to visualize the tube in the pharynx. The feeding tube is grasped slightly proximal to the end bulb using Magill forceps, and the end bulb is directed toward the esophagus in the posterior inferior pharynx. Once the tube is properly directed, it may be possible to advance the remainder of the tube through the external ostomy. Sometimes it is

necessary to use the forceps to advance the entire length of the feeding tube. An alternative method is to allow the feeding tube placed through the ostomy to exit the mouth for the entire distance that must be passed down the esophagus. The end bulb of the tube is then directed into the posterior pharynx, and the patient is directed to swallow as for an orogastric tube. Toward the end of tube passage, it may be necessary to use a Magill forceps or to pull back the tube slightly at the ostomy to eliminate a short loop of extra tubing in the oropharynx.

Tube replacement is more difficult in the first week after the creation of a pharyngostomy or an esophagostomy. A tract forms after the first week and helps prevent tissue dissection by the tube. The angle of a well-formed tract also encourages appropriate esophageal passage. A well-formed tract closes more slowly than a new ostomy, although in some people even a long-term ostomy may begin sealing within a few hours. If an ostomy is too narrow for the replacement tube, the ostomy should be stented with a narrower tube and the patient's surgeon contacted.

**Complications**

Complications of pharyngostomy and esophagostomy include local soft tissue irritation, accidental extubation because of excess length of the external tube, pulmonary aspiration from vomiting, arterial erosion with exsanguination, and esophagitis or stricture of the esophagus from reflux. Accidental pulmonary intubation is less common with cervical ostomy tubes than with nasogastric tubes, at least partially because patients with cervical ostomies are more likely to be alert and have functioning cough reflexes. Auscultation and aspiration are still advisable techniques to check tube placement. Radiographic evaluation may also be necessary and is essential to confirm duodenal feeding.

## GASTROSTOMY, GASTROENTEROSTOMY, DUODENOSTOMY, AND JEJUNOSTOMY TUBES

The mid-19th century physician Sedillot described the first functioning gastrostomy, which formed as a complication of a war wound. The gastrostomies performed by Sedillot on 2 patients resulted in peritonitis and death. The jejunostomy procedure was first performed by Surmay in 1879. It was not until the 1890s that further innovations in surgical technique allowed the gastrostomy to be popularized. The Witzel serosal-lined gastric tunnel technique (Fig. 43-14) (Figure Not Available) and the Stamm procedure of concentric pursestring sutures placed around the gastrostomy tube were developed in the 1890s. These 2 techniques prevent significant intraperitoneal gastric fluid leakage, a complication that had frequently resulted in the deaths of gastrostomy patients. Both Witzel and Stamm gastrostomies tend to close rapidly without a stenting gastrostomy tube. In the early 1900s the tubular gastrostomy (Depage-Janeway) was developed. The Depage-Janeway gastrostomy results in the creation of a permanent mucocutaneous ostomy. Since the turn of the century, more than 30 different operative techniques have been described for tube gastrostomy. [44]

**Operative Indications and Contraindications**

Neurologic diseases constitute the most frequent indication for a gastrostomy tube. [47] Facial fractures, oropharyngeal trauma, and tracheal and laryngeal injuries may be indications for placement of a temporary feeding gastrostomy. Rare indications for gastrostomy include enhancement of nutrition by continuous feeding in severely debilitated patients who still are capable of oral intake, provision of a route for bile replacement in patients with an external biliary fistula, and the need for long-term gastric decompression. Indications for gastrostomy tube placement in children include neurologic diseases, facial reconstructive surgery for congenital deformities, and maxillofacial trauma. Young children who require long-term administration of unpalatable medications or dietary components may also require a gastrostomy. Tube duodenostomies are created almost exclusively for duodenal decompression after partial gastrectomy with Billroth II anastomoses. [68] Permanent jejunostomies are rarely used. Tube jejunostomy is indicated when the proximal bowel has a fistula or is obstructed, when recovery of small bowel motility is anticipated long before recovery of gastric motility, and after a gastrectomy.

Contraindications for gastrostomy feeding include severe gastroesophageal reflux, upper gastrointestinal fistulas, repeated aspiration of gastric contents, and intestinal or gastric outlet obstruction. [44] Jejunal feeding is contraindicated if the highly osmolar feeding solutions required for jejunal feeding are poorly tolerated and cause copious diarrhea.

**Indications and Contraindications for Tube Replacement**

The nursing home patient with a nonfunctioning or displaced feeding tube represents a common ED presentation. When available, details regarding the type of tube, details of placement (when, where, and how), and its most recent use can help guide the physician. If the tube has been removed, often the information on the tube can guide acquisition of an appropriate replacement tube. When unclear, a Foley catheter (16 Fr or larger) is generally a reasonable temporary replacement. Information should be sought from the patient's medical record, nursing home record, and private physician, when available.

If the tube is nonfunctioning yet still in place, the physician must make a judgment as to the risk versus benefit of removal and replacement versus an attempt at unclogging the tube (see subsequent section on unclogging). *The major concern is that a new tube may be misplaced (i.e., into the peritoneal cavity)*. If it appears that a skin incision was used to place the tube, it is unlikely that the patient has an easily removable tube. If the patient has signs of a complication (e.g., infection, ileus, or intestinal obstruction), surgical consultation is warranted.

Ease and safety of transabdominal feeding tube replacement depend on the surgical procedure performed and the length of time since placement of the feeding tube. For a simple gastrostomy, the insertion site of the tube through the gastrointestinal wall is sealed by either annular or plication sutures. The gastrointestinal wall is approximated

to the peritoneum around the site of penetration to provide a further leakage barrier. The tube is then secured outside the abdominal wall. A Witzel tunnel is a serosal tunnel created when the feeding tube is placed alongside the viscus for a distance after exiting the viscus, and the bowel is pulled up over it along this distance and secured with sutures (see Fig. 43-14) (Figure Not Available) . In 1 type of Hickman catheter jejunostomy, the tube passes through a Dacron cuff and a Witzel tunnel. [71] Reinsertion of a Hickman catheter through a tortuous, rough Witzel tunnel is unlikely to be simple. [72] A percutaneous gastrostomy may have been placed without any attempt to affix the stomach to the abdominal wall.

Nonoperative tube replacement techniques are safe only through an established tract between the skin and the bowel. Catheter replacement should not be attempted in the immediate postoperative period. A simple gastrostomy takes about a week to form a tract. [67] A Witzel tunnel may take up to 3 weeks after the operation to mature sufficiently for safe nonoperative tube replacement. A nonfunctional tube can still serve as the stent for the gastrostomy tract and should not be removed if it cannot be promptly and safely replaced.

## Equipment

Gastrostomy tubes come in an unusually varied selection of styles and materials. Rubber, silicone, and polyurethane tubes are all in common use. Many gastrostomy tubes are designed with a flange or a crossbar (bumper) to anchor them in the stomach (Fig. 43-15) .

Equipment for feeding tube insertion includes gloves, stethoscope, feeding tube, external bolster, lubricant, basin, and a syringe that fits the tube. Tincture of benzoin, tape, and absorbent dressing material may be used to dress the wound, although many are better left undressed. [73] Some feeding tubes require special plugs or connectors. Others need to be pinched with a clamp when not in use to prevent leakage. Some tubes are placed with the aid of accompanying guide wires or stents. For still others, it is necessary to use a clamp or hemostat, endotracheal tube stylet, urinary or uterine sound, laryngeal dilator (No. 14), guide wire, or other appropriate rod or support as an aid to tube passage (Fig. 43-16 A and B).

A stylet to assist introduction of de Pezzer catheters can be fashioned by cutting half the stylet from a 9 Fr pediatric chest tube inserter. The tip can be filed smooth. The device will be 10 to 12 cm long and can be inserted alongside the de Pezzer catheter and into its tip to distend and flatten the mushroom (Fig. 43-17) .

## Transabdominal Feeding Tube Removal

A feeding tube may have to be removed because it is irreversibly clogged, leaking, or broken; persistently developing kinks; too large or too small; causing a hypersensitivity reaction; associated with an abscess; or not the appropriate length for feeding into the desired viscus. Before a new transabdominal feeding tube is inserted, the old tube must be removed. Most, but not all, tubes can be removed without endoscopy. It is imperative to know whether the tube in place is safe to remove before attempting to remove it.

Standard de Pezzer or mushroom catheters that have been modified with bolsters or rings at the time of endoscopic or surgical insertion may no longer be safe to remove with traction. Tubes are occasionally secured with sutures or rigid internal bumpers or stays. Recently placed feeding tubes may need to be left in until a tract has formed (1 to 2 weeks depending on the procedure) even if the tube is nonfunctional. *The externally visible tube does not always reveal the internal stabilization* (see Fig. 43-15) .

A simple Foley catheter gastrostomy is easiest to remove. Once the Foley balloon is deflated, the tube should slide right out. If the Foley balloon cannot be deflated, cutting the tube may allow the balloon to deflate. The catheter must not be cut so close to the abdomen that it will be impossible to maintain a grip on it for a traction removal if the balloon still does not deflate. The balloon also may be punctured to cause it to deflate. To puncture a Foley balloon, traction is applied to the catheter to draw the balloon up against the ostomy. Using the taut feeding tube as a guide, an 18- or 19-ga needle is passed along the tube to puncture the balloon. It may be necessary to try again on the other side of the catheter, because the balloon may be asymmetrically inflated, and contact with the needle may be established on 1 side and not the other. The clinician should be careful not to track away from the ostomy into the patient's abdominal wall or to cause separate punctures of the stomach. The balloon is allowed a minute to deflate before another attempt is made at traction removal. Large balloons should probably be punctured, whereas small balloons may be removed with traction.

Traction is an acceptable removal technique for feeding tubes that are secured by a small mushroom. A towel is placed over the orifice, and the physician applies counterpressure with the flat part of the hand against the abdominal wall as the tube is placed under tension (Fig. 43-18) . This causes the tube and end mushroom to narrow, and the tube should come out easily. The inner crossbar, if present, may remain in the stomach when the rest of the feeding tube complex is removed by traction. Obstruction from the crossbar, which will pass in the stool, has yet to be reported for adults. In small children, obstruction is a possibility, and the crossbar should be removed by endoscopy.

A local anesthetic may be useful in selected cases of feeding tube removal, especially when the tube is in some way secured subcutaneously--for example, by a Dacron cuff. [71] It may be difficult to remove a catheter accidentally caught by a fascial suture during operative closure. [75]

Removal of gastrostomy tubes with moderate to large mushrooms may be easier if the mushroom is distended with a sound or stylet. The length of the gastrostomy tube should be known so that the sound may be inserted to the correct depth. Firm resistance should be noted at that point. Firm resistance at deeper depths represents pressure on the viscus wall and can result in viscus puncture. [53] The premeasured stylets that come with feeding devices are useful instruments for assisting in removal. This is particularly true of gastrostomy "buttons," whose ends resemble de Pezzer catheters. Because buttons come in a variety of lengths, it is important to have the proper stylet. Following elective permanent removal of a gastrostomy tube, a pressure dressing should aid in closure of the fistula. [53]

If it is not possible to pull the inner bolster or mushroom out through the ostomy, it may

be acceptable to cut the tube at the skin, push the remaining short stump into the stomach, and rely on later rectal passage. Although obstruction or impaction is infrequent, it can occur, and this alternative should not be chosen with children or patients who have had previous impaction, potential for bowel obstruction, or stool-passing problems. Rigid or large internal mushrooms and bolsters, the very kind that cause the most difficulty with percutaneous removal, also are more likely to cause difficulty with rectal passage. In no case should a device be released into the gut with a long length of tubing attached. Remember that double-part tubes may have an additional length of tubing for duodenal or jejunal feeding that extends far past the inner bolster. Korula and Harma reported the successful intestinal elimination of 63 of 64 gastrostomy tubes that were cut at the skin entrance and advanced into the stomach through the stoma. [76] These cases included tubes with internal bumpers, and success occurred regardless of the nature of the patient's underlying medical disorder, age, or method of original tube placement. However, no patient had suspected obstruction or potential for obstruction (e.g., no prior radiotherapy, inflammatory bowel disease). The 1 lodged tube required endoscopic removal from the pylorus. In most cases tube passage was documented by sequential radiographs, with a mean interval to passage of 24 days (range, 4 to 181 days).

Some physicians and surgeons may strongly condemn cutting off the tube at the skin, even when the risks posed by the procedure are very low. It is always advisable to contact the patient's private physician before cutting the tube. In some cases endoscopic retrieval of the tube remnant will be preferred to allowing rectal passage, and the tube should not be cut until just before or during endoscopy to ensure that migration does not occur before endoscopy.

**Transabdominal Feeding Tube Replacement**

A Foley catheter is a simple gastrostomy tube to replace. After the tract opening and distal Foley catheter are lubricated, the Foley balloon's integrity is checked by inflation. The catheter is then inserted into the tract. Good placement can be recognized by easy passage, prompt borborygmi with 20 mL of air insufflation, and rapid return of stomach juices with aspiration. The balloon is then inflated with saline (30-mL balloons are best), and gentle traction is applied to draw the balloon against the stomach wall. *Always inflate the Foley balloon with saline*, because balloons inflated with air deflate more easily. Tube replacement is usually successful if the tube has not been dislodged for more than 4 to 6 hours. If passage is impossible, a radiologist may be consulted to advance the tube over a guide wire that has been passed through the tract using fluoroscopy.

An external bolster may be threaded onto the catheter. The external bolster is a ring or bar of material threaded onto a tube that creates a large bulge on the tube and prevents inappropriate ingress of the tube into the ostomy on the side of the bolster. The anchor must adhere strongly to the tube so that mild stress on the tube does not cause the bolster to migrate up the tube. The bolster can be salvaged from the old tube or constructed in a number of ways. An anchor may be made from the end nipple of a de Pezzer catheter. The ring from a 24 Fr catheter, taken off at its junction with the end nipple, fits snugly over a 22 Fr Foley catheter when the balloon is distended slightly. The

nipple can be pushed forward to an anchoring position near the stoma. The nipple can be fixed in this position by fully distending the Foley balloon and applying a circle of adhesive tape just adjacent to the nipple on the stem on the side away from the body. [77] Adhesive tape sticks better if the lubricant is removed and the stem is prepared with tincture of benzoin.

An external anchor may be made from a segment of tube from a large rubber catheter (Fig. 43-19) . A segment approximately 3 cm in length is cut to form the bolster. Two diamond-shaped openings can be formed on both sides of the segment by bending the segment and clipping it with scissors on either side of the bend. The diameter of the holes should be slightly smaller than the catheter. A hemostat or a Kelly clamp can be inserted through both holes to grasp the external end of the gastrostomy tube, which can be bent in half--with some difficulty--to narrow its diameter (Fig. 43-20) . The hemostat can then pull the tube through the bolster, which can be threaded down the tube and anchored with tape as described previously (Fig. 43-21) . The outer crossbar should be located 1 cm away from the skin. [78] Contact between the crossbar and the skin promotes moisture entrapment and maceration. Too much tension on the gastrostomy tube can result in necrosis of the gastric wall where it abuts the inner mushroom or balloon. Proper placement of the external bolster helps avoid this complication (Fig. 43-22) .

Many physicians prefer mushroom or de Pezzer gastrostomy tubes, which are more difficult to replace than Foley catheters. The advantage of these catheters over Foley catheters is that the mushroom nipple keeps its shape more reliably than the Foley balloon, which tends to deflate. [62] Foley catheters also have a greater tendency to migrate internally and block the pylorus. [44] A Kelly clamp or other stylet can be placed through a side hole into the tip of a gastrostomy mushroom and used to elongate the end for easy passage through the gastrostomy (see Figs. 43-16 and 43-17) . Lubrication of the mushroom may make it more difficult to maintain the stylet's position in the mushroom. Some stylets are suitable for passage down the catheter lumen to elongate the end. Tubes should never be forced through a stoma for replacement, because this can cause separation of the viscus from the external stoma and lead to viscus leak or tube misplacement. [53]

The replacement tube provided in the ED does not have to be--and in a few cases should not be--the same type placed at surgery. The tube must be compatible with the feeding system, terminate in the same viscus, and fit through the ostomy. When a Witzel tunnel jejunostomy is created, the catheter most frequently used is a Broviac catheter. An appropriate replacement is a lubricated polyurethane tube shortened to a total tube length of 30 to 40 cm. Although the polyurethane tube is strong enough to be used for tube replacement through the Witzel tunnel without a guide wire, Broviac (silicone) catheters are too pliant to be coaxed through the resistive tunnel. [70] Jejunal feeding tubes are generally advanced 20 to 30 cm into the jejunum.

Jejunal feeding tubes may be placed through or alongside a decompressing gastrostomy. Original placement of the jejunal feeding tube is endoscopic. Replacement of these tubes also generally requires endoscopic assistance.  Fluoroscopic techniques can be used to help guide these tubes; however, these techniques are out of the realm

of emergency practice. [43] Occasionally, feeding tubes are placed in the jejunum because of gastric ileus.  If gastric ileus is no longer present, a gastrostomy tube may suffice. The rationale for jejunal feeding, risk of aspiration, and acceptability of gastric feeding to the primary physician should be established before changing from a jejunal feeding tube to a feeding gastrostomy tube. Techniques discussed in the nasoenteric feeding section of this chapter (metoclopramide and right decubitus position) may in selected cases coax gastrostomy-placed feeding tubes into the small bowel. Gastric decompression tubes are either clamped or put at continuous drainage. [80]

Verification of tube placement can be made radiographically using a fluoroscope or with a small amount of contrast material passed into the tube (Figs. 43-23 and 43-24) . In the latter scenario, a catheter-tip syringe is used to introduce water-soluble contrast solution (e.g., diatrizoate meglumine-diatrizoate sodium [Gastrografin]) into the tube. *Barium is contraindicated.* Generally 20 to 30 mL of water-soluble solution is adequate for documenting the intraluminal tube position. A supine abdominal film should be taken within 1 to 2 minutes of dye instillation to optimize gut visualization. *If the contrast material does not flow freely into the tube, the procedure should be terminated immediately and the position of the tube questioned.* With proper positioning, contrast will outline the gut containing the tube (e.g., stomach with gastrostomy tube). An irregular or rounded blotch with wispy edges or streamers suggests peritoneal leakage. In questionable cases, dye injection can be performed under fluoroscopy.

### Transabdominal Low-Profile Feeding Tube Replacement

Low-profile feeding tubes are almost flush with the skin, held in place with a preformed plastic external bolster. Commonly called *feeding buttons*, they are only slightly more difficult to place than a Foley catheter. The key to replacing a low-profile gastrostomy is to select the correct-length tube, which can be done by measuring the length using the stoma measuring device included with the tube. The stoma measuring device is inserted through the stoma and has a retaining hook or balloon on the end and markers on the portion protruding out of the abdomen. By counting the number of markers, the physician determines whether a long, medium, or short tube is appropriate. Once the appropriate tube is selected, the enclosed pre-sized obturator will distend the tube's end bulb, which can then be passed through the well-lubricated stoma for insertion of the tube.

### Complications

If the ostomy is not a mucocutaneous type, it will close rapidly without a stenting tube. Often the stoma begins to contract within hours of feeding tube removal. The physician may be presented with a very narrow ostomy and a tract that is difficult to identify or thread. A sound or blunt stylet can be passed down the tract more easily than a tube. This procedure can identify the opening and direction of the tract for easier tube passage. When a guide wire passes easily

down a narrow tract but the needed feeding tube does not, it may be possible to dilate the tract with dilators or a dilation catheter. [83] Viscus puncture, viscus abdominal wall separation, and false tract creation with subsequent tube misplacement are risks of

dilation procedures. Such procedures should generally be left to the surgeon. If tube replacement will be delayed, maintain the narrow tract with the largest available easily placed stent, usually a Foley catheter. Always secure the stent against internal migration.

The position of the gastrostomy tube should be checked by air insufflation and aspiration of gastric fluid, as is done with nasoenteric tubes. It is wise to document the results of this testing in the medical record. Air should enter the stomach without resistance and should produce immediate borborygmi. Gastric fluid should return with aspiration. It may be necessary to insert a small volume of water to get good return. Water pooling in the soft tissue may be aspirated back through a misplaced catheter. Good tube placement is indicated when more fluid returns with aspiration than was originally placed into the catheter. If replacement of the gastrostomy or jejunal tube required the overcoming of any resistive force or if either the air or the aspiration test yields uncertain results, a radiographic study with contrast should be performed. Peritoneal infusion of feeding solution can be fatal.

Complications of gastrostomy include wound infections around the catheter, performance of an unnecessary laparotomy for suspected leakage, gastrocolic fistula, pneumatosis cystoides intestinalis, bowel obstruction, peritonitis, and hemorrhage. [74] Jejunostomies can cause most of these complications, as well as other types of fistulas and small bowel obstruction from adhesions or volvulus around the jejunostomy site. The most common complications of gastrostomy and gastroenterostomy are local skin erosions from leakage, wound infections, hemorrhage, and tube dislodgment. [46] Peritonitis and aspiration are the most critical complications of gastrostomy feedings. Jejunostomies are less prone to stomal leakage and cause less nausea, vomiting, bloating, and aspiration than do gastrostomies.

Dislodgment of gastrostomy and jejunostomy tubes is most common in the 2 weeks following ostomy creation. [85] Extrusion of the gastrostomy tube is usually caused by excessive tension applied to the tube. Only gentle contact of the gastric and abdominal walls is desirable. Uncooperative patients should be restrained, and mittens are often particularly helpful. Sutures and large mushrooms or balloons do not prevent purposeful removal of the gastrostomy tube by the uncooperative patient.

A small amount of drainage is to be expected at the tube entry site. Local leaks of gastric juices may macerate and irritate the skin, predispose to local infections and abscesses, and encourage the development of small granulomas.  Granulomas are particularly common in children. They can be treated with silver nitrate at the time of dressing changes. Any dressing used around the entry site of an enteral nutrition tube should absorb fluid and not encourage persistent moisture. [73] An unusually large stoma may promote a leak. Although insertion of a larger tube or firmer traction on the tube may be transiently effective, these measures often result in further stomal enlargement. Rigid gastrostomies promote leakage by widening the stoma as they pivot. Insertion of a soft, pliant feeding tube through the widened stoma is often easy and allows later contraction of the stoma. [1] If these techniques are ineffective, temporary removal of the feeding tube may allow the stoma to shrink. Large amounts of drainage around the stoma site may occur with high residual volumes. [79] The residual should be checked

and feedings withheld until residuals are <100 mL. Feeding residual should be checked every 4 hours when a patient is on continuous drip feeding. [82]

Pneumoperitoneum after percutaneous gastrostomy is neither unusual nor dangerous. Benign pneumoperitoneum may be present as long as 5 weeks after percutaneous endoscopic gastrostomy. Pneumatosis cystoides intestinalis can occur through the defect in the bowel wall created for the enterostomy tube. Although often clinically insignificant, its occurrence suggests air under pressure in the small bowel. Nasogastric suction and diet change generally permit resolution of the problem. Catheter or feeding tube removal is usually not required. [75]

Clinically significant pulmonary aspiration can occur with gastrostomy feeding. Methods of checking for silent pulmonary aspiration include checking tracheal aspirates with a glucose oxidant reagent strip or placing methylene blue in the formula and monitoring tracheal aspirate for pigmentation.

A Foley balloon accidentally inflated in the small bowel or esophagus can lead to perforation or obstruction. [87] Careful inflation of the balloon soon after it has entered the stomach prevents viscus perforation. A gastrostomy tube may migrate in the stomach and obstruct the gastric outlet. This complication manifests itself clinically with vomiting and high residuals of feeding solution. Volvulus and jaundice may also occur as a result of balloon migration. This problem can be alleviated by gently pulling back the tube. If the balloon of a Foley catheter has migrated into the small bowel, deflation of the balloon before pulling it back further reduces the risk of intussusception. An outer crossbar will prevent distal migration.

Gastrocolic fistula usually manifests itself as copious diarrhea. Once it is confirmed, treatment consists of removal of the gastrostomy tube. Later creation of a gastrostomy in a different location may be possible. The patient may require hospital admission for nutritional support and monitoring of fluid and electrolyte status.

An external bolster that is snugged down too tightly may result in a short stoma and embedding of the internal bolster into the abdominal wall. An abscess may result. Overly tight external bolsters should be loosened. The correct position is 1 cm from the external abdomen.

Dacron cuffs can serve as the nidus for an abdominal wall abscess. Generally the cuff cannot be removed independently of the tube. A replacement tube without the cuff can be inserted, if one is careful not to dissect the tube into the wall of the abscess. Extensive abscesses may require incision and drainage.

## CLOGGED FEEDING TUBES

Clogging is a problem common to all feeding tubes. Although it may only be a temporary solution, it is prudent to attempt to unclog a tube before it is replaced, especially if the tube has a complex placement or the physician is unsure of how the tube is secured internally. However, whenever feasible, clogged tubes should be replaced with a new

tube.

Large gastrostomy tubes are the least likely to clog. Gastrostomy tubes at least 28 Fr in size can tolerate home-blenderized foods and viscous feeding solutions. Isosmotic feeding solutions are tolerated by fairly narrow tubes and cost one sixth of what elemental feedings cost. Isosmotic feedings will clog needle catheters. [45] When tube lumina are 14 Fr or smaller, all pills and the contents of all capsules should be dissolved in water to prevent tube obstruction. [73]

Acid precipitation of feeding formulas is an important factor in the occlusion of gastrostomy and nasogastric feeding tubes. Sodium and calcium caseinate and soy protein molecules are most soluble at a pH of 6.3 to 6.6 and least soluble at a pH of approximately 4.7. [88] They are insoluble in nonpolar organic solvents. A study of 14 feeding solutions showed Pulmocare, Ensure Plus, and Osmolite to be the most clog-prone on exposure to acidic solutions; Citrotein had the least tendency to clog. [89]

Kinking is a frequent cause of tube blockage during the immediate post-reinsertion period. Withdrawing the tube a few centimeters usually relieves the kink and obstruction. A persistently recurring kink requires tube removal and insertion of a fresh tube.

Accumulated feeding solution or medication precipitates are very difficult to clean or remove. Milking a pliant tube backward may remove some of the cheesy precipitates. Guide wires or stylets may clear the proximal portion of a clogged tube lumen but are unsafe to use in subcutaneous (SQ) areas of the lumen because they can puncture the tube and injure the patient or create a tube leak.

Fogarty arterial embolectomy catheters can be used to unclog jejunostomy [90] and gastrostomy tubes. The soft tip of the Fogarty catheter is inserted into the feeding tube and advanced while the insertion distance is monitored to avoid penetrating farther than the length of the feeding tube. The allowable length of insertion should be premeasured. A No. 4 embolectomy catheter is suitable for a 10 or 12 Fr tube, whereas a No. 5 catheter should be used in 14 Fr feeding tubes. When the catheter meets an obstruction, the balloon can be inflated, which usually opens the obstruction sufficiently that catheter passage can continue. Once the Fogarty has been manipulated to just proximal to the internal feeding opening, it is withdrawn while the balloon is intermittently inflated and deflated gently. The catheter should not be withdrawn while inflated, because it and the feeding tube tend to move as a unit. The procedure may need to be repeated several times. Contrast injection to confirm tube position and integrity should be performed after declogging is completed. [90]

Irrigation with carbonated beverages and high-pressure irrigation with small-volume syringes have also been recommended as techniques for unclogging feeding tubes. Although irrigation seems like a straightforward and simple solution, these techniques are generally ineffective; furthermore, the possibility exists for dangerous tube ruptures with internal leakage. Broviac catheters are especially prone to tube aneurysms that can rupture under pressure. [70] Tubes unclogged by forceful irrigation or by deep luminal

probing should be radiographed after injection of contrast to check tube integrity.

Enzymatic declogging of feeding tubes may sometimes be effective. Most enzymatic preparations are insoluble in an acidic environment and require mild alkalinity for effective action. Such precipitates can add to the obstructing material. One study suggested that 2 enzyme preparations can soften clog consistency sufficiently to enhance tube clearing with insufflation. [88] A crushed chymotrypsin tablet or 2 papain tablets (effervescent Allergan) can be dissolved in 2 mL of distilled water and irrigated into the drained feeding tube. A 12.5-cm catheter may make such irrigation more effective. The feeding tubes should be closed or clamped and left for 1 to 4 hours. One study attempted tube insufflation only at 4 hours, but clog changes were noted after one-half hour. [88] Insufflation of air with a 50-mL syringe also may clear the tube.

Most clogged tubes should be replaced. Tubes that have been unclogged by using force should be radiographed after water-soluble contrast injection (see section Transabdominal Feeding Tube Replacement) to check for internal leaks. Regular tap water irrigations and selection of a feeding solution appropriate for the diameter of the feeding tube are vital to prevent tube clogging.

## CONCLUSION

Emergency physicians must commonly manage the patient who requires nasogastric tube placement or management of a dislodged or malfunctioning feeding tube. While placement of these tubes is generally straightforward in the cooperative patient, many patients requiring such tubes will be uncooperative or have an altered sensorium. Therefore, careful attention to the details recommended for these procedures is encouraged, as the complications associated with a misplaced tube can be disastrous.

# Chapter 44 - Balloon Tamponade of Gastroesophageal Varices

**Edward A. Panacek**

More than 2% of all hospital admissions in the United States and more than 5% of admissions from emergency departments (EDs) are related to acute gastrointestinal (GI) bleeding. In the vast majority of these patients, the bleeding source is in the upper GI tract. Bleeding from varices, both esophageal and gastric, is the third most common cause of upper GI bleeding, accounting for about 20% of cases. [1] Variceal hemorrhage is the most dramatic and life-threatening complication of portal hypertension. After 1 bleeding episode, the risk of a second is 70%, accounting for up to 50% of all deaths in patients with cirrhosis. [2] The risk of mortality from each bleeding episode ranges from 20 to 84% and depends on the etiology of the portal hypertension, the hepatic functional reserve of the patient, and the duration of hemorrhage prior to presentation. [3] Individuals with noncirrhotic portal hypertension and intact hepatic function generally tolerate the hemorrhage better than those patients with cirrhosis and advanced hepatic dysfunction. The greatest risk of death is in the first few days after the onset of variceal hemorrhage. [3] Therefore, prompt and effective therapy during this initial period is essential to maximize the patient's chance of survival.

## BACKGROUND

The use of balloons to control variceal hemorrhage dates back to the 1930s, although the balloons were initially filled with water rather than air. [4] In 1950, Sengstaken and Blakemore first described the technique of a double-balloon tamponade system similar to what is currently used. [5] During the subsequent 30 years, the Sengstaken-Blakemore tube underwent a number of modifications or refinements by Linton, Nachlas, Boyce, and Edlich.  These modifications generally carried the name of their originator and altered the size or shape of the respective balloons or added aspiration ports in the stomach or the esophagus to allow monitoring of ongoing bleeding.

Currently there are 2 types of gastroesophageal balloon tamponade (GEBT) tubes commonly available: the 3-lumen Sengstaken-Blakemore tube (gastric balloon, esophageal balloon, and gastric aspiration) and the 4-lumen Minnesota tube (which adds an esophageal aspiration port). [8] [9] Figures 44-1 and 44-2 show these 2 types of tubes. Institutions generally stock a single type of tube. Most hospitals also generally only stock adult sizes, although pediatric tamponade tubes do exist. [10] Although GEBT tubes are used less frequently today than previously, their use can still be temporizing or life-saving. Although these tubes are not usually placed in the ED, most emergency physicians will encounter a GEBT tube during their professional career and should be familiar with their structure, placement, function, and management. This may be particularly true in more remote settings, where an invasive gastroenterologist is not readily available.

## INDICATIONS AND CONTRAINDICATIONS

A nasogastric (NG) tube should be placed in all patients in whom GI bleeding is suspected or known, regardless of the presumed source or degree of the bleeding. There is no evidence that passage of an NG tube in a patient with esophageal varices results in variceal trauma or increases bleeding. [11] Lavage should be carried out, ideally with the patient in the left lateral decubitus position, to determine if the upper intestinal bleeding is limited in extent (i.e., gastric aspiration becomes clear with about 1 to 2 L of fluid). Lavage is generally performed to decompress the stomach and minimize the risk for further emesis. Removal of large clots also allows gastric wall contraction in those cases where the upper GI bleed has a gastric origin (see Chapter 43) . Ongoing lavage has little effect on gastric hemorrhage, and extended lavage with cold solutions may induce hypothermia.

When the gastric aspirate does not clear with lavage, more aggressive therapeutic approaches must be considered. The approach to patients with acute hemorrhage from esophageal varices is substantially different from that used with other causes of upper GI bleeding. [11] The presence of advanced hepatic disease, evidence of portal hypertension, or prior known variceal hemorrhage greatly increases the likelihood of a variceal source for the bleeding. [1] However, in 25%

to 50% of such cases, upper GI bleeding can still be due to nonvariceal sources. [12] For this reason, emergent endoscopy is indicated in such patients whenever available. [11] Endoscopic sclerotherapy and other techniques are the most effective therapy to stop active bleeding from varices and to stabilize varices that have recently bled.

The other early therapeutic option for variceal hemorrhage is IV vasopressin, octreotide, or somatostatin, potent nonselective vasoconstrictors. They can be used in conjunction with sclerotherapy or as the sole therapy when sclerotherapy is unsuccessful or unavailable. These drugs must be used with caution in patients with coronary artery or other vascular disease because of the risk of adverse ischemic effects. [15]

When sclerotherapy is unavailable or unsuccessful and vasoconstrictor therapy is not adequate, balloon tamponade is the next therapeutic option. Balloon therapy is less successful than sclerotherapy but can control bleeding acutely in up to 80% of cases. [16] [17] [18] The role of balloon tamponade in these other therapies is depicted in Figure 44-3 .

Some authors have advocated the use of esophageal balloon tamponade based upon transfusion requirements or ongoing hemorrhage exceeding a certain amount (usually 2000 mL of blood within a 24-hour period). [1] Other recommendations have focused on endoscopic findings, including active variceal bleeding that cannot be controlled with sclerotherapy or traumatic longitudinal tears of the esophagogastric junction (Mallory-Weiss tear), with active persistent bleeding. [16] However, these indications are generally more relevant for the inpatient setting. In the absence of endoscopy, the indication in the ED would be a patient with known portal hypertension or prior variceal hemorrhage who has substantial ongoing upper GI bleeding that does not clear with

gastric lavage and vasoconstrictor therapy. When endoscopy is available, the indication for a GEBT tube is substantial ongoing variceal hemorrhage that cannot be controlled with endoscopic interventions. [16]

The use of esophageal balloon tamponade tubes is associated with a number of serious and potentially lethal complications. The overall complication rate is much higher than for most other procedures that emergency physicians perform. [19] Therefore, clear indications for GEBT tube use and a complete understanding of the instructions for their placement are required. GEBT tubes are best placed by the individual most knowledgeable about their use.

GEBT tubes are meant only for temporary bleeding control. [16] Although most studies have shown that they provide initial hemostasis in up to 80% of patients with bleeding from esophageal varices, they have not measurably affected the long-term mortality of these patients. [17] Their success rate improves when used in conjunction with vasoconstrictor therapy. [20] However, these therapies often do not control hemorrhage definitively, and arrangements for more definitive endoscopic procedures should be initiated.

## EQUIPMENT

Most hospitals stock only 1 type of GEBT tube. Ideally, the tubes are stocked in the ED itself or are readily available when needed. There are multiple companies making these tubes, and each company may include some of the items listed below. The equipment required for this procedure is as follows:

1. GEBT tube
2. Traction device or setup, including weights (see below)
3. Manual manometer or sphygmomanometer
4. Y tube connector, if not already part of the tamponade balloon ports
5. Vacuum suction device
6. Connectors for the suction tubing
7. Plastic tubing to connect to suction
8. Soft restraints
9. Topical anesthetic (spray and jelly)
10. Water-soluble lubricating jelly
11. 4 tube clamps (see text)
12. Large (e.g., 50 mL) catheter tip irrigating syringe
13. Surgical scissors for emergency balloon decompression

There are multiple ways of providing a traction setup for the tube, and individual hospitals may have specific policies. [9] Tubes can be taped to the mouth guard of a football helmet, although currently it is extremely unusual to see that technique used. Even in those institutions that try to keep a helmet available, it can rarely be located when needed. More often traction is maintained with a cube of foam rubber that is generally included in the package with the tube itself, as shown in Figure 44-2 . The tube is inserted between the 2 halves of the foam, and the foam "cuff" is placed against

the nose and taped.

The more common way of providing traction, and generally the way considered most effective, is through a pulley apparatus similar to the system used for orthopedic traction on lower extremity fractures. [21] It is a bit more cumbersome, and there are concerns regarding its use in disoriented or agitated patients, but it can effectively and consistently maintain the desired 0.45 to 0.91 kg (1 to 2 lb) of traction. The usual trapeze bar apparatus is used, but this requires 2 pulleys: 1 over the patient and 1 at the end of the bed. It also requires a longer rope (about 3 m [10 feet]), as well as the suspension hook and 0.23- to 0.45-kg (0.5- to 1.0-lb) weights.

Clamps are needed for the process of inflating the balloons, checking pressures, and maintaining balloon inflation. Never use bare standard hemostats for this purpose, as the serrations on the clamp jaws can puncture the rubber tubing. Specific tube clamps are best, if available (e.g., hosecock clamps). Alternatively, large surgical clamps or large hemostats can be modified by covering the clamping surfaces with sections of plastic or rubber tubing or multiple layers of tape.

## PROCEDURE AND TECHNIQUE

Patients requiring GEBT tubes are critically ill and must be closely monitored within the ED or in an intensive care unit. In the best of circumstances, these tubes can be difficult to place. However, these patients are often uncooperative and sometimes even combative due to encephalopathy. Control of the patient is imperative. Soft restraints should be used routinely, and sedating medications considered in most patients. If the patient is not awake, alert, and fully able to protect the airway, endotracheal intubation should precede placement of the GEBT tube (see Chapter 2) . These patients are at extremely high risk for regurgitation and aspiration, and the threshold for endotracheal intubation should be lower than usual. [22]

The procedure itself is unfamiliar to most emergency physicians and requires a number of items of equipment. Each of these should be checked prior to beginning the procedure. In particular, the balloons on the tamponade tube must be checked for patency and lack of leaks. If there is any question, the balloons should be submerged beneath water for checking. During testing, inflation of the gastric balloon is done in progressive steps (generally in 100-mL increments), up to the maximum recommended volume (generally 500 mL), while the pressure is monitored with the manometer. The pressure within the gastric balloon should not increase by >15 mm Hg with each progressive administration of 100 mL of air. The operator should note the pressure at full inflation of the gastric balloon. The esophageal balloon is simply checked for patency. The balloons are then deflated and coated with water-soluble lubricating jelly, preferably with a topical anesthetic.

The patient is positioned for the procedure by having the head of the bed elevated to about 45°, if possible. If the patient is unable to tolerate that position, the left lateral decubitus position is also acceptable. The posterior pharynx and nostrils are anesthetized with a topical anesthetic. The patient should already have undergone gastric lavage with a standard NG tube. At this point the stomach is maximally

evacuated (to decrease regurgitation), and the NG tube is removed.

After suctioning all air from the gastric and esophageal balloons and ensuring that they are maximally collapsed, each of the balloon ports is clamped, or the plastic plugs (if provided with the tube) are inserted into the lumens. This maneuver maintains deflation of the balloons during placement. In awake and alert patients, passage through the nostril is an option, but passage through the mouth is preferred, especially in tracheally intubated patients. The alert patient can assist the process by taking sips of water through a straw. The tube is then passed to at least the 50-cm mark, or the maximum depth allowed by the length of the tube, in the same manner as a standard NG tube. Suction is then applied to the gastric and esophageal aspiration lumens to minimize chances of further regurgitation or aspiration. Aspiration of gastric juice or fresh blood from the gastric lumen provides further evidence for proper location of the tube. However, its position must be confirmed radiographically.

Some authors recommend initial inflation of the gastric balloon with 50 mL of air to assist with radiographic visualization to ensure that the gastric balloon is below the diaphragm. [16] However, this is not routinely necessary, and the position of the distal end of the tube is usually obvious on radiograph. Absolute confidence regarding tube location is extremely important to reduce the risk of esophageal perforation caused by inflation of a misplaced balloon. Once gastric positioning of the distal balloon is ensured, the tube clamps (and plastic plugs if used) are removed from the gastric balloon inflation ports.

The pressure monitoring outlet for the gastric balloon lumen is connected to a manometer, similar to Figures 44-2 and 44-4 . Increments (generally 100 mL) of air are then introduced through the gastric balloon inflation lumen until the recommended total volume (generally 450 to 500 mL) fills the gastric balloon. Only air, never water or any liquid, is used to inflate the balloons. As the air is introduced into the gastric balloon, the intragastric balloon pressure is monitored. If the gastric balloon pressure at any step is 15 mm Hg greater than the intragastric balloon pressure at an identical volume noted prior to intubation (i.e., during testing), the gastric balloon may be located within the esophagus, and further inflation of the balloon could result in overdistention and rupture of the esophagus. If an elevated intragastric balloon pressure is recognized, the balloon should be deflated, advanced, and reintroduced into the stomach. A repeat radiograph is then obtained to confirm proper positioning prior to restarting the inflation procedure.

When the gastric balloon, positioned correctly in the stomach, has been inflated with the recommended full volume (generally 450 to 500 mL) of air, the air inlet and pressure monitoring outlet of the gastric balloon are each clamped. The tube is then pulled back gently until the resistance of the diaphragm is firmly felt. With a small amount of tension on the tube, the proximal end is secured using a traction device. The tube kit generally includes a sponge rubber cuff, which can then be fixed at the nostril and taped, if that approach is used. If the tube has been passed orally, an external pulley traction device is preferred. Once the tube is secured in place, the gastric aspiration port is attached to high intermittent suction. A final check should be made by irrigating the tube with water under auscultation. Lavage the gastric port until clear. [21] If there is any question

regarding correct tube position, obtain a repeat radiograph before proceeding.

If blood is detected continually in the gastric aspiration port (or in the esophageal aspiration port on a 4-lumen tube) despite lavage, the esophageal balloon should be inflated to the pressure recommended in the accompanying instructions (generally 30 to 45 mm Hg). Inflation of the esophageal balloon should be monitored continuously by a manometer attached to the esophageal balloon monitoring outlet. The esophageal balloon pressure should be maintained at the lowest level that will stop bleeding from each of the aspiration suction ports. In addition, the esophageal balloon pressure should not exceed the maximum listed in the instructions (generally 45 mm Hg). Once the balloon has been inflated to the desired amount, the port for the esophageal balloon is clamped, and periodic readings are taken.

If bleeding continues from the gastric aspiration port after full inflation of the gastric and esophageal balloons, it usually originates from a gastric rather than an esophageal varix. In this case, external traction on the tube should be increased. If a foam rubber block is used, the tube should be pulled to a more taut position and refixed to the nasal cuff at the point where the tube emerges from the nose. If the tube was passed through the mouth, and a trapeze-bar pulley system was used, adding additional traction is easy. An additional 0.45 to 0.91 kg (1 to 2 lbs) of weight can be applied to the pulley system, in progressive steps (maximum of 1.1 kg [2.5 lb]). [21] The pulley system should be attached to the GEBT tube through use of a nonoccluding knot (e.g. a bowline knot) passed around the area of the tube where all the respective ports separate. Tightening the knot directly onto the tube itself can occlude the ports.

Direct pressure from the tube can cause ulceration of mucosal surfaces within a matter of a few hours. Therefore, there should be frequent examinations to ensure that the tube is not placing excessive pressure on a given mucosal surface. If one is using an external pulley traction device, the angle of the tube, as it exits the mouth, is adjusted to minimize any such pressure by altering the location of the pulley. If the tube is passed through the mouth, precautions are also needed to preclude patient biting of the tube. A rubber or plastic mouthpiece should be placed in dentulous patients. Suction of the aspiration ports is then adjusted. The gastric port should have intermittent 60- to 120-mm Hg suction, and the esophageal port (for 4-lumen tubes) should be attached to 120- to 200-mm Hg continuous suction. [21]

After bleeding has been controlled by the tamponade, the pressure in the esophageal balloon is generally reduced by 5 mm Hg every 3 hours (or as specified in product instructions), until an intraesophageal balloon pressure of 25 mm Hg (or as specified in instructions) is achieved without ongoing bleeding. Note that continual elevated balloon pressures (>30 mm Hg) on the esophagus for long periods of time can result in mucosal ischemia and may induce esophageal necrosis. [21] Therefore, it is recommended that periodic deflation of the esophageal balloon be undertaken for approximately 5 minutes every 6 hours. If bleeding can be controlled with an intraesophageal balloon pressure of 25 mm Hg, this pressure is generally maintained for the next 12 to 24 hours. [16] [21] The pressure in the esophageal balloon can transiently vary with respiration and esophageal spasm. This may result in intermittent increases in the measured pressure of 30 mm Hg above baseline, although this should only be for transient periods. If it remains high, air

must be removed until the pressures are acceptable.

If a 3-lumen GEBT tube has been used, it will not have an esophageal aspiration port. Because the amount of oropharyngeal and esophageal secretions can exceed 1500 mL/day, additional suction proximal to the esophageal balloon must be provided. [21] This can be done with a standard 14 to 16 Fr NG tube passed to a position measured or calculated to be just above the esophageal balloon. This should be placed even if the esophageal balloon is not maintained in an inflated position, because an inflated gastric balloon will also interfere with the ability to swallow or pass secretions.

Once satisfactory positioning of the GEBT tube has been confirmed, the tube is generally then not disturbed for some 24 hours, unless necessary due to complications. [21] During this time, adjunctive therapy is ongoing and additional evaluation instituted. [16] These tubes are very uncomfortable, and patients should be provided with analgesics and sedation. In addition, soft restraints are needed on the arms to prevent the patient from dislodging the tube. If the bleeding does not remain controlled, other therapeutic interventions must be considered. These include endoscopic interventions with sclerotherapy, as well as emergency surgery. If these are not available, patient transfer may be necessary.

## COMPLICATIONS

Complications associated with the use of GEBT tubes are frequent and often very serious. [19] Table 44-1 lists both major and minor complications. Major complications have been reported to occur in 8% to 16% of patients. [9] Mortality directly related to use of these tubes is generally reported to be 3%.  However, 1 study reported the GEBT tube directly caused death in 22% of the patients in which it was used. [23] This must be considered in the context that variceal hemorrhage itself carries a 20% to 80% overall mortality. [16] Therefore, use of a GEBT tube is sometimes viewed as a desperate measure for a desperate disease process. GEBT tubes can be temporizing or even life-saving, but the high associated complication rate requires an individual risk-benefit analysis for each patient in whom it is being considered.

Aspiration pneumonitis is probably the most frequent major complication. [19] This can result from aspiration of oral secretions; gastric contents; or, most commonly, blood. [9] The volume of aspiration can be substantial, and associated deaths have been reported.  The likelihood of this complication can be decreased by evacuating the stomach prior to placement of the GEBT tube and having a low threshold for tracheal intubation to protect the airway. [22]

Asphyxia due to airway obstruction has been reported to occur with dislodgment of the tube such that the esophageal balloon migrates into the oropharynx. This is more likely when the tube is passed through the nares such that the inflated esophageal balloon cannot pass through the nasopharynx, and it is one of the reasons that oral placement is preferred. Tube migration can be prevented by maintaining full inflation of the gastric balloon through periodic monitoring

| TABLE 44-1 -- Complications of Esophageal Balloon Tamponade Therapy | | |
| --- | --- | --- |
| | **Common** | **Uncommon** |
| Major complications | Aspiration pneumonia<br>Asphyxiation<br>Esophageal necrosis | Esophageal perforation<br>Duodenal rupture<br>Tracheobronchial rupture<br>Periesophageal abscess<br>Mediastinitis |
| Minor complications | Gastroesophageal ulceration<br>Regurgitation<br>Chest discomfort<br>Back pain<br>Pressure necrosis of nose or lip | Epistaxis<br>Pharyngeal erosions<br>Pressure necrosis of tongue<br>Hiccups |

of pressures and radiographic reconfirmation. Of course, airway obstruction can be completely prevented by prophylactic tracheal intubation. If the tube suddenly migrates, resulting in an airway obstruction in a nontracheally intubated patient, the balloons must be immediately deflated and the tube extracted. This can be achieved most quickly by cutting across all of the tube lumens just distal to the bifurcation points. This will immediately vent all of the balloons, and the entire tube can be extracted. For this reason, it is recommended that surgical scissors be kept available at the patient's bedside whenever a GEBT tube is in place.

The other relatively common major complication is esophageal perforation or rupture. [19] [23] [24] This occurs with overinflation of a misplaced gastric balloon and can be prevented through careful employment of the placement steps already outlined. However, this can also occur as a result of esophageal mucosal necrosis caused by excessive or prolonged pressure in the esophageal balloon. The treatment is immediate removal of the GEBT and initiation of diagnostic studies (e.g., contrast swallow) and broad-spectrum antibiotics (for potential mediastinitis). Again, risk for this complication can be decreased by periodic deflation of the balloons at 6-hour intervals and limiting the amount of pressure in the esophageal balloon to the minimum amount necessary to control bleeding. This problem is also more common when the balloons are left inflated for >24 hours. [21]

Common minor complications include pain, discomfort, and local pressure effects of

gastric or esophageal erosions or mucosal ulcers.  These latter can be minimized by frequently checking and optimizing tube position to minimize pressure on the nasal or oral mucosa, the tongue, and other structures. Although less common, other complications can occur; these are also listed in Table 44-1 .

## INTERPRETATION

GEBT tubes are used to temporarily control bleeding varices. This is assessed by monitoring the rate of blood aspirated from the gastric and esophageal ports after tube placement. It is not uncommon for the GEBT tube to fail to control the hemorrhage. When significant bleeding continues, correctable causes should be considered which include malpositioned balloons, inadequate balloon pressures, and misdiagnosis of the site of bleeding (e.g., a duodenal ulcer instead of variceal source). [21] [24] When these have been addressed but bleeding continues, other therapeutic options must be considered, including sclerotherapy, angiographic embolization, and surgery (see Fig. 44-3) .

## CONCLUSION

Gastroesophageal balloon tamponade is an uncommon procedure in general, and particularly for emergency physicians. Some emergency physicians may not have performed this procedure previously. However, the procedure can temporarily control an exsanguinating hemorrhage from gastric or esophageal varices in about 80% of patients when other options are unsuccessful or unavailable. As such, the emergency physician may periodically be called upon to place a balloon tamponade tube. Because of the high incidence of associated serious complications, the procedure should be used in carefully selected patients in whom the potential benefit is favorable, given the risk of exsanguination.

Each hospital is unlikely to stock more than 1 tube type, and this may not be the type most familiar to the physician. However, currently most balloon tamponade tubes are fairly similar and are packaged with a complete set of instructions. These instructions must be carefully reviewed prior to tube placement. There can be great difficulty locating these tubes within the hospital when they are suddenly needed, so advance planning is recommended, when possible.

The most serious common complications are those of aspiration and airway occlusion. [19] A low threshold for prophylactic tracheal intubation is recommended. The most serious iatrogenic complication is due to balloon inflation of a misplaced tube, with resultant esophageal rupture. This is avoidable by carefully following the instructions and insisting on radiographic confirmation of tube position before fully inflating the balloons.

# Chapter 45 - Decontamination of the Poisoned Patient

**Patrick E. McKinney**

At least 2 million cases of accidental and intentional poisoning occur each year in the United States. [1] In 1 report, approximately 1% of all patients seen in an emergency department were suffering from acute drug overdose. [2] The diagnosis and management of acute poisoning in children and adults are common problems and require that physicians caring for these patients be expert in the procedures used in their management.

After initial stabilization of the acutely poisoned patient, an attempt is usually made to prevent further absorption of any toxin. If the patient has a dermal exposure, skin decontamination may be indicated.  When the toxin is ingested, attempts may be made to decrease further absorption from the gastrointestinal (GI) tract. [3] Methods for accomplishing this vary and have evolved over time. Techniques include ipecac-induced emesis; gastric lavage; administration of 1 or more doses of activated charcoal; administration of cathartics; whole-bowel irrigation; and, rarely, gastrotomy or endoscopy. This chapter focuses on decontamination of the poisoned patient and discusses its indications and contraindications, as well as technical aspects of specific procedures and their complications.

## BACKGROUND

The historical use of a hollow tube to evacuate poison from a patient's stomach dates from the early 1800s and has been thoroughly reviewed by Major. [5] Kussmaul, who is commonly credited with inventing gastric lavage for the treatment of poisoning, did much to popularize its use after his 1869 publication. [6] During the next 75 years, gastric lavage was widely held to be the most effective procedure for emptying the stomach of an acutely poisoned patient.

Although the benefit of stomach emptying for the routine treatment of poisonings appears obvious, the safety and efficacy of gastric lavage was first questioned by Harstad and coworkers, who found that in only 5 of 80 patients suffering from severe sedative poisoning could >500 mg of drug be recovered in the gastric washings. [7] In addition, they reported that particles of previously administered activated charcoal could be found in the respiratory tract of some of the patients who died, suggesting that aspiration of gastric contents had occurred. They concluded that gastric lavage was relatively ineffective and potentially unsafe, but a number of methodologic problems exist with this study. The size of the stomach tube was not specified, the drug assay method used (crystallization) was crude, and the positioning of patients may not have been optimal. In addition, patients' airways were often unprotected during the procedure. As a result of this study, gastric lavage fell into some disrepute and was thereafter performed less often at many medical centers. [8] This practice change was accompanied by a growing interest in the use of induced emesis as a means of evacuating the stomach in poisoned patients.

The 1950s saw the beginning of a controversy regarding the relative efficacy of gastric lavage vs. ipecac-induced emesis that persists to this day. Boxer and colleagues compared ipecac vs. lavage for gastric emptying in children who had ingested unknown quantities of aspirin. [11] Their 17 patients were randomized to be treated with ipecac-induced emesis followed by gastric lavage or to undergo gastric lavage followed by ipecac-induced emesis. The relative quantities of drug retrieved by each method were then compared for each patient, and a ratio was established. More drug was apparently removed by induced emesis than by lavage, but the description of the lavage technique used in this study was incomplete. Particularly important omissions from the report included specifications of the gastric tube diameter, the patient's position, the lavage solution volume, and the time delay before instituting treatment. Finally, only negligible amounts of aspirin were removed by either method.

In a series of 259 severely poisoned patients, Matthew and colleagues showed that carefully performed gastric lavage could remove clinically important amounts of ingested drug. [12] However, because the actual ingested dose was unknown, the true efficacy of gastric lavage could not be determined. Burke lavaged 10 adult volunteers undergoing general anesthesia for elective bronchoscopy and demonstrated that water-soluble radiopaque dye can be removed from the stomach if a large gastric tube is used and careful attention is paid to lavage technique and patient position. [13]

Tandberg and associates compared lavage to emesis using cyanocobalamin tablets as a tracer in 18 adult volunteers on 2 separate days. [14] Ipecac-induced emesis was initiated 10 minutes after ingestion of the tracer. Gastric lavage was carried out on another day, also beginning 10 minutes after tracer ingestion. A mean of only 28% (range, 6% to 70%) of the administered cobalt tracer was recovered by ipecac-induced emesis, whereas 45% (range, 19% to 68%) was recovered with gastric lavage. These recovery rates are disappointing, especially in view of the short time to the initiation of gastric emptying, the absence of other materials in the stomach, and the optimal circumstances of the procedures.

Young and Bivens also examined the effectiveness of lavage compared to emesis using technetium 99m capsules in volunteers. [15] In their study, lavage was delayed until the point at which emesis occurred during the preceding ipecac treatment day. They found a slight advantage to emesis, with a mean of 54% of marker returned when ipecac was administered within 5 minutes of marker ingestion. Volunteer studies have been unable to detect a difference in drug recovery when 1 hour was allowed to elapse between drug administration and commencement of ipecac-induced emesis or gastric lavage.

Prospective studies of actual overdose patients have been unable to demonstrate a clinical advantage to gastric emptying (by either emesis or lavage) over simple administration of activated charcoal. However, Kulig and colleagues found evidence for benefit if obtunded patients were lavaged within 1 hour of ingestion. [18] Merigian and colleagues found no benefit to lavage in a similar study, which also excluded patients who had ingested methanol, ethylene glycol, lithium, monamine oxidase inhibitors, heavy metals, formaldehyde, mushrooms, digitalis, and sustained-release products. [19] The number of seriously poisoned patients was relatively small in both of these studies.

However, these studies do support the use of activated charcoal as the sole method of GI decontamination in mildly poisoned patients.

Activated charcoal use has been extensively reviewed by Cooney. [20] Charcoal has been used in medicine since the time of ancient Egypt, but its use in the treatment of poisoning was first documented in France in the 1800s. A French chemist, M. Bertrand, swallowed 5 g of arsenic trioxide mixed with charcoal in a public demonstration and survived. [21] A similar demonstration using strychnine was performed by the pharmacist P. F. Touery in 1831. Despite in vitro and in vivo evidence in animals and humans, activated charcoal use for treatment of poisoning remained sporadic until the 1960s, when it was popularized by Holt and Holz. [22]

One of the newest methods of GI decontamination is whole-bowel irrigation (WBI). WBI appears to be most useful in specific situations where substances are not well adsorbed to activated charcoal or require "flushing" through the GI tract. Case reports document the use of WBI for ingestions of extended-release verapamil, extended-release theophylline, iron, lead glaze, disc batteries, zinc sulfate, diquat, and paraquat and in "body packers" and "stuffers." Controlled volunteer studies have shown beneficial effects from WBI after ingestion of extended-release lithium and enteric-coated aspirin.

In summary, the data on gastric emptying are conflicting. In most circumstances it is probably impossible to predict which procedure will most effectively accomplish gastric decontamination and to determine how much drug should be removed to provide clinical benefit. Currently available human studies suggest that activated charcoal alone may be adequate decontamination therapy for most asymptomatic or mildly symptomatic patients or for those symptomatic patients who present for treatment >1 to 2 hours post ingestion. In the setting of potential delayed drug absorption (e.g., sustained-release medications or medicines producing delayed gastric emptying), this time interval may be extended. One of the strongest arguments supporting the use of lavage over emesis in the emergency department is that the procedure can be done immediately on presentation (compared with the 15- to 20-minute delay for ipecac to produce emesis), and charcoal can be immediately instilled to limit further absorption. This immediacy of emptying may provide a benefit to patients who have taken an otherwise lethal overdose.

## GENERAL CONSIDERATIONS

Routine gastric emptying procedures are no longer the standard of care for *all* suspected drug overdoses. Most clinicians agree that gastric emptying (whether by ipecac or gastric lavage) should be avoided in patients with a clear history of ingestion of a nontoxic substance or ingestion of an inconsequential amount of toxic substance. [33] Ingestion of a substance with a low order of toxicity that is well adsorbed to activated charcoal does not require gastric emptying (e.g., benzodiazepines). In addition, gastric emptying should not be performed if a significant time after ingestion has passed and there is no reasonable suspicion that the substance is still in the stomach (e.g., 4 hours after ethanol ingestion). Finally, gastric emptying is rarely indicated if the patient has vomited repeatedly prior to arrival.

The decision to pursue gastric emptying with ipecac-induced emesis or lavage should be based on 4 major factors: the time since ingestion, the amount of medication ingested, the relative toxicity of the substance, and the clinical condition of the patient. The first factor, the time delay between ingestion of the poison and initiation of gastric emptying, is an important consideration. For rapidly absorbed drugs, such as the short-acting barbiturates, older evidence suggests that attempts at gastric emptying >4 hours from the time of ingestion are not likely to be of clinical value. [12] The prospective randomized study of clinical outcome in poisoned patients by Kulig and colleagues found fewer cases of clinical deterioration with gastric lavage (compared with those from activated charcoal alone) *only in that subset of patients who were both obtunded and seen within 1 hour of their ingestion.*[18]

However, many conditions could theoretically result in delayed gastric emptying and the potential for retrieval of clinically important amounts of drug >1 hour after ingestion. Physiologic gastric emptying is especially likely to be delayed in patients with diminished or absent bowel sounds; in those who have ingested opiates or drugs with anticholinergic properties (e.g., cyclic antidepressants, antihistamines, benztropine, and other strong anticholinergics); or in patients who have ingested large amounts of relatively insoluble drug, such as aspirin, ferrous salts, or sustained-release preparations. Other conditions that may delay physiologic gastric emptying include the presence of food in the stomach, a high proportion of solid material in the stomach, [34] high nutritive density of the last meal, [35] high viscosity of stomach

contents, [36] and positioning in the left lateral decubitus position. [37] Finally, the history of time of ingestion may be inaccurate, a fact that should be kept in mind when considering timing as a guide to treatment choice.

The second and third factors in assessing the need for gastric emptying are the toxicity and amount of the ingested substance. Minimally toxic agents clearly do not require gastric emptying. Likewise, most authorities agree that *recent ingestion of significantly toxic agents* (e.g., cyclic antidepressants, beta-blockers, theophylline, cyanide) should be considered for gastric emptying. [33] However, there is a considerable gray area involving substances of intermediate toxicity (e.g., sedative-hypnotics, anticonvulsants, antihistamines). In these cases, activated charcoal and supportive care may be sufficient. Many clinicians forego gastric emptying in these cases for activated charcoal, but patients who have ingested large amounts and who present early to the emergency department (even if asymptomatic) may benefit from gastric emptying. Finally, given the availability of potent antidotes (naloxone, flumazenil, Digibind, *N*-acetylcysteine), the need for gastric emptying in specific poisonings may be diminished.

The final factor in determining the need for gastric emptying is the clinical condition of the patient. As noted above, Kulig and associates found a clinical benefit to lavage only in patients who were obtunded and presented within 1 hour of ingestion. [18] Although no benefit was detected when obtunded patients were lavaged >1 hour after ingestion, there were few patients in this group. It seems reasonable to lavage all obtunded, intubated patients, provided there are no contraindications.

If the decision is made to empty the patient's stomach based on the criteria above, then the patient's level of consciousness is the most important factor to consider. Neither ipecac-induced emesis nor gastric lavage *without* airway protection should be performed in patients likely to have or to develop diminished airway protective reflexes during the procedure. This includes patients with a diminished sensorium, seizures, or the potential to develop these clinical findings. Gastric lavage with a cuffed endotracheal tube in place should be carried out instead. The use of depolarizing or nondepolarizing muscle relaxants may be required to intubate the combative, confused, or obtunded patient with diminished airway protective reflexes.

# IPECAC-INDUCED EMESIS

### Indications and Contraindications

Ipecac use in the emergency department is currently limited to a few specific indications. It may be used in pediatric patients who present early after ingestion, in adults who present immediately after large ingestions of agents with delayed toxicity that may be poorly adsorbed to activated charcoal (e.g., iron, lithium, extended-release preparations), and after plant or mushroom ingestion.

Ipecac should not be given if central nervous system depression or seizures could potentially occur within the expected time frame of emesis (up to 2 hours after ipecac administration). Ipecac is also contraindicated in patients who are already vomiting or in patients who have ingested acids, alkalis, or hydrocarbons, as emesis might cause further esophageal injury or aspiration. Most clinicians feel that ipecac use in the emergency department is contraindicated after acetaminophen ingestion. [39] Persistent vomiting could preclude the timely administration of *N*-acetylcysteine.

### Technique

Syrup of ipecac is packaged in 15- and 30-mL containers. Standard dosages of ipecac and accompanying clear fluids based on patient age are as follows [3] :

> *6 to 12 months:* 5 to 10 mL ipecac plus 15 mL of clear fluids per kilogram body weight

> *12 months to 12 years:* 15 to 30 mL ipecac plus 240 mL of clear fluids

> *>12 years:* 30 mL plus 240 to 480 mL of clear fluids (repeat once in 20 to 30 minutes as needed)

Some clinicians recommend that the child be jiggled or the stomach massaged beginning 10 to 15 minutes after ingestion of the ipecac and fluids if no spontaneous emesis has occurred.

A single dose of 10 mL has been recommended by some investigators [40] for infants 6 to 12 months old, although there is some concern as to whether the gag reflex is sufficiently developed in these infants.

## Complications

The major advantage of ipecac use is its ease of administration and its potential for home use or administration by emergency medical service personnel. The main disadvantages of ipecac administration are the delay after administration to onset of emesis and the duration of emesis. The delay to emesis averages 20 minutes, but some patients may not vomit after a single dose and may require a second dose. [41] Patients usually experience 2 or 3 episodes of emesis over 20 minutes to 1 hour. [42] Drug absorption and clinical deterioration with compromise of airway protective reflexes may occur while waiting for emesis to begin or resolve. Persistent emesis may also delay administration of activated charcoal or oral antidotes such as *N*-acetylcysteine. Pediatric patients receiving ipecac in the emergency department had an additional delay of 1.7 hours to receive activated charcoal and were more likely to vomit afterward. [43]

Complications from syrup of ipecac use are uncommon. Perhaps the most significant complication from syrup of ipecac-induced emesis is pulmonary aspiration in a patient who has lost airway protective reflexes. Patients may become drowsy, but significant sedation has not been reported from ipecac alone. Other rare reported complications include Mallory-Weiss tear, [44] gastric herniation through a diaphragm defect, [45] gastric rupture, [46] cerebral hemorrhage, [47] pneumomediastinum, [48] and vagally mediated bradycardia.

# GASTRIC LAVAGE

### Indications and Contraindications

The advantages of gastric lavage include the fact that it can be performed immediately; it can be performed on patients with a decreased level of consciousness once the airway is protected; and finally, drug that is removed by lavage is no longer available for absorption (there is the possibility of desorption of drug from the activated charcoal remaining in

the GI tract). The main disadvantages of gastric lavage are the significant time commitment of health care personnel for the procedure and the risk to the patient. Watson and colleagues found that mean time required by experienced emergency department nurses to perform lavage was 1.3 ± 0.6 hours while lavaging an average volume of 12.8 L. [49]

Lavage should be considered in patients who present early (within approximately 1 hour) after ingestion of a potentially significant toxin, especially when large numbers of pills or gram amounts of toxin have been ingested. It may also be considered in patients with highly toxic ingestions in the setting of anticipated delayed physiologic gastric

emptying. Finally, gastric lavage should be performed when patients with suspected toxic ingestion are already intubated, provided there are no contraindications present.

A common dilemma involves the decision of whether to lavage the combative patient. If the patient requires intubation for airway protection, lavage can be accomplished after sedation and paralysis for intubation. If the patient does not require intubation for airway control, a careful assessment of the potential lethality of the ingestion should be made. If the ingestion is thought to be significantly life-threatening, lavage following rapid-sequence induction and intubation may be indicated (see Chapter 3) . However, in many combative patients, alternatives to lavage, such as activated charcoal, should be considered. Evacuation of gastric contents by nasogastric tube can occasionally remove significant quantities of ethanol, although patients with a significant gastric ethanol load may not be easily identified. [50] Repeated attempts at lavage risk injury to both the patient and staff.

Contraindications to gastric lavage include (1) an unprotected airway, (2) possible ingestion of strong alkalis, (3) known esophageal strictures, and (4) ingestion of hydrocarbons (unless containing highly toxic substances such as pesticides, heavy metals, halogenated or aromatic compounds, or camphor). The regional poison center can be of great help in determining whether gastric emptying is indicated, especially when unusual hydrocarbon products are involved.

The ingestion of strong acids rarely produces severe esophageal injury but may cause deep burns of the stomach and the duodenum. Continuing injury for as long as 90 minutes has been demonstrated in experimental animals [51] and suggests that rapid removal of the ingested acid could be beneficial. Gentle passage of a small-bore flexible rubber nasogastric tube and aspiration of gastric contents after acid ingestion would be reasonable; however, this approach remains controversial.

**Equipment and Preparation**

If the decision is made to perform gastric lavage, careful attention to the details of the procedure results in increased safety for the patient and more effective removal of the ingested poison. Prior to lavage, the patient should have IV access secured and should be placed on a cardiac monitor and pulse oximeter. Suction with a large, rigid suction tip should be immediately available.

Before initiation of gastric lavage, the airway should be protected with a cuffed endotracheal tube if the level of consciousness is significantly depressed or the patient's airway-protective reflexes are diminished. The traditional way to test for this is to stimulate the posterior pharynx with a tongue blade or a cotton-tipped applicator. Alternatively, the lack of a grimace or blink reflex after touching the eyelashes is strong evidence for an unprotected airway and the need to intubate the trachea before gastric lavage. [38] Cautious direct laryngoscopy with prompt endotracheal intubation in the patient who tolerates laryngoscopy may be used in questionable cases; the patient who tolerates the procedure probably needs it. If patients are fully alert and awake, lavage may be done without tracheal intubation. Attention to patient positioning, prompt suctioning, and consideration of immediate gastric tube removal should be made in

nonintubated gastric lavage to optimize protection of the airway.

The position of the patient during gastric lavage is important. All patients should be placed in the left lateral decubitus position with the head down (approximately 10° to 15°). This position diminishes the passage of gastric contents into the duodenum during lavage, a fact that has been well documented by fluoroscopy during the procedure (Fig. 45-1) . [13] In addition, the head-down position probably decreases the risk of pulmonary aspiration of gastric contents should vomiting or retching occur. The uncooperative patient's hands should be restrained to prevent removal of the gastric or endotracheal tube.

Large-diameter gastric hoses with extra holes cut near the tip are recommended for gastric lavage (Fig. 45-2) . There are no convincing data on humans to refute or support this recommendation, and 1 study of a small number of dogs failed to show any difference in efficacy with lavage through a 32 Fr tube compared with a 16 Fr lavage tube. [52] It is generally held that large-diameter nasogastric or orogastric tubes (>1 cm) are more likely to retrieve particulate matter successfully. Also, smaller, more flexible tubes may kink and are significantly more difficult to pass. [53] A 32 to 50 Fr tube is usually recommended for adults. A 28 Fr tube is the size of a pediatric endoscope and may be passed in an infant; proportionately larger tubes should be used for larger children.

Before passage, the length of the tube required to enter the stomach should be estimated by approximating the distance from the nose to the mid-epigastrium (Fig. 45-3) ; premeasurement avoids the curling and kinking of excess hose in the stomach. In addition, passage of an excessive length of hose may cause gastric distention, bruising, and perforation. Passage of an insufficient length of hose may result in lavage of the esophagus and the increased risk for emesis and aspiration. One study of radiographs done to document proper placement of the lavage tube in children found that 7 of 14 patients had improper tube placement (too high or too low), despite positive gastric auscultation in all cases. [54]

Commercial lavage systems are available and often use either a gravity fill-and-empty system with a Y connector (e.g., Travenol or Ethox) or a closed irrigation syringe system. Alternatively, an irrigation syringe can be used for intermittent lavage fluid input and withdrawal. Closed systems minimize the potential for creating a mess and allow 1-person operation, but may permit excessive gastric filling if the operator is not attentive.

### Technique

If gastric lavage is considered essential in the highly anxious or agitated patient, rapid-sequence induction and intubation should be considered. Alternatively, small doses of a benzodiazepine agent (e.g., 1 to 2 mg midazolam IV) may be considered. The procedure should proceed deliberately without significant patient resistance. *The procedure is intended to be therapeutic, not punitive.*

The gastric tube should be passed gently to avoid damage to the nose or the posterior

pharynx. It may be passed through either the nose or the mouth, although tubes of 36 Fr or larger should be passed orally to avoid nasal mucosal or turbinate injury. Passage through the mouth is generally more comfortable for the patient, but the inserter is in danger of being bitten when this technique is used. In addition, orogastric tubes tend to be chewed and occluded by stuporous or combative patients. These problems may be minimized by the concomitant use of a bite block or an oral airway. Elliptically shaped endoscopy bite blocks are recommended.

Nasogastric intubation with larger diameter tubes can be carried out more easily if lubricating or local anesthetic jelly is used; shrinkage of the nasal mucosa with phenylephrine spray (Neo-Synephrine) is advised. Passage of the tube into the esophagus once the pharynx has been entered can be facilitated by putting the patient's chin on the chest. Cough, stridor, or cyanosis indicates that the tube has entered the trachea; the tube should be withdrawn immediately and passage reattempted. Once the tube is passed, its intragastric location should be confirmed by auscultation of the stomach during injection of air with a 50-mL syringe and aspiration of gastric contents. If this step is omitted, the physician may occasionally irrigate the esophagus with a tube that has doubled back on itself during passage or, worse, lavage the patient's lungs.

Before gastric irrigation, a large fraction of the gastric contents can be removed by careful gastric aspiration with repeated repositioning of the tube tip. Only after the stomach has been thoroughly "vacuumed" should gastric lavage be carried out. Indeed, some believe that aspiration of gastric contents may be the most effective part of the procedure. After gastric aspiration, the tube should be taped in place.

With the Y connector closed system (Fig. 45-4) , lavage is performed by clamping the drainage arm of the Y adapter and infusing aliquots of fluid into the stomach from a reservoir. The reservoir arm of the Y is then clamped, and the drainage arm is opened to permit gravity drainage of the stomach contents. The procedure is then repeated. Some resistance is produced by the Y connector and tubing. Suction can be applied intermittently to the drainage tubing to enhance stomach emptying. Rudolph has described a device that performs this task automatically. [55]

Generally lavage can be performed adequately with tap water. However, since severe dilutional hyponatremia has occurred in children lavaged with tap water, physiologic saline is generally recommended for children. [56] Ideally the solution should be prewarmed (45 °C) to minimize patient cooling. Warmed lavage fluid also increases the solubility of most substances, delays gastric emptying, and theoretically should increase the effectiveness of the procedure. Small aliquots of lavage solution should be repeatedly introduced into the stomach and removed. Empiric recommendations for the volume of each aliquot range from 150 to 300 mL in adults and 10 to 15 mL/kg, or 50 to 100 mL, in children.Larger amounts may increase the risk that the gastric contents will be washed into the duodenum, and much smaller amounts are not clinically practical because of the dead space in the tubing (approximately 50 mL in the 36 Fr hose) and the increase in time that is required. The amount of fluid that is returned should approximate the amount that is introduced.

The fluid should flow in freely and drain easily by gravity. If this does not occur, the tube

is usually malpositioned or kinked in the stomach and should be withdrawn or advanced a few centimeters. Manual agitation of the patient's stomach before removal of each aliquot may increase recovery and is recommended. [58] This is done by gently "kneading" the stomach with a hand placed on the abdominal wall. The optimal total volume of lavage solution is unknown. A common recommendation is a minimum volume of 1 to 2 L additional fluid *after* the return becomes grossly clear.

Rarely, special lavage solutions may be used, but the clinical efficacy of these lavage solutions is generally unproven. Soluble starch such as cornstarch or milk may be used after iodine ingestion. The starch converts iodine to less toxic iodide. Calcium salts, milk, or magnesium-containing antacids may be used after fluoride or oxalic acid ingestion. These salts form an insoluble precipitant with the ingested agent. However, in the vast majority of cases where lavage is indicated, the procedure should be rapidly performed with normal saline or tap water rather than delaying gastric lavage while awaiting specialized lavage solutions that have not been clinically proven. The use of a "neutralizing" solution in strong acid or base exposures remains controversial, but the heat of neutralization in vivo appears minimal. [63]

After gastric aspiration and lavage have been completed, a slurry of activated charcoal should be administered through the gastric tube (Fig. 45-5) . When no longer needed, the gastric tube should be pinched or clamped during its removal to avoid "dribbling" fluid into the airway. With the increasing use of repetitive doses of activated charcoal, the gastric tube is often left in place after the lavage procedure is completed. However, because this large tube is irritating and may predispose the patient to gagging, drooling, or aspiration, it should be removed. The alert patient should take subsequent doses orally (see Activated Charcoal below). The patient who remains obtunded may receive additional doses via a standard nasogastric tube, although the high viscosity of the charcoal makes this route challenging. If endotracheal intubation was required prior to lavage tube placement, the endotracheal tube should *not* be removed until the patient is clearly awake and able to control his or her airway, since emesis is common after the procedure.

## Complications

The complications associated with gastric lavage can be subdivided broadly into those associated with the placement of the tube and those resulting from the lavage fluid itself.

Because gastric intubation is a blind procedure that may be required in uncooperative or obtunded patients, some complications are inevitable, but acceptable where clinical benefit is anticipated. If the nasal route is selected, care must be exercised not to damage the delicate nasal turbinates. During tube advancement, injury to the pharynx, esophagus, or stomach rarely may occur. This risk is of most concern in cases in which substantial damage has already been done to these tissues as with caustic ingestions. Preexisting esophageal stricture or previous gastric bypass surgery also makes tube passage more hazardous. [69] Once the tube has been placed, it is important to make sure that it is in the stomach and not in the lungs, as inadvertent placement of the tube in the lungs can be fatal. [70]

Gastric lavage has also been associated with changes in cardiorespiratory function. The patient's pulse rate may rise and oxygen saturation may fall. Thompson and colleagues found that 41% of overdose patients had electrocardiographic changes, and 29% had a fall in oxygen tension to 60 torr or less during lavage. [71] Electrocardiographic changes consisted of atrial or ventricular ectopy in 36% of patients and transient ST segment elevation in 4.8% of patients. [71] Patients at greatest risk for these findings were the elderly, smokers, and those with underlying lung disease. These changes may be more marked in patients ingesting cyclic antidepressants. [72]

The lavage fluid itself is a potential but generally inconsequential source of problems. The large amount of fluid used during a lavage procedure has the potential to produce fluid and electrolyte disturbances in the patient; these imbalances have been reported with both hypertonic and hypotonic [56] lavage fluids. These problems appear to be encountered most commonly in children; adults seem to be more resistant to lavage-induced electrolyte disturbances. [56] Rudolph, however, was unable to document electrolyte changes in 60 patients, age 2 to 94 years, lavaged with tap water. [55] Hypothermia is another possible complication of gastric lavage, leading many pediatricians to warm the lavage fluid after the first liter.

The use of large aliquots of lavage fluid (>300 mL per wash in adults or 10 mL/kg in children) has the potential to force the gastric contents through the pylorus and into the upper small intestine, where more rapid absorption may take place.

Pulmonary aspiration of gastric contents or lavage fluid is the primary potential risk during lavage, although if small aliquots are used and the patient is positioned properly, this risk should be low. One group reported a 10% incidence of aspiration pneumonia in lavaged patients who underwent careful follow-up. [19] Most of the patients with aspiration pneumonia had been intubated prior to gastric lavage to protect the airway. It is generally believed that both emesis and gastric lavage increase the incidence of pulmonary radiographic changes in patients who have ingested petroleum distillates or turpentine. [75] One case of legionnaires' disease has been attributed to gastric lavage with contaminated tap water. [76]

Occasionally it may be difficult to remove a lavage tube once the decontamination procedure is finished. If the tube is not easily withdrawn, it is unwise to use excessive force, because significant gastric or esophageal injury may occur. Kinking or knotting of the tube can occur, but occasionally a tube may be stuck because of lower esophageal spasm. If fluoroscopy or a contrast study fails to demonstrate tube deformation as the cause of impaction, one can administer glucagon (1 to 2 mg IV) in an attempt to relieve spasm of the lower esophageal sphincter. [77] If a knot or kink is demonstrated, surgical removal is often required to avoid esophageal or gastric rupture due to forceful withdrawal of the tube.

## ACTIVATED CHARCOAL

## Background

Charcoal is a pyrolyzed, powdered carbon product. The term *activated* refers to oxidation, which greatly increases the surface area of the charcoal and converts molecules on the surface to chemical moieties that have the capacity to adsorb a wide variety of substances. Currently available products have surface areas ranging from 850 to 2000 m [2] /g. [20] Gram for gram, preparations with a high surface area (e.g., Charcoaid, Requa) are theoretically superior becaue they adsorb more toxin than similar amounts of lower-surface-area products. [77A] The use of activated charcoal in the emergency department has consistently increased since the 1980s; in 1995, 29.4% of poisoned patients treated at health care facilities received activated charcoal. [1]

Activated charcoal may be beneficial in 2 ways. First, it may bind drug or toxin present in the GI tract and make it unavailable for absorption by the body. Unlike ipecac and gastric lavage, activated charcoal may act on drug that has passed the pylorus by "catching up" to drug further along in the GI tract. Second, activated charcoal may increase clearance of drug already absorbed into systemic circulation. For some drugs such as digitoxin, this increase in serum clearance occurs when the drug is secreted in the bile and thus comes into contact with activated charcoal in the bowel prior to reabsorption, interrupting enterohepatic circulation. [78] For most drugs, the increase in clearance is thought to be a result of passive diffusion along a concentration gradient from the circulation into the gut, where it is bound to activated charcoal. Because of the mechanism of diffusion and the large surface area of the small bowel, this mechanism is known as *gut dialysis.*

## Indications

The advantages of activated charcoal are that it is easy to administer, it is effective, and significant complications are rare. Disadvantages include problems with palatability, constipation, nausea, and emesis. Emesis with resultant pulmonary aspiration of gastric contents is a potentially significant complication.

Activated charcoal is indicated if the clinician estimates that a clinically significant fraction of the ingested substance remains in the GI tract and further absorption may result in clinical deterioration. It is also indicated if there is a reasonable expectation that it will result in increased clearance of an already absorbed drug and thereby be of clinical benefit. Studies have shown the ability of activated charcoal to decrease drug absorption and/or increase clearance in volunteers.  There are limited data documenting significant clinical benefit from charcoal administration, but in view of the minimal risk, low cost, ease of administration, and potential benefit, activated charcoal should probably be given to most poisoned patients who present to health care facilities for evaluation and treatment.

Activated charcoal is *not* indicated when it is unlikely to adsorb the substance ingested. Metals such as iron and lithium are not well adsorbed to activated charcoal. For many metals, definitive adsorption data do not exist. However, some metallic salts such as mercuric chloride are well adsorbed by activated charcoal. If data regarding adsorption

are inconclusive or if there is a suspicion of coingestion, activated charcoal should be given, provided there are no contraindications.

Acids and bases are not well adsorbed by activated charcoal. In addition, charcoal may precipitate vomiting, as well as obscure the endoscopist's view, and therefore it should not be given in these cases. Hydrocarbons are not

well adsorbed by activated charcoal, and it should not be used in cases of a pure aliphatic petroleum distillate ingestion. Many hydrocarbons are potential systemic toxins (e.g., carbon tetrachloride, benzene) or are mixed with other potentially significant toxins such as pesticides. In these cases, data are lacking, and charcoal administration is probably appropriate. In some cases, charcoal affinity for a toxic compound may be relatively poor, as with cyanide. However, animal data demonstrate the utility of activated charcoal in cyanide ingestion, even though the in vitro affinity is relatively poor. [81]

The use of activated charcoal in the treatment of acetaminophen ingestion presents a special problem since the antidote, *N*-acetylcysteine, is administered orally in the United States. Although activated charcoal could potentially prevent acetaminophen from reaching toxic concentrations in the blood, there are concerns that charcoal may also adsorb significant amounts of the antidote as well. Pharmacokinetic studies evaluating the effect of activated charcoal on *N*-acetylcysteine absorption have produced conflicting results.  However, based on these data, some authors recommend increasing the *N*-acetylcysteine dose or considering lavaging charcoal from the stomach if *N*-acetylcysteine is to be given within 1 hour of activated charcoal administration. Other authors speculate that the standard *N*-acetylcysteine dose currently in use is much larger than needed to treat the vast majority of acetaminophen overdoses, and therefore no adjustment is required in this scenario. [39] Each of these opinions is based on the same data, and pending more conclusive studies, more definitive recommendations cannot be made. [87]

Multiple doses of activated charcoal (MDAC) may be indicated to increase systemic clearance of a drug that has already been absorbed or to maximize binding of a substance present in large amounts. MDAC may also be indicated when the physical form of the toxic substance might preclude maximal or prolonged contact with a single dose of activated charcoal (e.g., drug packets, plant or mushroom ingestion, extended-release preparations). Table 45-1 (Table Not Available) is a list of some drugs for which MDAC may be of benefit. For some compounds, such as theophylline, MDAC may significantly increase clearance, decrease drug half-life, and potentially avoid the need for other extracorporeal drug removal techniques such as hemodialysis and hemoperfusion.Despite volunteer and animal data showing increased drug clearance during MDAC therapy, no controlled clinical studies demonstrating a beneficial impact on clinical outcome from MDAC have been published.

MDAC is contraindicated if there is evidence of bowel obstruction or ileus. In addition, vomiting may expose the patient to an increased risk of pulmonary aspiration if the stomach is distended with activated charcoal. Another risk of MDAC therapy is the potential for exposure to multiple doses of cathartics. Repeated doses of activated charcoal with sorbitol have been associated with hypernatremia and dehydration.

Repeated doses of magnesium cathartics have been associated with hypermagnesemia, neuromuscular weakness, and coma. When MDAC therapy is being considered, the initial dose may contain a cathartic, but subsequent doses should be the aqueous formulation. Some clinicians administer activated charcoal with cathartic until the first charcoal stool, then switch to aqueous activated charcoal. Either approach is satisfactory.

## Technique

Activated charcoal may be given orally if the patient is awake and cooperative or by nasogastric tube if the patient is uncooperative or unconscious. It may also be given through the lavage tube after gastric lavage. In many formulations the contents settle with time, and vigorous shaking prior to administration is recommended. [95] Rinsing the container with a small amount of tap water and giving this slurry to the patient will allow administration of the full dose to the patient. Aqueous activated charcoal has no taste but does have a gritty texture that most patients find unpleasant. Formulations premixed with sorbitol have a sweet taste and smoother texture. Many attempts have been made to improve the taste and texture of charcoal. Mixing activated charcoal with melted chocolate, chocolate syrup, or ice cream has been shown to increase palatability in children. However, mixing with these additives decreases the adsorptive capacity of activated charcoal to some extent. [97]

Other methods of administering activated charcoal to reluctant patients include using a cup with a lid and a straw or simply clipping the tip of the spout on the lid off and placing a straw in the opening. Manufacturers continue to work with flavorings and texturizers to improve patient acceptance. Calvert and coworkers described 50 children presenting to an emergency department after ingestion who were given slurries of activated charcoal in paper cups. [98] Children were asked in a "firm but kindly manner" to drink the charcoal and were told that it would make them feel better and not sick and that it would not taste bad. Most (86%) readily drank the slurry, and the majority of these drank the entire dose.

If the patient refuses to drink activated charcoal, another option is administration of the dose by nasogastric tube after confirmation of tube placement. Remember: Any gastric decontamination procedure loses efficacy with time, so if the decision is made to give activated charcoal, the patient should be given a brief (5- to 10-minute) period of time to drink the dose. If the dose is not consumed by that time, the clinician should consider giving the dose by nasogastric tube.

## Dose and Concurrent Cathartic Use

The older literature suggests that a charcoal-to-toxin ratio of at least 10:1 is desirable. However, this ratio has not been systematically evaluated for many compounds, and the amount of toxin ingested is often unknown. However, the optimal dose of charcoal is the maximum amount that can be safely given based on estimated stomach volume. Most

authors recommend 50 to 100 g in adults and 1 g/kg in children under 5 years. Palatnick suggests that dosing charcoal in children on a "per-kilogram" basis may lead to

inadequate dosing and recommends a total dose of 25 to 50 g in children under 5 years. [101] Since activated charcoal is usually packaged in containers containing 25, 30, or 50 g, doses in multiples of these units are most practical.

Coadministration of a cathartic with activated charcoal remains a controversial but common practice. [102] Cathartics are given with the intention of preventing activated charcoal-induced constipation and decreasing GI transit time of the activated charcoal-drug complex. Theoretically, this will minimize the chance of continued drug absorption in the gut, as well as the possibility of desorption of drug from activated charcoal. However, the majority of data suggest minimal or nondetectable clinical benefits on drug absorption from cathartic use. There is little evidence that a single dose of aqueous activated charcoal is significantly constipating; however, cathartics are often given for this potential problem. The most commonly available cathartics are sorbitol, sodium sulfate, magnesium citrate, and magnesium sulfate. Sorbitol is most effective in decreasing GI transit time [40] but is also associated with nausea, GI cramping, and abdominal pain. Because cathartics are of unproven benefit and have possible adverse effects, some clinicians avoid their use.

The optimal dose of sorbitol remains to be determined. Dosing studies in healthy volunteers indicate that an initial dose of 60 g of sorbitol with charcoal would best produce reliable catharsis while minimizing adverse effects. [105] Many charcoal formulations come premixed with sorbitol, but there is considerable variation in sorbitol content. Liqui-Char with Sorbitol contains 50 g of activated charcoal in 54 g of sorbitol. Actidose with Sorbitol contains 50 g of activated charcoal in 96 g of sorbitol. Activated charcoal USP with sorbitol contains 25 g of activated charcoal in 27 g of sorbitol. Charcoaid contains 30 g of activated charcoal in 110 g of sorbitol. [20] Thus, there is an almost three-fold variation in sorbitol content between the various formulations. Attention should be paid to the brand of charcoal and the sorbitol content to avoid excessive sorbitol administration.

If aqueous activated charcoal is used and a cathartic is desired as well, a single 50- to 100-mL dose of sorbitol 35% to 70% solution in adults or 2 mL/kg in older (i.e., > 5 years) children can be used. Commonly used doses of the saline cathartics are as follows:

  Magnesium citrate as a 6% solution: 300 mL in adults and 4 mL/kg in children

  Magnesium sulfate as a 10% solution: 15 to 20 g in adults and 250 mg/kg in children

  Sodium sulfate as a 10% solution: 15 to 20 g in adults and 250 mg/kg in children [106]

Multiple doses of magnesium-containing cathartics have been associated with severe hypermagnesemia. Multiple doses of sorbitol have been associated with volume depletion. [92] Children are particularly susceptible to the adverse affects of cathartics, and therefore cathartics should be used with caution, or totally avoided, in this group.

**Multiple Doses of Activated Charcoal**

MDAC may be useful in select cases. It may be used to prevent continued absorption if there is a large amount of drug present, if drug is dispersed throughout the GI tract, or if the drug is in the form of extended-release or enteric-coated preparations. MDAC maximizes the possibility that the majority of drug remains in contact with charcoal during transit through the gut. MDAC may also increase clearance of drug that has already been absorbed by interrupting either enterohepatic circulation or the continuous diffusion and reabsorption of drug from the circulation to the gut (so-called *entero-entero circulation*). Table 45-1 (Table Not Available) is a list of drugs for which MDAC is thought to be of benefit. For most of these drugs, data are sparse, and clinical benefit remains to be proven. For some drugs such as theophylline and phenobarbital, clearance is well documented to be increased by MDAC. Despite increases in drug clearance, clinical benefits of MDAC have not been demonstrated. For example, Pond and colleagues described 10 patients comatose from phenobarbital overdose who were randomized to receive either single activated charcoal or MDAC therapy. Although the serum half-life of phenobarbital was significantly shorter in MDAC patients, there was no difference between groups with regard to duration of intubation or time in the hospital.

When using MDAC therapy, the first dose is 50 to 100 g of charcoal, usually with cathartic. Subsequent dosing is not standardized, and doses range from 12.5 to 50 g without cathartic every 2 to 6 hours. Charcoal therapy should continue until there is clinical improvement and plasma drug levels have fallen to acceptable values.

Emesis with activated charcoal therapy may be a problem, particularly when using MDAC therapy for poisonings by drugs that cause nausea and vomiting (e.g., theophylline). Some clinicians have reported fewer problems with vomiting if a continuous nasogastric infusion of activated charcoal is used. [109] Standard antiemetic therapy such as prochlorperazine or promethazine IV may be tried. [110] Some authors have reported success in controlling severe drug-induced vomiting with ondansetron. If there is evidence of ileus or obstruction, charcoal should be stopped and the nasogastric tube placed on low intermittent suction.

**Complications**

Considering the frequency of its use, the number of complications from activated charcoal is relatively small. The most significant complication of activated charcoal use is pulmonary aspiration. Since the overdose population as a whole is at risk for sedation and loss of airway protective reflexes, the instillation of 8 to 16 oz of liquid activated charcoal into the stomach may put these patients at risk for aspiration. Charcoal is inert, but mixture of charcoal with acidic gastric contents may cause aspiration pneumonitis. [113]

The charcoal slurry is viscous, and its aspiration has been associated with tracheobronchial obstruction. [114] Activated charcoal has also been directly instilled into the lung inadvertently through a nasogastric tube. [115] Patients who have been traumatically lavaged and have sustained esophageal or gastric perforation have been

noted to have charcoal hemothorax and charcoal peritoneum, respectively.

Other residual effects after activated charcoal aspiration include bronchiolitis obliterans and charcoal empyema.  One immunocompromised patient who received MDAC therapy vomited, aspirated the vomitus, and developed pneumonia with *Aspergillus niger*, *Paecilomyces variotii,* and

*Penicillium* spp; interestingly, *Aspergillus* and *Paecilomyces* were grown from activated charcoal in the hospital pharmacy. [120]

Activated charcoal is also reported to cause constipation. [121] The frequency of this problem in the overdose population receiving a single dose of activated charcoal is unknown. However, GI obstruction and perforation have been reported in patients receiving MDAC therapy, particularly in conjunction with ingestion of drugs with anticholinergic properties. [122] As charcoal passes through the gut and water is resorbed, charcoal may harden to form "briquettes." An activated charcoal stercolith with bowel perforation and a rectal ulcer have been reported.

Finally, corneal abrasions have been noted when charcoal has inadvertently come in contact with the eye in comatose or combative patients.

## WHOLE-BOWEL IRRIGATION

### Background

WBI is the newest method of GI decontamination. It involves the oral administration of polyethylene glycol (PEG)-balanced electrolyte lavage solutions (i.e., GoLYTELY or Colyte) in a sufficient amount and at a sufficient rate to physically wash ingested substances through the GI tract. These lavage solutions are traditionally used to prepare the bowel for surgical, radiologic, or endoscopic procedures and have been specifically formulated to minimize fluid or electrolyte shifts across the gut wall. [126] Other lavage solutions, such as normal saline and lactated Ringer's solution, are not recommended. WBI may be performed in children and has been reported after iron ingestion in an 11-month-old. [27] WBI has also been used without adverse effects after iron ingestion during pregnancy. [127]

### Indications and Contraindications

Tenenbein has suggested that WBI be considered for 4 indications: ingestion of a large amount of a toxic substance, late presentation after ingestion of a toxin, ingestion of delayed-release preparations, and overdoses of iron and other substances not adsorbed by activated charcoal. [128] Current widely accepted indications for WBI include ingestion of sustained-release products and iron and lithium ingestion, as well as in body packers and stuffers. The value of WBI for large ingestions or delayed presentations is unknown.

Contraindications to WBI are few and include GI ileus, obstruction, or perforation.

Significant GI bleeding has also been suggested as a contraindication. Adverse effects are usually limited to nausea, vomiting, bloating, abdominal discomfort, and anal itching. However, 1 study found that most volunteers preferred WBI to activated charcoal with sorbitol because of the sorbitol-induced cramps and diarrhea. [32] Proposed therapies for ingestion of several drugs, including extended-release theophylline, aspirin, and cocaine packets, include both WBI and MDAC therapy, since in vitro data suggest that PEG-balanced electrolyte lavage solution can cause desorption of these drugs from activated charcoal,  but the clinical significance of this finding is unknown.

A special consideration after overdose is the possibility that semisolid masses of drug (pharmacobezoars) can form in the stomach and remain there for long periods. Table 45-2 lists drugs that have been associated with bezoar or concretion formation. Drug bezoars or concretions should be suspected when plasma drug concentrations continue to rise or if clinical deterioration occurs, despite customary GI decontamination procedures, especially in cases involving ingestion of medications known to cause drug bezoars. The best approach to the patient with a suspected pharmacobezoar remains to be determined. MDAC therapy, gastric lavage with gastric massage, and WBI are all reasonable initial managements.  In cases where the initial management strategy is failing or the diagnosis remains in doubt, endoscopy can be performed. The advantage of endoscopy is that the stomach contents can be directly visualized and an attempt can be made to break up the drug mass with the endoscope. In rare cases involving iron, gastrotomy has been used to remove drug bezoars. [135]

**Technique**

Once the decision has been made to use WBI, the solution is prepared by mixing the PEG electrolyte powder with water to a volume of 4 L. It is then administered to the patient at a rate of 1 to 2 L/hour to adults and adolescents. Children may receive WBI solution at a rate of up to 40 mL/kg/hour. [136]

Cooperative patients with intact airway protective reflexes may drink this quantity, although taste and volume may be limiting factors. Patients who are unable or unwilling to drink at this rate may receive the solution through a small-bore nasogastric tube after tube placement is confirmed. Unconscious patients with protected airways may receive WBI. With more rapid rates of infusion, vomiting may become a problem. Emesis during WBI usually responds to a temporary decrease in rate of infusion. In these cases, after an arbitrary time of 15 to 30 minutes without emesis, the rate should be slowly increased to a goal of 2 L/hour in adults. Metoclopramide has been suggested as an antiemetic in this circumstance because of its additional prokinetic effects on the GI tract; however, controlled studies have failed to show a benefit in decreasing transit time of a marker compared to WBI alone. [137]

The temperature of the fluid should probably be between ambient and body temperature. One study using chilled PEG electrolyte solution noted that subjects were shivering and uncomfortable. [138] Pediatric patients might be at risk for hypothermia in these conditions. On the other hand, there is the potential for increased drug solubility and thus increased absorption in a warmed solvent. Thus, a compromise would be to

use a solution at a temperature tolerated by the patient but not above body temperature.

| TABLE 45-2 -- Examples of Drugs Associated with Pharmacobezoar Formation | |
| --- | --- |
| Barbiturates | Extended-release lithium |
| Bromides | Extended-release verapamil |
| Ethchlorvynol | Enteric-coated aspirin |
| Carbamazepine | Iron |
| Glutethimide | Meprobamate |
| Extended-release theophylline | |

Once WBI is begun, the awake patient may be seated on a commode if feasible. Comatose patients may have a rectal tube inserted as stool becomes liquid to accommodate the large volume of rectal effluent.

The end point of WBI is controversial. When the ingested substance is radiopaque, as with iron, radiographs may be obtained to follow progress and document decontamination. Most ingested substances are not radiopaque, however, and end points are more arbitrary. Suggested end points include the appearance of clear rectal effluent or total time of WBI, usually 4 to 6 hours. However, case reports have documented passage of tablets, drug packets, or plant parts after clear rectal effluent. Hence, existing data do not provide a clear end point for WBI. Clear rectal effluent appears to be a minimum end point, and one may argue for an additional arbitrary amount after clear rectal effluent. [137] If the clinical or laboratory evidence points to continued drug absorption, WBI should be continued until the patient has stabilized.

Patients who ingest packets of drugs, usually cocaine or heroin, especially wrapped for smuggling are called *body packers*. In contrast, *body stuffers* may hastily ingest drugs that may be poorly wrapped in an attempt to avoid arrest. Both types of patients should

be placed on a cardiac monitor, and an IV line should be established. Fifty grams of activated charcoal should be given. If the patient is asymptomatic from the ingestion, WBI should be started (Fig. 45-6 *A* and *B*). Body packers usually know the exact number of packets ingested, and these may be counted in the commode. Up to 20 L of PEG electrolyte solution have been used in the decontamination of body packers. [30] After WBI is finished, efficacy of evacuation may be confirmed by a contrast radiologic study or a plain radiograph if the packets are radiopaque. Because of the potentially fatal amount of drug contained in each packet, symptomatic patients should be considered for emergent surgical removal of packets. Body stuffers usually ingest smaller quantities of drug, but it may be unwrapped or poorly wrapped, and there is a greater likelihood of absorption. Management of these patients should consist of activated charcoal and WBI. If the ingested substance can realistically be detected by contrast radiologic study (crack vials), these studies should be performed. Since the ingested material is often of a smaller quantity and dispersed through the GI tract, surgical intervention is unlikely to be indicated, although the amount, form, and type of drug ingested should be considered in each case.

### Complications

There have been few reported complications from WBI therapy. The procedure may cause bloating, nausea, vomiting and abdominal discomfort, and anal itching. [39] [138] Vomiting may respond to a decrease in the rate of administration or to antiemetics. [128] As with all other methods of GI decontamination, attention should be directed to the airway and the potential for aspiration. Administration of a large amount of chilled or room temperature WBI fluid to pediatric patients could potentially cause hypothermia, and therefore warmed fluid should be considered in these patients. Mild asymptomatic hypoglycemia (61 mg/dL) occurred after 5 hours of WBI in a 11/2-year-old child. [136] It may be prudent to check the serum glucose level in children or other individuals at risk for hypoglycemia if prolonged WBI is required.

## DERMAL DECONTAMINATION

### Indications and Contraindications

Decontamination of the skin is indicated after a cutaneous exposure (skin, eyes, mucosa, open wounds) to potentially hazardous materials. A dermally contaminated patient is managed like any other patient, except health care personnel must also protect themselves and others from the danger of secondary contamination. Dermal decontamination of most hazardous materials has not been systematically evaluated. In addition, the hazardous material may not be identified or confirmed prior to patient arrival.

Decontamination is contraindicated if hospital personnel cannot adequately protect themselves from exposure and health risk. Every emergency department should have a disaster plan with contingencies for hazardous materials incidents. A variety of forms of chemical protective clothing should be available. Life-threatening issues should be promptly addressed before decontamination takes place. If the patient arrives at the emergency department after treatment and decontamination by out-of-hospital

personnel, further decontamination may not be needed. In these cases, the patient should be evaluated by the triage officer on arrival to the health care facility for assessment of clinical stability and adequacy of decontamination. Some substances, such as radioactive materials, pose a significant risk of contaminating the health care facility and hospital personnel. In these cases, provisions should be made for containment of the decontamination area.

**Technique**

Cutaneous exposures to hazardous materials pose several problems that must be managed simultaneously by the emergency physician. Continued exposure to the hazardous material must be prevented, and dermal and systemic manifestations of the specific toxic exposure must be anticipated and treated. In addition, the physician must also prevent contamination of health care workers and of the emergency department and the hospital. Guidelines for managing hazardous materials incidents are available from the Emergency Response and Consultation Branch (E57), Division of Health Assessment and Consultation, Agency for Toxic Substances and Disease Registry, 1600 Clifton Road, N.E., Atlanta, GA 30333. [4]

Emergency departments should have preexisting hazardous materials incident protocols that designate the decontamination area and the triage and decontamination team. The decontamination area must meet several qualifications. First, it should be secured to prevent spread to other areas of the hospital. Second, the ventilation system should be separate from the rest of the hospital or it should be shut off to prevent airborne spread of contaminants. Third, provisions must be made to collect the rinsate from contaminated patients to prevent contamination of the facility and water supply. Some hospitals may have designated decontamination rooms. In most facilities, the best place to begin initial treatment and evaluation is outside, weather permitting. Portable decontamination facilities are available, but their cost may be prohibitive for many institutions. A practical alternative is to have a warm shower nozzle, soap, and a wading pool available outside the entrance to the emergency department. A tent or screen can provide privacy. Stable patients may be able to decontaminate themselves by disrobing and discarding clothing and jewelry (including watches) and using soap and water as directed by hospital personnel.

Health care workers can protect themselves in most circumstances with hospital gowns, shoe covers, masks, eye protection, and gloves. Intermediate levels of chemical protective clothing can be purchased and stored for use as needed. If it is anticipated that the facility will receive heavily contaminated patients, specialized personal protective equipment may be needed. This level of protective equipment carries some expense and requires training prior to use.

Care should be taken to prevent unnecessary contamination outside the decontamination area. The floor should be covered with plastic or paper that is taped in place. Doorknobs, light switches, and cabinet handles should be covered. As patients or health care personnel leave the decontamination area, protective clothing and contaminated articles should be left behind for appropriate disposal.

Actual skin decontamination should be started as soon as possible. Decontamination with soap and water should not be delayed while awaiting specialized decontamination fluids. Starting from the head and working down, the patient should be washed with warm tap water, with special attention paid to skin folds, which may retain the contaminant despite thorough rinsing. A mild soap, such as dish soap, should be used. Since mucosal surfaces and open wounds may potentially absorb toxic materials faster than intact skin, these areas should be cleaned first. Once wounds are decontaminated, they should be covered with a waterproof occlusive dressing to avoid recontamination. Contaminated nares or ears should be gently irrigated. Anything that abrades the skin or causes vasodilation, such as stiff scrub brushes, hot water, or vigorous scrubbing, should be avoided, as this may potentially increase toxin absorption. A commonly recommended minimum time for decontamination is 15 minutes, although this appears to have no scientific basis. Exposure to caustics such as strong acids or alkalis may require irrigation over extended periods of time. All runoff fluid should be collected for disposal. Decontamination of the eye is discussed in Chapter 67 .

## CONCLUSION

Gastric emptying techniques such as gastric lavage and ipecac-induced emesis are being used less frequently and for a more narrowly defined patient population. Concurrently, activated charcoal as the sole means of gastric decontamination is increasing in popularity. The evaluation and treatment of asymptomatic or minimally symptomatic patients who have not ingested significantly toxic substances continue to evolve, and it has been suggested that these patients will do well regardless of therapy. The major problem currently facing the clinician is the choice of GI decontamination in the significantly poisoned patient. Studies evaluating this subpopulation are few and usually involve small numbers of patients. Thus, the choice of decontamination method for these patients must be individualized using clinical acumen and common sense. Although the risks associated with properly conducted GI decontamination appear slight, they are

measurable, and no patient should undergo any of the available procedures unless it is anticipated that decontamination will provide clinical benefit.

# Chapter 46 - Peritoneal Procedures

**John A. Marx M.D.**

Paracentesis and diagnostic peritoneal lavage constitute the 2 primary intraperitoneal procedures. They are fundamentally similar in purpose and design. However, the former is generally reserved for medical concerns and the latter for traumatic pathology.

## DIAGNOSTIC PERITONEAL LAVAGE

Root and colleagues introduced diagnostic peritoneal lavage in 1964. [1] It has withstood the passage of more than 3 decades and remains a mainstay in the management of penetrating torso trauma. Following a blunt mechanism of injury, its greatest utility is as a triage tool in the hemodynamically unstable multiply injured patient. Otherwise, it serves with computed tomography (CT) and ultrasound (US) in the diagnostic armamentarium of blunt trauma.

While commonly referred to as *diagnostic peritoneal lavage* (DPL), this procedure comprises 2 distinct components: peritoneal aspiration and peritoneal lavage. In the lavage portion, normal saline is introduced by catheter into the peritoneal cavity, recovered by gravity, and analyzed. Peritoneal aspiration, in which an attempt is made to retrieve free intraperitoneal blood, precedes lavage. A finding of intraperitoneal blood presages intraperitoneal organ injury and precludes the need for subsequent lavage.

Peritoneal lavage can be used as a therapeutic tool in hypothermia and as a means of removing toxins. [2] It has also been used as a diagnostic instrument for suspected intra-abdominal infection and nontraumatic sources of hemorrhage. However, its primary use is as a determinant for the need for laparotomy following trauma, and this chapter focuses on that use.

### Indications

#### Blunt Trauma

Prior to the advent of CT and US, DPL was the sole diagnostic option to physical examination for predicting the need for operative intervention. It was integral both to the reduction of unnecessary laparotomies and to the discovery of unsuspected and life-threatening intra-abdominal hemorrhage in patients with significant closed head injury.

In a number of respected centers in the United States, DPL continues to be the focal diagnostic instrument. It serves 2 primary functions. First, it can rapidly determine or exclude the presence of intraperitoneal hemorrhage (Table 46-1) . Thus, the patient with a critical closed head injury, the unstable motor vehicle crash victim with multiple potential sources of blood loss, or the patient with pelvic fracture and retroperitoneal hemorrhage can be appropriately routed to life-saving laparotomy.  Furthermore, given

its exquisite sensitivity, a negative peritoneal aspiration allows the clinician to proceed to alternative management steps and the patient to forego unnecessary laparotomy. Second, DPL can

| TABLE 46-1 -- Indications for DPL Following Blunt Mechanism | | |
|---|---|---|
| **Purpose** | **Circumstance** | **Alternate or Complementary Diagnostic** |
| Rapidly determine presence of IPH | Hemodynamically unstable multiple blunt trauma | US |
| Determine presence of organ injury | Suspected or known blunt trauma with unreliable examination: | CT |
| |   Head injury with altered mental status | |
| |   Alcohol intoxication | |
| |   Drug intoxication | |
| |   Spinal cord injury | |
| Determine presence of IPH or injury | Multiple trauma patients who require general anesthesia for other injuries | CT, US |

IPH, intraperitoneal hemorrhage; US, ultrasound; CT, computed tomography.

be employed in less exigent circumstances as a means of predicting solid or hollow visceral injury requiring laparotomy.  In this venue, its sensitivity to the presence of hemorrhage may prompt unnecessary laparotomy in a small percentage of patients with self-limited lacerations of the liver or spleen.  CT scan specifically evaluates all intraperitoneal structures as well as the retroperitoneum, a region inaccessible to DPL. Because resolution and the speed with which it can be undertaken have vastly improved, CT has become an invaluable adjunct in the management of blunt trauma. [15]
 It is most useful in the identification of injury to solid organs with accompanying intraperitoneal hemorrhage and greatly assists nonoperative management of those injuries. However, its ability to discern hollow viscus and pancreatic pathology remains inconsistent.  It is when serial clinical evaluations cannot be performed that gut perforation leads to preventable mortality. Therefore, certain authorities recommend the performance of DPL as a complement to CT for this express scenario.

Experience with US in North America is meager in comparison with that in Western Europe (notably Germany) and Asia (notably Japan). In the past, US in the United States had been used exclusively for the detection and serial examination of traumatic pancreatic pseudocysts. There are 2 paradigms that have brought US to the forefront. First, this modality has been considered for use as a triage instrument, similar to DPL, for the detection of intraperitoneal hemorrhage on the basis of identifying which pouches and gutters are fluid filled. Clinical success in this role has been mixed with reported sensitivities for intraperitoneal hemorrhage of 65 to 95%. Additionally, to be useful in this role, a competent technician, interpreter, and equipment must be present in real-time. It has been demonstrated that emergency physicians and surgeons can be trained in this technique to a level of competence sufficient for this need. [19]

Second, US can determine injury to solid viscera such as the liver, spleen, kidneys, and pancreas. This requires considerably greater expertise, and in most centers US has not supplanted CT for this purpose.

DPL is a readily available procedure that can be conducted rapidly in the safe confines of the emergency department. The ability to undertake CT or US in a similar manner requires careful consideration of clinical circumstances, equipment location, and the capabilities of available personnel.

**Penetrating Trauma**

The advent of DPL was seminal in the promotion of selective management for penetrating abdominal injury. Here its role is more dominant than that of either CT or US due to the far greater likelihood of occult injury to hollow viscera and the diaphragm following a penetrating mechanism.

Instruments and missiles may penetrate the abdominal cavity via the anterior abdominal wall, flank, back, or low chest. The intraperitoneal space is vulnerable if penetration occurs as high as the fourth intercostal space anteriorly and the sixth or seventh laterally and posteriorly, as the diaphragm may rise to these levels in the expiratory phase. [23] Indeed, coincident thoracic penetration has occurred in up to 46% of abdominal injuries. The likelihood of retroperitoneal injury increases when the entry site is over the flank or back, but the prospect of intraperitoneal pathology remains considerable with cited incidences of 21 to 44% for the flank and 7 to 14% for the back (Table 46-2) (Table Not Available) .

**Stab wounds.**

Because only one-fourth to one-third of patients who sustain stab wounds to the anterior abdomen require laparotomy, diagnostic algorithms are used to decrease the rate of unnecessary operation. An optimal approach would not sacrifice sensitivity for morbid intraperitoneal injury. A pathway using a combination of clinical mandates, local wound exploration, and DPL is well established (Fig. 46-1) (Figure Not Available) . [30] These clinical mandates are reasonably accurate predictors of significant intraperitoneal injury (Table 46-3) (Table Not Available) . Thus, the presence of one or more mandates suggests the need for urgent laparotomy and precludes the undertaking of other diagnostic studies.

DPL fills 3 roles in the evaluation of patients with abdominal stab wounds (Table 46-4) : (1) rapid determination of the presence of hemoperitoneum, (2) discovery of intraperitoneal injury requiring operation in stable patients, and (3) the establishment of diaphragmatic violation. As is the case in blunt trauma patients, DPL can be invaluable as a rapid triage tool when the source of hemodynamic instability is not known. Pericardial tamponade, intrathoracic hemorrhage, and intraperitoneal hemorrhage may be contributory to hemodynamic instability or wholly causal. Again, as for blunt trauma evaluation, US is the only diagnostic modality for intraperitoneal hemorrhage that is competitive for this role. In the determination of injury following stab wounds, DPL carries 90% accuracy.Serial examinations, CT, and laparoscopy are alternative modalities in specific circumstances and centers. Diaphragmatic rents created by stab wounds are generally small; thus, at the outset, they do not create apparent clinical or radiologic abnormalities.However, morbidity due to delayed herniation of bowel is common and substantive. [40] DPL is currently the most sensitive means of discerning this injury in the immediate post-trauma phase. [31] Physical examination and CT are notoriously insensitive. Laparoscopy has demonstrated promise in experienced hands.

**Gunshot wounds.**

Multiple organ injury is the rule following gunshot wounds, and mortality is significantly greater when compared with that for stab wounds. [41] The diagnostic approach is more conservative for gunshot wounds because the likelihood of intraperitoneal injury requiring operative intervention exceeds 95% when the projectile has entered the intraperitoneal cavity (Fig. 46-2) (Figure Not Available) . If clinical mandates are met (see Table 46-3) (Table Not Available) or if peritoneal violation has occurred, most

centers proceed to laparotomy. [30] DPL is reserved for 2 circumstances: (1) the wound tract is neither obviously superficial nor intraperitoneal, and (2) penetration is to the low chest, where diaphragmatic injury is more likely, yet the possibility of intraperitoneal injury exists.

## Contraindications

Diagnostic peritoneal lavage can be undertaken in virtually any patient irrespective of age, pregnant state, or comorbid illness. Adjustment of the technique and site of performance allows relative contraindications to be overcome. Relative contraindications include prior abdominal surgery or infections, obesity, coagulopathy, and second- or third-trimester pregnancy. The sole absolute contraindication is when clinical mandates for urgent laparotomy already exist.

## Technique

### Preliminary Steps

The stomach and bladder should be decompressed to prevent inadvertent injury. This is most relevant when the semi-open

| **TABLE 46-4** -- Indications for DPL Following Penetrating Mechanism | | |
| --- | --- | --- |
| **Purpose** | **Circumstance** | **Alternate or Complementary Diagnostic** |
| Rapidly determine presence of IPH | Hemodynamically unstable SW or GSW to low chest, abdomen, flank, back | Ultrasound |
| Determine presence of organ injury | SW or GSW to low chest, abdomen, flank, back | Serial clinical examinations, CT, laparoscopy |

| Determine diaphragmatic violation | SW or GSW to low chest, upper abdomen | Laparoscopy |
|---|---|---|
| IPH, intraperitoneal hemorrhage; SW, stab wound; GSW, gunshot wound; CT, computed tomography. | | |

technique is used and a trocar is passed through the peritoneum. DPL should be performed according to compliance with standards for body fluid precautions (see Chapter 75) . Prior to making the skin incisions described below, the site of placement should be prepped with standard skin antiseptics (e.g., povidone-iodine) and appropriately draped. The operator should observe sterile precautions throughout the procedure. Prophylactic antibiotics are not indicated for routine DPL because local and systemic infections are rare. [32] [45]

Local anesthesia (1% lidocaine *with* epinephrine) should be infiltrated liberally into the area for incision and dissection (Fig. 46-3) . It is best to delay the incision for >30 seconds following local anesthetic infiltration to permit local vasospasm, which minimizes wound bleeding during the procedure. Standard equipment for an open peritoneal lavage catheter placement is shown in Figure 46-4 .

**Catheter Placement**

DPL is performed via 2 basic methods: open and closed. The 2 open techniques are *semi-open* and *fully open,* and they typically require an assistant. DPL is clearly within the diagnostic armamentarium of the emergency physician and surgeon. It may be undertaken by either or both in keeping with clinical policies established at the particular trauma center.

**Open technique.**

In the semi-open method, sharp then blunt dissection using a No. 11 scalpel and Army-Navy retractors, respectively, proceeds to the rectus fascia (Fig. 46-5 *A* and *B*). The skin incision should be 4 to 6 cm in length. When the selected site is infraumbilical in the midline, the operator should reach the linea alba; its crossing bands of crural fibers may be apparent. [46] A small 2- to 3-mm opening is then made in the linea alba, preferably with a No. 15 scalpel blade ( Fig. 46-5C ). (The operator will notice a tough, gritty sensation when cutting the linea alba with the scalpel.) Towel clips can be placed through this opening to grasp each side of the rectus fascia (Fig. 46-5 *D*). These 2 towel clips are then lifted to allow safe percutaneous advancement of the catheter via trocar in a 45 to 60° caudad orientation through the peritoneum and into the peritoneal cavity (Fig. 46-5 *E* and *F*).

To decrease the likelihood of penetrating underlying viscera, some operators advocate holding the fingers low on the catheter-trocar instrument such that on entering the abdominal peritoneum, the fingers will prevent deep penetration. Excessive pressure during trocar penetration is a common error. Steady "one-finger pressure" applied to the handle is sufficient to "pop" through the fascia and peritoneum. After controlled peritoneal penetration of 0.5 to 1.0 cm in the midline, the trocar is retracted 1.0 to 2.0 cm within the catheter, and the catheter is carefully advanced toward the pelvis. Some operators prefer to advance the catheter toward the right or left side of the pelvis. Use of a slight twisting motion during advancement may minimize visceral or omental injury.

The *fully open* technique extends the semi-open technique by one step. The opening in the linea alba is lengthened, and the peritoneum is opened to allow direct visualization during catheter placement into the peritoneal cavity.

The 2 open techniques can be accomplished with a single technician, but an additional pair of hands can be helpful for retraction and handling of instruments. The fully open method is the more technically demanding and time consuming. It is reserved for clinical circumstances in which closed or even semi-open technique is not deemed safe or has been attempted and failed. These include pelvic fracture, pregnancy, prior abdominal surgery or infections, and obesity.

**Closed technique.**

With closed techniques, the catheter is introduced into the peritoneal space in a blind percutaneous fashion. [49] Formerly, this was accomplished via trocar. This has been abandoned in favor of the much safer but equally simple Seldinger (guide wire) method, in which a small-gauge guide needle is inserted into the peritoneal cavity in the infraumbilical midline (Fig. 46-6 *A*). A flexible wire is then passed through the needle (Fig. 46-6 *B*), and the needle is removed, allowing the over-the-wire placement of a soft catheter into the peritoneal cavity. A stab with a No. 11 scalpel at the entry site of the wire allows easier passage of the catheter through the abdominal wall (Fig. 46-6 *C* and *D*). Rotating the catheter while pushing it over the guide wire is recommended to facilitate entry into the peritoneal cavity. The catheter is placed as above, into the right or left pelvic gutter.

The wire is then withdrawn, and aspiration conducted, followed by lavage when necessary. The guide wire should always be controlled to avert intra-abdominal migration of the wire. Proponents of the guide wire technique promote its ease and rapidity. Those who prefer the semi-open method argue that the time to peritoneal aspiration, the more critical interval, is minimally different, and that this method may have fewer complications and thus be more accurate than the guide wire technique. Note that time to aspiration for semi-open or closed approaches should entail no more than 2 to 5 minutes.

**Site**

The optimum location for DPL is at the infraumbilical ring, at the inferior border of the umbilicus (Table 46-5) . Here, between the rectus abdominis muscles, there is adherence of the peritoneum and relative lack of vascularity and preperitoneal fat. [46] Closed DPL should always be conducted here. In the event of second- or third-trimester pregnancy, a suprauterine approach is used. If there is midline scarring, a fully open technique at the lateral border of the rectus abdominis in the left lower quadrant may be necessary. The left is preferred to avoid later confusion about whether an appendectomy has been performed. It is interesting to note that Moore and colleagues found no increase in complications or misclassified lavages when the closed technique was used in a small series of patients with prior abdominal surgery. [59] In the presence of a pelvic fracture, a fully open supraumbilical approach is recommended. This greatly decreases the likelihood of passing the catheter through retroperitoneal hematoma that has dissected from the fracture anteriorly and across the abdominal wall. [60] In penetrating trauma, DPL should not be conducted through the stab or missile entry site. Such an approach may contaminate the intraperitoneal cavity and potentially exacerbate abdominal wall bleeding, which could in turn lead to a false-positive result.

**Aspiration and Lavage**

Once the catheter has been placed successfully into the peritoneal cavity and the right-angle adapter, extension tubing, and a non-Luer-Lok syringe have been attached, aspiration is attempted (Fig. 46-7) . The recovery of 10 mL of blood is considered positive, and the procedure terminated. In penetrating trauma, the acquisition of lesser amounts may be meaningful because of the tendency for the diaphragm and bowel to hemorrhage minimally when injured. However, there are no established rules in this regard.

If little to no blood is aspirated, the peritoneal cavity is lavaged with either normal saline or lactated Ringer's solution (Fig. 46-8) . A blood pressure cuff or blood infusion pump can be applied to the plastic intravenous (IV) bag to speed the influx (i.e., decrease lavage time), but is generally not needed. Large-bore infusion tubing (e.g., urologic irrigation tubing sets; Abbott No. 6544 cystoscopy/irrigation set) also shortens fluid influx time. The normal amount is 1 L in adults or 15 mL/kg in children. When possible, the patient is rolled or shifted from side to side after infusion to increase mixing. The IV bag or bottle is placed on the floor (or below abdominal level), and the fluid is allowed to return by gravity.

The fluid may not continue to return because of several factors. Some IV tubing contains a one-way valve; if tubing with a valve was used in error, valveless tubing must be reinserted into the IV bag. Another reason for poor return is inadequate suction. This problem can be corrected by insertion of a needle into the second opening at the bottom of the IV bag or into the head of the IV bottle for aspiration of 10 mL of air. Alternatively, the catheter may be adherent to peritoneum. If so, relieving some of the pressure in the IV bottle or gently wiggling and twisting the catheter as well as applying abdominal pressure may aid flow return.

It is generally accepted that the return of 700 mL in the adult is adequate for

interpretation of findings. However, as little as 10 to 20% of the infusate may give a representative sample for both gross and microscopic determinations. Only 10 mL of fluid from the return need be sent to the laboratory for cell count analysis; another 10 mL can be sent for enzyme analysis (see interpretation below). Some operators prefer to leave the dialysis catheter in place until the returned fluid is analyzed. The physician may wish to re-lavage when the initial results are borderline or an occult bowel perforation is suspected.

| TABLE 46-5 -- Preferred Site of DPL | | |
|---|---|---|
| **Clinical Circumstance** | **Site** | **Method** |
| Standard adult | Infraumbilical midline | C or SO |
| Standard pediatric | Infraumbilical midline | C or SO |
| Second- and third-trimester pregnancy | Suprauterine | FO |
| Midline scarring | Left lower quadrant | FO |
| Pelvic fracture | Supraumbilical | FO |
| Penetrating trauma | Infraumbilical midline [*] | C or SO |
| C, closed; SO, semi-open; FO, fully open. | | |

* The SW or GSW site should be avoided.

## Complications

### Local and Systemic

Local wound complications, including infection, hematoma, and dehiscence, have occurred in only 0.3% of patients in 2 large series. Systemic infection has been described rarely.

### Intraperitoneal

Iatrogenic intraperitoneal injury can be inflicted by the trocar or wire and, rarely, the catheter. Virtually any structure in the peritoneal cavity has been breached, including the small and large bowel, the bladder, and major vessels. Typically, if the needle is the culprit, and even if the trocar is responsible, injury to these structures is minimal and self-limiting, and observation of the patient is sufficient.

### Technical Failure

Inability to recover peritoneal aspirate or lavage fluid can result in a false-negative interpretation. This can occur in several circumstances. It follows unwitting placement of the catheter into the preperitoneal space, which is less likely to occur with either open technique. Compartmentalization of fluid by adhesions or obstructing omentum can impede egress of fluid. Finally, large diaphragmatic tears typical of blunt pathophysiology allow flow of lavage fluid from the intraperitoneal to the thoracic cavity.

False-positive findings can occur in 2 ways. First, iatrogenic misadventure can be responsible. Second, in penetrating trauma, particularly stab wounds, bleeding from the abdominal wall injury site into the peritoneal cavity can lead to positive findings when no injury to intraperitoneal structures has occurred. [32]

## Interpretation

### Gross Blood

The recovery of 10 mL blood via aspiration is considered a positive finding. Lesser-volume aspirates are generally discarded and are not factored into lavage analysis. Grossly bloody aspirates are typically indicative of solid visceral or vascular injury, with a positive predictive value >90%. Aspiration of blood is responsible for approximately 80% of true-positive DPL findings in blunt trauma and for 50% of those following stab wounds.

A positive aspiration in the blunt trauma patient who is hemodynamically stable or has been resuscitated to apparent stability need not mandate urgent operation. Unnecessary laparotomy will occur if there has been minimal and self-limited damage to the liver, spleen, bowel serosa, or mesentery. [15] In this situation, CT and clinical indicators may be used in concert with the DPL findings.

**Red Blood Cell (RBC) Count**

The recommended RBC threshold varies according to mechanism and, in the case of stab wounds, the external site of injury (Table 46-6) . The optimum criterion will deliver excellent sensitivity, a high positive-predictive value, and, therefore, a minimum incidence of unnecessary laparotomy. Negative laparotomy incurs a prolongation of hospitalization and increases the cost of care, in addition to creating the potential for procedural complications.  RBC counts >10$_5$ /mm$_3$ (i.e., >10$_5$ /muL) are generally considered positive with a blunt mechanism or following stab wounds to the anterior abdomen, flank, or back. Counts of 20,000 to 100,000/mm$_3$ should be considered indeterminate.  For stab wounds to the low chest, where the diaphragm is at increased risk of injury, the red blood cell criterion should be lowered to 5000/mm$_3$ to maximize sensitivity for isolated injury to this structure. With gunshot wounds to the abdomen or low chest, the same criterion is applied. This is intended to increase the sensitivity of the test, because intraperitoneal entry by a missile carries a likelihood of intraperitoneal injury of 95%.  An uncomplicated DPL should not create more than several hundred to several thousand RBCs in the peritoneal lavage fluid.

The incidence of false-positive RBC interpretation in the setting of pelvic fracture is considerable. However, aspiration of free blood in the critical pelvic fracture patient predicts active intraperitoneal hemorrhage in >80% of cases. [69] A positive RBC count should generally prompt corroboration or refutation of intraperitoneal injury by CT. In this fashion, needed pelvic angiography and embolization will not be delayed unnecessarily.

**White Blood Cell (WBC) Count**

An inflammatory peritoneal response occurs to a multitude of stimuli, including stool, blood, and enzymes. [70] The WBC count in lavage effluent was formerly touted to predict small bowel injury but has since been proven unreliable. [71] It is insensitive in the immediate postinjury period, as 3 to 5 hours are necessary before the test becomes positive (Table 46-7) (Table Not Available) . Moreover, a positive finding is likely to be falsely so. Therefore, the WBC level in and of itself should not determine the need for laparotomy.

**Enzymes**

Alkaline phosphatase is contained in intramural small bowel as well as in hepatobiliary secretions released into the proximal intestine. Amylase is contained in the latter only. Perforation of small bowel allows access of these 2 markers into the peritoneal cavity where they can be recovered by peritoneal lavage.  While levels of the 2 markers usually rise in

| | Positive (RBCs/mm$^3$) | Indeterminate (RBCs/$^3$) |
|---|---|---|
| **TABLE 46-6** -- Diagnostic Peritoneal Lavage RBC Criteria<br>*From Marx J: Abdominal trauma. In Rosen P, Barkin RM, Braen GR, et al (eds): Emergency Medicine: Concepts and Clinical Practice. 3rd ed, vol 1. St Louis, Mosby-Year Book, 1992, p 476.* | | |
| Blunt trauma | 100,000 | 20,000-100,000 |
| Stab wound | | |
|   Anterior abdomen | 100,000 | 20,000-100,000 |
|   Flank | 100,000 | 20,000-100,000 |
|   Back | 100,000 | 20,000-100,000 |
|   Low chest | 5000 | 1,000-5,000 |
| Gunshot wound | 5000 | 1,000-5,000 |

tandem, lavage amylase has been shown to be a more accurate marker than lavage alkaline phosphatase (see Table 46-7) (Table Not Available) . In contradistinction to the WBC count, these tests will be positive in the immediate postinjury period. However, they may not be economical if used on a mandatory rather than a selective basis. Neither is helpful in discerning the presence of pancreatic pathology.

**Miscellaneous**

Routine bile staining, Gram stain, and microscopy to identify vegetable fibers are rarely productive and are of untested accuracy.

**Summary**

DPL remains an invaluable diagnostic instrument in trauma. It should be used in common-sense fashion. Laboratory parameters are guidelines and should not be embraced to the exclusion of pertinent clinical features. CT, US, or both, can serve in lieu of or in addition to DPL in its various roles. [78] Optimal strategies depend largely on the capability of an institution's resources and personnel in each clinical scenario.

# PARACENTESIS

Ascites connotes an abnormal accumulation of fluid within the peritoneal cavity. The word derives from the Greek *askos,* which means bag or sack. It is a symptom with important diagnostic, therapeutic, and prognostic implications.

Therapeutic abdominal paracentesis is one of the oldest medical procedures, dating to approximately 20 B.C. Paracentesis was first described in modern medical literature by Saloman at the beginning of this century, and it became a valued decompressive therapy. [79] With the advent of diuretics in the early 1950s, paracentesis fell out of favor as a treatment option. Controlled clinical trials in the late 1980s and early 1990s restored its reputation by demonstrating the safety and efficacy of large-volume paracentesis. Because this mode is invasive and consumes physician hours, it is generally reserved for the treatment of patients with chronic ascites who have tense ascites or whose condition is refractory to diuretic therapy. [83] [85] However, paracentesis remains an important diagnostic agent for patients with new-onset ascites or to determine the presence of worrisome conditions, notably infection, in those with preexistent ascites. [86]

## Clinical Features

### Determination of Ascites

Small amounts of ascites may be asymptomatic. Larger collections typically cause a sense of abdominal fullness, anorexia, early satiety, and perhaps nausea and abdominal pain. Considerable accumulations create symptoms of respiratory distress by virtue of restricting lung capacity. [87]

The most predictive history and physical findings for *excluding* the diagnosis of ascites are the absence of ankle swelling and increased abdominal girth and the inability to demonstrate bulging flanks, flank dullness, or shifting dullness.  Positive predictors for

the diagnosis are a positive fluid wave, shifting dullness, or peripheral edema.

Patients who lack obvious clinical markers may benefit from the performance of ultrasonography, which can discern the presence of as little as 100 mL fluid. [92] Additionally, it is a useful adjunct to determine the location of fluid that may be compartmentalized by preexistent infection or surgical adhesions.

**Differential Diagnosis**

The etiologies of ascites can be categorized in several ways. On a structural basis, these are divided into diseases of the peritoneum and diseases not involving the peritoneum. The former group includes infections, neoplasms, collagen vascular diseases, and idiopathic causes. The latter includes cirrhosis, congestive heart failure, nephrotic syndrome, protein-losing enteropathy, malnutrition, myxedema, pancreatic disease, ovarian disease, chylous effusion, Budd-Chiari syndrome, and hepatic venous occlusive disease. Pathophysiologic categories are found in Table 46-8 (Table Not Available) . In this country, parenchymal liver pathology is overwhelmingly the most likely cause and within this group, alcoholic liver disease is responsible for approximately 80% of cases (Table 46-9) (Table Not Available) . Finally, ascites can be classified on the basis of a serum-ascites albumin gradient, that is the difference between albumin values obtained simultaneously from serum and ascites samples (Table 46-10) .

Based on a series of 1500 paracenteses performed in a predominantly inpatient hepatolgy/general internal medicine setting (B.A. Runyon, unpublished observations).

## Indications and Contraindications

Therapeutic paracentesis may be undertaken in the emergency setting to relieve the cardiorespiratory and gastrointestinal manifestations of tense ascites. Diagnostic paracentesis is indicated in any patient whose ascites is of new onset or to disclose the presence of infection in patients with known or suspected ascites, particularly in the context of alcohol-related cirrhotic liver disease. Diagnostic paracentesis is also useful in the management of the AIDS patient, in whom the etiology of ascites will be non-AIDS related in three-quarters of cases. [101]

There are few relative contraindications to abdominal paracentesis. Certain systemic and anatomic risks should be considered.

---

**TABLE 46-10** -- Classification of Ascites by Serum-Ascites Albumin Concentration Gradient
*From Runyon BA: Ascites. In Schiff L, Schiff ER (eds): Diseases of the Liver. 7th ed. Philadelphia, Lippincott-Raven, 1993, p 997.*

| High Gradient (1.1 g/dL) |
| --- |
| Cirrhosis |
| Alcoholic hepatitis |
| Cardiac ascites |
| Massive liver metastases |
| Fulminant hepatic failure |
| Budd-Chiari syndrome |
| Portal vein thrombosis |
| Veno-occlusive disease |
| Fatty liver of pregnancy |
| Myxedema |
| Mixed ascites |

| Low Gradient (<1.1 g/dL) |
|---|
| Peritoneal carcinomatosis |
| Tuberculous peritonitis |
| Pancreatic ascites |
| Biliary ascites |
| Nephrotic syndrome |
| Serositis in connective tissue diseases |

**Systemic**

Given the predominance of alcohol-related cirrhotic liver disease as the cause for ascites, as many as two-thirds to three-quarters of patients subjected to paracentesis will have a coagulopathy. A conservative approach is to administer platelets to patients with levels <50,000/3 (i.e., <50 × 10$_9$ /L), or to give fresh frozen plasma to those with a prothrombin time exceeding 20 seconds (1.5 times the therapeutic level). [102] However, there are no controlled data to support these contentions. [103] Rather, the only prospective study to evaluate the complications of paracentesis determined that transfusion-requiring abdominal hematomas occurred in <1% of cases despite the fact that 71% of patients had an abnormal prothrombin time. [104] Because transfusion-requiring hematoma is so unlikely, even in this population, prophylactic administration of fresh frozen plasma or platelets imposes considerable cost, in addition to the risk of post-transfusion hepatitis, with little net gain.

**Anatomic**

Structural impediments to the safe introduction of a paracentesis needle can include the bladder, bowel, and pregnant uterus. The bladder is normally tucked into the recess of the pelvis. However, neuropathically distended bladders caused by pharmacologic

agents or medical conditions should preferably be emptied by voiding or by catheterization to avoid puncture. Intestines typically float in ascitic fluid and will move safely away from a slowly advancing paracentesis needle. [102] Even if penetrated by an 18- to 22-ga needle, leakage of intestinal contents will not occur unless the intraluminal pressure is 5- to 10-fold greater than normal conditions. [105] Therefore, US guidance may be indicated in cases of suspected adhesions or bowel obstruction. In second- and third-term pregnancy, an open supraumbilical or US-assisted approach is preferred.

The abdomen should be carefully inspected for evidence of abdominal hematoma, engorged veins, or superficial infection, and these sites should be strictly avoided.

## Technique

### Preliminary Actions

The operator should comply with guidelines for body fluid precautions (see Chapter 75) . The patient should be carefully draped and the skin thoroughly prepped in sterile fashion to prevent the iatrogenic introduction of bacteria into the abdominal wall tract or peritoneal cavity.

### Site of Entry

The preferred site is approximately 2 cm below the umbilicus in the midline (Fig. 46-9) . The fasciae of the rectus abdominis join to form the fibrous, thin, avascular linea alba. [102] Large collateral veins may occasionally be present and should be avoided, as should suspected areas of skin infection. If the patient has midline scarring, the preferred alternate site is in either lower quadrant, approximately 4 to 5 cm cephalad and medial to the anterior superior iliac spine (see Fig. 46-9) . The importance of remaining lateral to the rectus sheath is to avoid the inferior epigastric artery. Patients with a large quantity of ascites can readily undergo the procedure in the supine position with the head of the bed slightly elevated. Those with lesser amounts of fluid may profit from a lateral decubitus position with introduction of the needle into the midline or dependent lower quadrant (Fig. 46-10) . Some clinicians prefer to use the lateral decubitus position routinely because the bowel tends to float upward and away from the path of the needle. Rarely, patients may need to be placed in a facedown, hands-on-knees position. [104] In the patient with multiple abdominal scars or suspicion of compartmentalized abdominal fluid for any reason, US guidance is prudent.

### Procedure

Following sterile preparation of the skin, local anesthesia is administered at the paracentesis site. A standard 3.8 cm (1.5 in.) metal needle is sufficient in most cases. [93] An 8.9 cm (3.5 in.) spinal needle may be required in obese patients. Plastic sheath cannulas tend to kink and run the risk of being sheared off into the peritoneal cavity. A steel needle can be left in the abdomen during a therapeutic tap for intervals of an hour or more without injury. A smaller-gauge needle (20- to 22-ga), is appropriate for diagnostic taps, as these lessen the likelihood of postprocedural ascitic fluid leak

through the wound site. However, large-volume therapeutic paracenteses benefit from 18-ga needles, as this permits expeditious outflow. [107]

The needle can be inserted directly perpendicular at the preferred site. Certain authors prefer the "Z-tract" method wherein the skin is pulled approximately 2 cm caudad in relation to the deep abdominal wall by the non-needle-bearing hand while the paracentesis needle is being slowly inserted (Fig. 46-11) . [108] The skin is not released until the needle has penetrated the peritoneum and fluid flows. When the needle is removed following the procedure, the skin will slide to its original position and help seal the tract. In any case, slow insertion of the needle in 5-mm increments is preferred. This allows the operator to detect undesired entry of a vessel and helps prevent unnecessary puncture of small bowel. Continuous suction should be avoided as it may attract bowel or omentum to the end of the paracentesis needle with resultant occlusion. Once fluid is flowing, the needle should be stabilized to ensure a steady flow. If flow ceases, the needle should be gently rotated and advanced inward using 1- to 2-mm increments.

## Complications

Complications can be divided into systemic, local, and intraperitoneal categories.

### Systemic

An oft-cited but poorly documented concern is hemodynamic compromise caused by overzealous removal of ascitic fluid. Because upward of 6 L has been reported to be removed in <15 minutes without complication, certain authorities decry this issue as folklore. [109] [110] Others believe that rapid total paracentesis is accompanied by marked cardiovascular and humoral changes, some of which are explained by mechanical factors directly or indirectly related to relief of abdominal pressure. [111] Other changes, including systemic vasodilation and humoral deactivation, are of a nonmechanical nature. Because fluid and electrolyte shifts may accompany the removal of large amounts of fluid, certain authors recommend routine administration of colloid. [84] While albumin was the de facto choice, colloid dextran 70 is currently favored due to cost and infection concerns. However, it is not clear that routine usage is justified. [114]

### Local

Local complications include persistent ascitic fluid leak at the wound site, abdominal wall hematoma, and localized infection. Persistent fluid leak can be corrected with a single suture at the site of puncture. An abdominal wall hematoma requiring transfusion is very uncommon, but careful observation in such cases is necessary.

### Intraperitoneal

Intraperitoneal complications include perforation of vessels and viscera. [115] In experienced hands, these are uncommon, and in most circumstances they are self-sealing and clinically inconsequential. However, generalized peritonitis and

abdominal

| TABLE 46-11 -- Ascitic Fluid Laboratory Data to Be Obtained on Patients With Ascites *From Runyon BA: Ascites. In Schiff L, Schiff ER (eds): Diseases of the Liver. 7th ed. Philadelphia, Lippincott-Raven, 1993, p 997.* | |
|---|---|
| **Routine** | **Unusual** |
| Cell count | Tuberculosis smear and culture |
| Albumin | |
| Culture in blood culture bottles | Cytology |
| | Triglyceride |
| **Optional** | Bilirubin |
| Total protein | **Unhelpful** |
| Glucose | pH |
| Lactate dehydrogenase | Lactate |
| Amylase | Cholesterol |

| Gram stain | Fibronectin |
| --- | --- |

wall abscess have been reported after paracentesis in rare cases. The most common cause of postparacentesis intraperitoneal hemorrhage is bleeding due to a coagulopathy, rather than large-vessel injury per se.

## Interpretation

Ascitic fluid should undergo gross inspection. Routine laboratory testing includes a differential cell count, albumin assay, and cultures (Table 46-11) .

### Inspection

Ascitic fluid is typically translucent and yellow. A dark greenish-brown hue may reflect biliary perforation. Cloudy fluid generally indicates particulate matter, including neutrophils: fluid with counts >5,000/muL (i.e., >5,000/mm3 ) are cloudy and those >50,000/muL are purulent. An opaque, milky appearance may indicate elevated triglyceride levels. [116] A blood-tinged appearance requires at least 10,000/muL RBCs. This may reflect an iatrogenic complication, malignancy, hemorrhagic pancreatitis, or tuberculous peritonitis, although the last diagnosis creates hemorrhagic-appearing fluid in <5% of cases. [104]

### Cell Count

Several milliliters of ascitic fluid are sufficient to obtain a differential cell count. Cirrhotic ascites should generally contain <250 WBCs/muL (Table 46-12) (Table Not Available) . However, because cells may exit through the peritoneal cavity more slowly than fluid does, the WBC count can rise in the ascitic fluid during the procedure. [117] Thus, an upper limit for uncomplicated cirrhotic ascites is reported as 500 cells/muL. [119] [120] Lymphocytes should predominate, and clinical signs or symptoms of peritoneal infection should be absent. [121] In cases in which spontaneous bacterial peritonitis is a clinical consideration, the WBC criterion is 250 WBCs/muL with >50% polymorphonuclear leukocytes.

### Albumin

A serum-ascites albumin gradient can be obtained by simultaneous measurement of ascitic and serum-ascites albumin gradient. A serum-ascites albumin gradient >1.1 g/dL indicates portal hypertension with >95% accuracy (see Table 46-10) . [123] [124]

### Culture and Gram Stain

The most valuable method for determining the presence of infection is culture. The sensitivity of this test is markedly increased by the direct inoculation of blood culture

bottles at the bedside in contrast to simply delivering the ascitic fluid to the laboratory. [108] [125] Approximately 10 bacteria/muL of fluid is required for a positive Gram stain. Thus, the Gram stain is notoriously insensitive in spontaneous bacterial peritonitis in which the medium concentration of bacteria is 10-3 organisms/muL of fluid. [125] The Gram stain can only be expected to be helpful in cases of free gut perforation.

**Miscellaneous**

Optional tests include measurement of total protein, glucose, lactate dehydrogenase, and amylase. These will be beneficial in selected circumstances and need not be obtained on a routine basis. Immunosuppressed patients, including those with AIDS, should undergo microbiologic testing for opportunistic infections, including tuberculosis. [101] Cytologic analysis is recommended in patients with suspicious constitutional symptoms and signs. [126] Triglyceride and bilirubin studies are indicated if the gross appearance of the fluid is suggestive of increased levels. [127]

## Summary

Abdominal paracentesis is a straightforward, commonly used procedure in the emergency department, with few relative contraindications. The imperative of paracentesis is to disclose the presence of infection in the peritoneal cavity. It is also useful in the diagnosis of other conditions and contributes to the relief of manifestations of tense ascites.

# Chapter 47 - Abdominal Hernia Reduction

**Lee E. Payne, Jonathan M. Glauser**

The emergency physician plays an important role in evaluating and treating the patient with abdominal hernia. One must distinguish abdominal hernia from other potential causes of groin or abdominal swelling. If a hernia is present, the physician must determine if incarceration or strangulation has occurred. An incarcerated hernia that cannot be reduced is a surgical emergency. In many instances the skilled emergency physician will be able to reduce an incarcerated inguinal hernia, allowing for a less urgent surgical repair. Special concern is warranted in neonates, and infants who may present with irritability as their only symptom of incarcerated hernia. Finally, those patients with a newly diagnosed or an easily reducible hernia must be given timely and appropriate referral for future surgical repair.

## EPIDEMIOLOGY

Abdominal hernia is a common problem in the general population, and multiple varieties exist, including inguinal hernias (direct and indirect), femoral hernias, incisional hernias, ventral hernias, lateral abdominal wall hernias, internal hernias, and post-traumatic hernias. Although the exact prevalence of hernias is unknown, Zimmerman and Anson estimate that approximately 5% of the total adult male population is afflicted. [1] Hernias of all types are 5 times more common in males.

Herniorrhaphy is one of the most common procedures in the United States, with 500,000 performed annually. Groin hernias include indirect and direct inguinal hernias, as well as femoral hernias. The most common hernia for both sexes is the indirect inguinal hernia, accounting for 90% of cases in children and young adults. [2] The male-to-female ratio for inguinal hernia is 9:1 secondary to the anatomic differences in embryologic development. The causes of abdominal wall hernia change with increasing age. Congenital inguinal hernias are common in children and young adults, whereas acquired hernias rise in frequency in the elderly. [3] [4] Inguinal hernias still make up nearly 50 to 60% of hernias in older males, but there is an increase in acquired hernias in older males as well. [3] Acquired hernias are associated with age-related decreased tissue elasticity and conditions that

increase intra-abdominal pressure, including chronic constipation, chronic cough (e.g., with chronic obstructive pulmonary disease), bladder outlet obstruction, and kyphoscoliosis. [2] Direct hernias are unusual in women, whereas femoral hernias occur more commonly in women than in men. Inguinal and femoral hernias are much more likely to incarcerate due to the anatomic structures involved when compared with other categories of abdominal hernia.

In the past 20 years, neonatal intensive care units have improved the long-term survival of premature infants. The occurrence rate of hernias in the premature infant is near 30%.  The rate of incarceration, strangulation, and gonadal infarction for premature infants is twice the rate for the general pediatric population. [7] This fact should heighten

the emergency physician's awareness to the potential presence of incarcerated inguinal hernia in children who present to the emergency department (ED) with a history of premature birth and irritability.

# CLASSIFICATION OF ABDOMINAL HERNIAS

Detailed discussions of the historical development of the treatment of hernias, including recognition, preoperative management, and operative repair, can be found elsewhere.

A hernia can be defined as a potential weakness in the abdominal wall, and this defect permits protrusion of peritoneum and abdominal contents. If a contained viscus can be returned from the defect in the abdominal wall to the abdominal cavity, the hernia is defined as *reducible*. When the contents of the hernia sac cannot be reduced, the hernia is considered *incarcerated*. For the purposes of this chapter, a hernia is defined as incarcerated when the patient, family, or physician on initial evaluation (before medication and other techniques described later), is unable to reduce the hernia sac. When there is ischemia of the contents of the hernia sac, the herniated tissue is said to be *strangulated*. A *sliding* hernia occurs when a portion of the wall of the hernia sac is composed of an organ (e.g., the cecum or ovary). If only a portion of the antimesenteric wall of the bowel is incarcerated or strangulated, the hernia is known as a *Richter* hernia.

Groin hernias can be categorized as femoral or inguinal hernias; inguinal hernias can be either direct or indirect (Figs. 47-1 (Figure Not Available) and 47-2) (Figure Not Available) .

## Direct Inguinal Hernia

A direct inguinal hernia represents a weakening of the abdominal wall (transversalis fascia) that permits protrusion of peritoneal contents through the Hesselbach triangle. The triangle is bounded laterally by the inferior epigastric artery, inferiorly by the inguinal ligament, and medially by the lateral margin of the rectus muscle sheath. The key to diagnosis of a direct inguinal hernia is the observation that the hernia does not traverse the inguinal canal. In the rare situation in which the hernia sac enters the scrotum, the hernia sac must pass through the external ring separate from and behind the spermatic cord. In contrast with other groin hernias, the direct hernia represents a diffuse weakness of the abdominal wall. The absence of a narrow neck with the direct hernia sac makes incarceration unusual. When incarceration does occur, it is usually associated with rare entrapment of the hernia sac at the external ring.

## Indirect Inguinal Hernia

An indirect inguinal hernia represents the passage of the hernia sac (peritoneum and contents) through the internal inguinal ring. During fetal development of the male gonads, the processus vaginalis is formed, allowing the descent of the testes into the scrotum. Failure to completely close this opening after birth allows a hernia or a hydrocele to develop. The peritoneal contents may progress a variable distance down

the inguinal canal, even into the scrotum. The internal ring is defined medially by the inferior epigastric artery. Indirect hernias frequently are associated with scrotal swelling, and incarceration can develop as a result of swelling at either the internal or the external ring. The scrotal anatomy defining inguinal hernia and hydrocele is presented in Figure 47-3 (Figure Not Available) .

### Femoral Hernia

The weakness in the transversalis fascia that causes a femoral hernia is similar to the weakness that causes a direct inguinal hernia, but it is inferiorly rather than anteriorly directed. The peritoneum and contents herniate beneath the inguinal ligament in a small potential space medial to the femoral vein. The resulting hernia sac has a small neck, although once the sac enters the subcutaneous (SQ) tissue of the thigh, it may enlarge considerably and even double back on top of the external oblique aponeurosis, thus masquerading as an inguinal hernia. The narrow neck makes incarceration common with this type of hernia and preoperative reduction unlikely.

### Ventral Hernia

Ventral hernias are frequently caused by postincisional weakness of the anterior abdominal wall. Umbilical hernias represent another common ventral hernia and rarely pose a problem. An *epigastric* hernia may penetrate through a defect in the linea alba above the umbilicus. The *Spigelian* hernia is a rare defect located at the inferior one third of the rectus abdominis where the arcuate line meets the lateral border of the rectus muscle.

### Other Hernias

Lateral abdominal wall hernias are uncommon but may develop following renal surgery. A rare site of abdominal weakness and herniation is the *Petit triangle.* This triangle is bounded anteriorly by the external oblique muscle, inferiorly by the iliac crest, and posteriorly by the latissimus dorsi muscle. Injury to the abdominal wall can cause traumatic hernia, with attendant risk of associated trauma and bowel incarceration. [12] Other hernias that are not discussed further include perineal hernias through the levator ani or through the sciatic foramina, obturator hernias into the medial thigh through the obturator foramen, diaphragmatic hernias, and internal hernias (e.g., bowel herniation through the foramen of Winslow).

## DIFFERENTIAL DIAGNOSIS OF GROIN MASSES

While inguinal hernia is a common diagnosis in a patient presenting with a groin mass, other diagnoses, some potentially dangerous, may masquerade as a groin mass. An enlarged inguinal lymph node, a lipoma, a hydrocele of the spermatic cord, a saphenous vein varix, a psoas abscess, or an incarcerated ovary can all present as a mass in the groin. [13] More serious diagnoses may present incidentally within the hernia sac itself; these include blood from a ruptured spleen, [14] appendicitis or appendiceal abscess, [15] tuberculous peritonitis, [16] or torsion of an undescended testis. The emergency

physician must be cognizant that a diagnosis distant from the groin swelling may be the cause and search for other findings prior to attributing the mass to inguinal hernia.

Inguinal lymphadenopathy is common and can have many causes. Lymph nodes are usually multiple; are found distal to the groin crease; and, in general, are equally mobile in all directions, unlike a femoral hernia, which tends to have decreased mobility in the transverse direction. Finding an inflammatory lesion on the lower extremity distal to the mass lends credence to the diagnosis of lymphadenopathy. Ulceration of the urethral meatus can rarely cause adenopathy about the inguinal ligament, so the genitalia must also be examined. In most circumstances, however, inguinal nodes are unusual in the anterior abdominal wall immediately superficial to the inguinal ligament.

Lipomas in the SQ fat can usually be separated from the underlying fascia and are not associated with a hernia sac neck extending under the inguinal ligament. A saphenous vein varix may reduce in size with pressure or protrude when the patient coughs or stands upright, similar to an inguinal hernia, but it usually has a softer consistency than a hernia. Furthermore, as the varix enlarges and displaces SQ fat, it appears bluish beneath the skin.

Differentiating between incarcerated inguinal hernia and acute hydrocele of the spermatic cord may be difficult in an infant presenting with a tender, hard bulge at the external inguinal ring. It may be helpful to palpate the internal inguinal ring with an examining finger in the rectum while another finger examines the spermatic cord. Bowel should be felt entering and exiting the internal ring, if the mass is an incarcerated hernia. Scrotal hydroceles are usually benign and can generally be dealt with electively. Acute hydroceles within the scrotum rarely become hard or painful, unlike those within the inguinal canal. Rapid expansion of the hydrocele within the rigid walls of the inguinal canal can lead to testicular infarction from pressure on the spermatic cord at the internal or external inguinal ring. This condition can mimic torsion of the testis if ischemia has produced tenderness in the testicle. The testicle is at risk, even though the hydrocele may transilluminate. In the case of testicular torsion, the cord may be shortened on the affected side.

Although Doppler ultrasound and radionuclide scanning examinations may be helpful (see Chapter 59) , surgical exploration is often necessary to differentiate these conditions. In general, an acute hydrocele of the cord should be treated aggressively, as an incarcerated hernia or a testicular torsion, unless the emergency physician can definitively conclude otherwise.

Psoas abscesses are uncommon and are only rarely confused with a femoral hernia. The margins of the abscess are softer and more ill defined than those of a hernia. Furthermore, psoas abscesses lie in a position lateral to the femoral artery, whereas hernias are medial.

## PATIENT PRESENTATION

**Children**

Inguinal hernias are prone to incarceration. The overall rate of incarceration in children is 10 to 12%.  The rate of incarceration in premature infants and term children in the first few months of life is near 30%.  Often the hernia has gone unnoticed by the child's parents and has developed since birth. In 1 series of 908 patients receiving herniorrhaphies, incarceration was the first sign of the presence of an inguinal hernia in 65% of the 85 patients who presented with incarceration. [21] In a small infant, irritability may be the main parental concern, and the physician must be certain to examine the groin to evaluate the child for a potential hernia. Any child presenting with irritability must be completely undressed and a thorough examination performed to avoid missing the diagnosis. Very large hernias containing several loops of bowel almost never incarcerate and even more rarely strangulate due to the large size of the hernia neck.

An incarcerated hernia presents as a tender mass in the inguinal canal. The skin over the mass may be edematous, erythematous, or blue. [19] Duration of incarceration in pediatric patients has not been shown to predict the presence of strangulated bowel, but prolonged incarceration should increase one's level of concern for strangulation. Findings suggestive of compromised blood supply to the bowel include abdominal distention, bilious vomiting, tachycardia, constipation and obstipation, blue discoloration of the skin, and radiographic evidence of an obstructive pattern on abdominal plain films. [19] Leukocytosis and fever are unreliable predictors of strangulation. [22] Obvious signs of strangulation contraindicate attempted reduction and warrant immediate operative intervention.

Inguinal hernias are rare in females but, when present, are more likely to incarcerate than similar hernias in males. [19] In addition, the rate of emergency operation for incarceration is higher for female infants. [19] In female infants, a sliding hernia may occur, with the ovary and/or the fallopian tube herniating into the inguinal canal. The herniated ovary is usually palpable as a mobile nodule measuring 1 × 2 cm. Although unusual, infarction of the herniated ovary may occur as a result of torsion or compression of the pedicle. [19] This condition may mimic inflamed lymphadenopathy, although the location of the mass would be atypical for adenopathy. Some authors recommend investigation for testicular feminization in female patients presenting with inguinal hernia. An undescended testicle may be present in the inguinal canal, even though the patient is a phenotypic female. Patients with testicular feminization have a 50% incidence of inguinal hernia, while females with inguinal hernia have an approximate incidence of testicular feminization of 1%. [23]

The importance of attempting early reduction for incarcerated inguinal hernias lies in the observed complication rates. The complication rate for elective herniorrhaphy is reported to be 1.7 to 4.5%, while the rate following emergency surgery is 22 to 33%. [6] [17] In children, the success rate for reduction of incarcerated hernia is 70 to 90%.Some have recommended elective surgical repair within 24 to 48 hours after successful reduction, as the rate of recurrent incarceration is high. [19]

**Adults**

Although inguinal hernias still predominate, other hernias become more common in the adult patient and thus have slightly different presentations and complications. Controversy exists regarding attempted reduction of incarcerated hernias in the adult patient. Although the potential complications include reducing necrotic bowel, reduction en masse, or rupture of the bowel wall, most authors agree that a gentle attempted reduction is warranted, as it appears unlikely that strangulated bowel will be able to be reduced.

Unusual conditions (e.g., a Richter hernia, which may mimic a groin abscess because of the absence of bowel obstruction symptoms, [25] and traumatic abdominal wall hernias, which may mimic a hematoma following injury) may be confusing. As in the pediatric patient, intra-abdominal hemorrhage and a variety of other more unusual diseases and pathologic conditions can present within an inguinal hernia and warrant the emergency physician's attention. [14]

Whereas incarceration occurs in approximately 6 to 10% of indirect inguinal hernias in the adult, [28] 14 to 56% of femoral hernias incarcerate. The femoral hernia may be difficult to diagnose and may present first with incarceration. The clinical presentation includes ill-defined groin pain; centralized abdominal pain; and pain that is increased by coughing, sneezing, or lifting. [32] Signs of intestinal obstruction may also be present. [33] A femoral hernia presents a special problem, in that the small neck present in the femoral canal and the usual generous overlying SQ tissue make a complete reduction virtually impossible. Therefore, patients with femoral hernias are best referred on an emergency basis to a surgical consultant for evaluation and consideration of operative reduction and simultaneous hernia repair. Attempts at reduction are best deferred until such consultation has been obtained.

Incisional hernias infrequently incarcerate or strangulate. Reduction of these hernias is generally routine or spontaneous, although sedation may be required. Incarceration in these cases usually results from adhesions of the contents of the sac either to each other or to the sac itself. Occasionally feces within the loops of bowel in the hernia prevent reduction.

If *strangulation* occurs, most commonly the clinical picture is one of intestinal obstruction. In fact, 20 to 25% of the adult cases of bowel obstruction may be caused by incarcerated hernia. [34] The patient experiences severe pain, vomiting, distention, and obstipation. The duration of incarceration is important in the adult, as patients who have had the incarceration for >24 hours have a higher likelihood of strangulation. [18] The patient may be febrile and may have a

leukocytosis, although these findings may be absent. [18] No impulse may be transmitted to the hernia sac on coughing, and the overlying skin often becomes inflamed and edematous. These features may be less marked if only omentum is contained within the sac. Ultimately gangrene of the bowel or the omentum occurs if strangulation is not

relieved and the involved gut perforates into the sac.

## INDICATIONS AND CONTRAINDICATIONS

Whenever possible, incarcerated hernias should be reduced. Reduction minimizes inflammation and tissue edema, thus preventing strangulation. Some clinicians state that it is impossible to reduce a strangulated hernia because of the extensive inflammation, edema, and tenderness. Fortunately, most patients present early after incarceration, and the question of whether strangulation has occurred is rarely an issue. In most cases the patient presents within hours of the incarceration without fever, leukocytosis, or other evidence of generalized toxicity. In that circumstance, reduction of the hernia is indicated. Depending on one's location and practice situation, it may be prudent and appropriate to involve a surgical consultant prior to an attempt at reduction. Given that the condition will require the consultant's services if the reduction is unsuccessful, when possible, it is advised that the treatment plan be discussed with the consultant early in the course of evaluation.

Peritonitis and other clinical evidence of strangulation (e.g., fever, marked leukocytosis, toxic appearance) are contraindications to an attempt at a nonsurgical reduction of an incarcerated hernia. In addition, immediate surgical repair of a traumatic hernia is recommended both for prevention of delayed morbidity from herniation and for evaluation of underlying organ injury. [26] Similarly, an ovary or an undescended testis in an incarcerated inguinal hernia represents an indication for surgical reduction.

## PROCEDURE

When clinically indicated, gentle reduction of incarcerated hernias should be attempted. Adequate sedation must be given to allay anxiety and to minimize discomfort (see also Chapter 35) . In recent years more emphasis has been placed on sedation of patients, particularly children, for painful procedures. The relief of pain and prevention of suffering is one of the emergency physician's most important responsibilities. The decision to use sedation, which agents to use, and the most appropriate route of administration, is complicated and involves many factors, including the patient's age, underlying disease states, intended duration of sedation, availability of reversal agents, and potential complications. The physician and nursing staff must be familiar with sedation techniques and recovery and capable of performing airway management procedures should a complication arise. Each ED should have available staff and standardized procedures for adequate monitoring, evaluation, and postsedation recovery. [35]

There is no lower age limit for sedation in children, but children below the age of 3 months, and particularly premature infants, require skilled observation due to the potential for apnea. [36] [37] Some authors recommend against the use of morphine in infants and premature neonates, due to exaggerated effects on the respiratory centers. [7] If patients are carefully selected, the staff are well prepared, and standardized procedures are in place, most pediatric patients can safely undergo ED sedation for incarcerated hernia reduction.

The sedation needed for hernia reduction should be sufficient to inhibit straining or struggling. If relaxation alone does not produce reduction, a period of protection from pain is needed as the hernia is manually reduced. There are several agents and routes of administration that can provide the needed sedation for this procedure. In most situations, IV administration of medication is preferable and is most likely to produce safe and controlled sedation. The combination of an IV opioid agent and an IV benzodiazepine agent (e.g., fentanyl and midazolam) is commonly used. This combination, when used cautiously, provides an excellent mixture of analgesia, anxiolysis, and amnesia. Dosing for this combination and for other agents is discussed in Chapter 35 .

Following sedation, the patient is placed in a head-down tilt of approximately 20° for groin hernias but with no tilt when other hernias are treated. A pillow may be placed under the buttocks. For infants, the legs should be swaddled and secured to prevent slippage. This technique is successful in spontaneously reducing 80% of pediatric incarcerated hernias over a 2-hour period without manipulation. In addition, the application of a cold pack or a padded ice bag to the hernia, covered with cloth or gauze (to prevent cold injury to the skin), may reduce local blood flow and diminish intraluminal gas pressure while the analgesic is taking effect.

If, after 20 to 25 minutes, spontaneous reduction has not occurred, gentle manipulation can be added. Fraser [38] has described the unilateral frog technique, depicted in Figures 47-4 (Figure Not Available) through 47-6. The patient is placed in the supine position and the pelvis firmly grasped by an assistant to prevent lateral movement of the hips (see Fig. 47-4) (Figure Not Available) . The ipsilateral leg is then externally rotated and completely flexed and placed in the frog position (see Fig. 47-5) (Figure Not Available) . This has the anatomic result of having the external inguinal ring more closely override the internal inguinal ring. The physician then places the first 2 fingers of the "guiding" hand over the hernial bulge, overriding the external inguinal ring in such a manner as to prevent the hernial sac from subluxating over the margin of the ring. The apex of the hernia is then grasped between the first 2 fingers of the "reducing" hand, and prolonged, firm, steady pressure is applied (see Fig. 47-6) (Figure Not Available) . The reducing hand should stay in place as long as possible; in fact, feeling stiffness in the first 2 fingers and an ache in the thenar eminence of the reducing hand is indicative of the correct position. These maneuvers, if successful, often result in a gurgle that signifies hernia sac reduction. A slow, steady, atraumatic manipulation is preferred. Repeated forceful attempts at reduction are contraindicated. Reduction of other abdominal wall hernias is accomplished in a fashion similar to that described for inguinal hernias.

Once reduction occurs, corrective surgery should be considered within 24 to 48 hours, after local edema has subsided. [19] The risk of recurrent incarceration is high, and relatively urgent surgery is indicated. Fortunately, the complications of surgery performed during this time frame are the same as for elective herniorrhaphy. If reduction cannot be achieved, emergency surgery is indicated after dehydration and electrolyte imbalances are corrected. Nasogastric suction is indicated if obstruction is present.

## COMPLICATIONS

An attempt at manual reduction of a hernia may be unsuccessful or only partially successful. The physician must recognize a partial reduction and obtain appropriate consultation. Persistent attempts at reduction produce patient discomfort and may delay operative intervention for relief of bowel obstruction or organ ischemia. Hernia reduction en masse may occur when the hernia sac is displaced into the preperitoneal space, giving the clinician the impression that reduction has been achieved. The hernia sac remains incarcerated, outside the abdominal cavity, and at risk for strangulation. This is a rare phenomenon and can occur spontaneously, without attempted reduction. Testicular infarction and subsequent atrophy may occur after an incarcerated hernia or (as mentioned above) with an acute hydrocele of the spermatic cord. This is presumably secondary to ischemia and venous engorgement limiting testicular blood flow.

## CONCLUSION

By far the greatest challenge in the immediate management of groin lumps is the differential diagnosis of conditions that may mimic a groin hernia. Reduction of groin and other abdominal wall hernias is usually straightforward. Once the hernia is reduced, the patient and family must be told that straining at stool, urination, or coughing may produce another prolapse of the hernia sac. The patient or family should rapidly reduce any recurrent herniation to avoid recurrent incarceration and the need for a return to the ED. A demonstration of the reduction technique to be used by the patient or family usually aids in gaining cooperation. The emergency physician should not overlook the need to evaluate and treat potentially complicating conditions (e.g., urinary tract infections, bladder outlet obstruction, constipation, and respiratory infections) that may increase intra-abdominal pressure and produce recurrent herniation. Appropriate referral for these related problems and subsequent care of the reduced hernia is recommended.

Finally, the emergency physician must be aware that to the patient, the development of hernia symptoms is often associated with employment-related activities. Although physical exertion can certainly exacerbate a preexisting weakness in the abdominal wall and can lead to frank herniation, most frequently the abdominal wall defect is congenital in origin. Therefore, to minimize confusion in worker's compensation cases, the physician should be careful to note that the patient's hernia became symptomatic during an activity rather than stating that the hernia was the result of an activity.

# Chapter 48 - Rectal Procedures

**Scott M. Davis**

Anorectal disorders are infrequent but by no means rare complaints in the emergency department. The emergency physician should have the ability to diagnose and treat common anorectal complaints. This chapter will discuss four procedures that are well suited to the emergency department setting: anoscopy, excision of thrombosed external hemorrhoids, removal of foreign bodies, and reduction of rectal prolapse.

Many anorectal conditions are painful and consequently prompt emergency evaluation. Bleeding, although usually not life threatening, is also worrisome to patients. The emergency physician should realize that patients are often self-conscious concerning anorectal complaints. By presenting to a public facility, the patient may have already subjected him- or herself to a great deal of embarrassment, and therefore, the physician should maintain a sense of objectivity and professionalism. A nonjudgmental and nonthreatening approach will enhance the physician-patient relationship and the probability of patient cooperation during any subsequent procedure.

Patients may be particularly anxious concerning anorectal procedures, especially if they are already in pain. As for most procedures, it may be difficult, if not impossible, to adequately examine and treat a patient who is not able to cooperate because of severe pain and/or anxiety. Therefore, *the emergency physician should consider the liberal use of analgesia and sedation prior to any invasive anorectal examination or procedure*. The common practice of coating the anoscope or examining finger with lidocaine jelly or ointment immediately prior to a procedure provides lubrication, but it offers no analgesia. Hence, parenteral benzodiazepines and opioids should be instituted in appropriate situations. The use of parenteral analgesia and sedation is outlined in detail in Chapter 35 .

## ANATOMY

The terminal portion of the gastrointestinal tract is divided into the sigmoid colon, the rectum, and the anus (Fig. 48-1) (Figure Not Available) . The anus constitutes the terminal 4 cm of the gastrointestinal tract. The rectum extends proximally an additional 12 to 15 cm from the anus and joins the sigmoid colon. The junction between the anus and rectum is called the *pectinate line*. This line divides the anus, which is covered by squamous epithelium, from the rectum, which is covered with mucous membrane. This explains why painful anorectal lesions usually involve the anus, as it is innervated by cutaneous fibers. Superficial (mucosal) rectal lesions usually cause little pain, as the rectum is innervated by visceral fibers.

At the pectinate line, there are anal crypts with mucus-secreting glands. This site is where most anorectal abscesses are believed to arise. Above the pectinate line is the internal hemorrhoidal plexus, where internal hemorrhoids usually become symptomatic with painless bleeding. The external hemorrhoidal plexus, where external hemorrhoids originate, is below the pectinate line. Because the lesions are covered with skin, they

usually cause significant pain.

# ANOSCOPY

## Indications and Contraindications

Anoscopy is used to visualize the anal canal. It is inadequate to properly evaluate for rectal pathology, as only the most terminal portion of the rectum can be visualized. Anoscopy is used most often to diagnose anal foreign bodies, infections, anal tears, or internal hemorrhoids.

Anoscopy can be helpful in evaluating the following conditions: bright red blood per rectum; recent change in stool character, such as diarrhea or constipation; pain on defecation; rectal discomfort; anorectal drainage; and conditions related to the anorectal area, such as warts, fistulae, anal discharge, or pruritus.

There are few contraindications to anoscopy, with imperforate

anus being the only absolute contraindication. Anal stenosis or severe pain are relative contraindications.

## Equipment and Setup

There are various designs of anoscopes available. One style consists of a stainless steel or clear plastic tube, with a removable obturator inside (Fig. 48-2 *A* and *B*). This style requires an external light source to visualize the anal canal. A second popular style of anoscope has an integrated light source which eliminates the need for a head lamp or other light source operated by an assistant to illuminate the anal vault during the procedure (Figs. 48-2 *C* and (Figs. 48-3 ).

Additional equipment required may include lubricants, topical anesthetic agents, cotton swabs, forceps, or culture media, depending on the situation.

## Technique

### Preparation

Appropriate sedation and analgesia as previously described should be instituted prior to the procedure, especially for particularly painful disorders. Mild pain relief may be afforded by prior application of a topical anesthetic such as lidocaine jelly (2%) for painful anal conditions such as external hemorrhoids or anal fissures. *Topical agents should be administered 5 to 10 minutes prior to the procedure for optimal effect.* It is common for the physician to underestimate the degree of pain produced by anoscopy for many common anorectal conditions.

**Positioning**

The examination can be performed with the patient in either the lateral decubitus position (Sims position) or the knee-chest position. The choice of position is dependent on the preference of the physician and the tolerance of the patient (Fig. 48-4) (Figure Not Available) .

In the lateral decubitus position, the patient is placed on his or her left side. The knees and hips are flexed, and the patient's buttocks are positioned just over the edge of the bed. The left lateral position is best for a right-handed examiner.

The knee-chest position may be done on a bed or, preferably, on a tilt table. It may be difficult for ill or elderly patients to maintain this position.

**Procedure**

A routine digital rectal examination should be performed prior to anoscopy. One must determine the presence or absence of fecal impaction, mass, enlarged prostate, or stricture. The well-lubricated finger is introduced to check for bleeding, mass, or foreign body and to localize pain or pathology. The axis of the anal canal should also be noted to properly direct the anoscope on insertion. Although digital rectal examination has been reported to cause cardiac dysrhythmias such as ventricular fibrillation, this is quite rare. [13] This should be kept in mind when dealing with patients with significant underlying diseases, such as severe coronary artery disease, or a history of cardiac dysrhythmias. One may wish to monitor such patients. In addition, the digital rectal examination does *not* significantly elevate levels of prostate-specific antigen (PSA). [14]

After the digital examination, the physician inserts a well-lubricated anoscope. With the obturator inserted completely into the anoscope, the examiner gently advances the instrument while the patient bears down slightly against the instrument. Once the instrument dilates the anal sphincters at the anal orifice, the anoscope usually passes gently into the anorectum. During insertion, the physician's thumb is placed on the obturator to secure it in place. If the obturator falls back, the anal mucosa may be pinched, causing pain or bleeding. Consequently, the obturator should not be reinserted unless the entire anoscope has been completely removed from the patient. The instrument should be inserted until the outer flange impinges on the anal verge (Fig. 48-5) . [5]

Once the anoscope is inserted to the hilt, the obturator is completely removed. The anal canal is then visualized as the anoscope is gradually removed. If discharge is present, the mucosa should be swabbed clear and appropriate cultures taken. If bleeding or debris is present, this may be swabbed to aid visualization.

As the anoscope is withdrawn, it may be rotated in a circular fashion so the entire wall can be visualized. Near the last stage of withdrawal, reflex spasm of the anal sphincters may cause the anoscope to be expelled quickly. To obtain an adequate view, the

physician may need to repeat the procedure, reinserting the anoscope several times as necessary.

The following pathologic entities should be looked for: internal and external hemorrhoids, anal fistulae, anal fissures, sentinel tags, tumors, blood or pus, ulceration, parasites, or mucosal irregularities. [5]

**Figure 48-4** (Figure Not Available) Positions for performing sigmoidoscopy or anoscopy. *(From Hill GJ II: Outpatient Surgery. 3rd ed. Philadelphia, WB Saunders, 1988. Reproduced by permission.)*

### Complications

Anoscopy is a safe procedure, and complications are unusual. Irritation of local tissues is the most common complication and can result in increased bleeding or abrasion of the anorectal wall.

Use of sterilized instruments is imperative, as transmission of infectious diseases (e.g., hepatitis B, condylomata acuminata, herpes virus, or human immunodeficiency virus [HIV]) could occur.

## MANAGEMENT OF HEMORRHOIDS

Hemorrhoids have been known to mankind for centuries. [10] Hemorrhoids are varicosities of the venous plexus in the wall of the anorectum (Fig. 48-6 *A*). There are two types of hemorrhoids: internal and external. *Internal hemorrhoids* originate *above* the pectinate line and are covered with the columnar epithelium of the gastrointestinal tract. They usually present with painless bleeding, often following a bowel movement. Internal hemorrhoids may prolapse out of the rectum spontaneously or during defecation; when prolapsed, these hemorrhoids can be painful or bleed (usually bright red blood).

*External hemorrhoids* arise *below* the pectinate line and are covered with squamous epithelium (see Fig. 48-6 *B*). They may be pruritic, but patients usually become symptomatic when the external hemorrhoids become either inflamed or thrombosed. Pain may arise quite suddenly. Patients often report a mass or lump in the rectal area, and bleeding is uncommon but may occasionally be noted if the external hemorrhoid spontaneously ruptures. Because they are covered with skin, pain is the main hallmark of thrombosed external hemorrhoids. A thrombosed hemorrhoid appears as a tender purplish mass distal to the pectinate line and is usually visible when a patient's buttocks are spread. The "thrombosed" external hemorrhoid actually most often represents a rupture of one of the veins of the external hemorrhoidal plexus into the surrounding connective tissue. This condition is more accurately referred to as an *anal hematoma* or *external anal thrombosis*.

### Indications and Contraindications

There is little the emergency physician can or needs to do in the emergency department

for patients with internal hemorrhoids. Suppositories (such as Anusol-HC) and sitz baths may be helpful. When the prolapsed tissue is inflamed and painful, appropriate analgesic agents may be prescribed. Reduction of prolapsed hemorrhoids gives immediate relief, but they often recur with the next bowel movement. These patients should be referred for definitive therapy (i.e., ligation).

Nonoperative treatment of the external anal thrombosis will eventually result in spontaneous resolution over a week or so, leaving a residual skin tag. However, operative treatment is often required for pain management. More conservative therapy also puts the patient at risk of developing an impaction due to fear of painful defecation and the use of opioid analgesics.

Surgical excision is recommended for large, painful, thrombosed external hemorrhoids and can provide almost immediate relief to the patient. [17] The examining physician must be certain that a thrombosis exists prior to excision, because cutting into a nonthrombosed venous plexus can result in significant hemorrhage. A diagnosis of thrombosis can usually be made easily on clinical grounds by palpation of a firm, tender hemorrhoid, often noted to have a bluish discoloration. This presentation contrasts with that of an inflamed but *not* thrombosed hemorrhoid, which is often soft and distensible. [15] A small, tolerable, thrombosed hemorrhoid can be managed satisfactorily with conservative therapy consisting of stool softeners, sitz baths, anesthetic cream, and oral analgesics.

**Equipment and Setup**

Excision of a thrombosed hemorrhoid should be a relatively painless procedure, except for the transient pain associated with local anesthesia. Since significant pain as well as apprehension are involved with this procedure, the physician should use analgesia and sedation as outlined in Chapter 35 .

Other needed items include antiseptic skin preparation, appropriate drapes, tape, local anesthetic, and a standard suture or incision-and-drainage tray, with appropriate instruments (i.e., scalpel, tissue scissors, small curved hemostat).

**Technique**

The patient may be placed prone on the stretcher or in the left lateral decubitus position, depending on patient comfort. However, the latter position may require an assistant to provide adequate visualization.

The buttocks may be taped open to expose the anus (Fig. 48-7) . Benzoin will enhance tape adherence. Topical anesthetics may be used to decrease the pain of the initial injection.

After appropriate analgesia and sedation have been administered, local anesthesia is introduced by injecting a local anesthetic, preferably a long-acting preparation such as bupivacaine (Marcaine). A 25-ga needle is inserted at the center of the hemorrhoid, just

under the skin, but *not into* the hemorrhoid itself (Fig. 48-8) . Blanching should determine the success of infiltration. For a large clot or multiple small clots, a field block may be necessary, but this technique will be more painful (Fig. 48-9) . [15]

When anesthesia is achieved, the skin is elevated with a forceps. An elliptical incision is made around the clot and directed radially from the anal orifice (Fig. 48-10) . [18] Either a scalpel or tissue scissors may be used to make the incision. *A simple linear incision should not be used,* because it may close prematurely, preventing proper drainage. The flap of skin is then picked up and excised to expose the underlying clot; multiple separate clots may be present. The clot(s) can be removed with forceps or digital pressure (Fig. 48-11 *A- C*). Gentle probing and blunt dissection should be performed with a hemostat to be certain that all thrombi are exposed and removed. No other incisions are necessary, as this is a simple "unroofing" procedure. Bleeding is usually mild, and suturing is unnecessary. A small piece of surgical foam (Gelfoam) may be placed directly in the wound to promote hemostasis. Gauze pads placed over the area with the buttocks taped together are usually adequate as a dressing. [19]

## Postprocedure Care

Patients should be advised to rest several hours after the procedure to minimize bleeding. Warm sitz baths at least 4 times per day for at least 2 days may be started after the anesthetic has worn off. A cotton vaginal pad or gauze dressing may be placed over the anal region, although bleeding and drainage should be minimal.

Oral analgesia should be offered for a few days as needed. A hemorrhoidal cream (e.g., Preparation H, Anusol-HC) or an anesthetic cream (e.g., Xylocaine cream or jelly) may be beneficial for some patients. Although a stool softener is often recommended for several days to decrease straining and pain associated with defecation, this is generally not indicated unless opioid analgesics are prescribed and the patient has had significant prior constipation (as is common in the elderly population). Patients should avoid prolonged walking or sitting for a few days. Following a bowel movement, the patient should be instructed to wash the area with soap and water and to avoid toilet paper for a few days. Antibiotics are not indicated. [6]

Long-term recommendations to decrease the incidence of hemorrhoidal problems include increasing dietary fiber and possibly the use of stool softeners. Patients with particularly severe problems or multiple recurrences should be referred to a general or colorectal surgeon.

## Complications

It is important to remove the skin over the excised clot in the elliptical manner shown (see Fig. 48-10) . A simple stab incision with expression of the clot leads to a higher incidence of infection because the skin edges close prematurely. Such a limited excision also may lead to a higher incidence of recurrence as well as the creation of residual skin tags.

Postoperative bleeding may occur but is usually minimal. Bacterial infection is quite uncommon. A postoperative check is recommended in 2 to 4 days. The patient should be warned that hemorrhoids may recur in the future, although excision usually resolves that specific hemorrhoid.

# MANAGEMENT OF RECTAL FOREIGN BODIES

Patients with rectal foreign bodies often seek initial medical care through the emergency department. It is therefore imperative that the emergency physician have a basic understanding of the evaluation, treatment, and disposition of these patients. Many rectal foreign bodies may be removed successfully in the emergency department. By following appropriate guidelines, treatment in the emergency department can be cost-effective and practical.

The relatively recent increase in reported rectal foreign body cases likely represents both an increase in reporting and, more important, a true increase in incidence. The etiology of rectal foreign bodies includes (1) autoeroticism (most common), (2) iatrogenic (e.g., thermometer, rectal tube), (3) sexual or criminal assault, (4) self-administered treatment (e.g., enema), (5) accidental ingestion, and (6) concealment (e.g., body packing to conceal cocaine).

The types of foreign bodies seem limited only by the anatomic size of the rectum and the limits of human imagination. The myriad of foreign bodies that have been described include vibrators, phalluses, spray cans, hairpins, pencils, toothpicks, chicken bones, corncobs, glass jars, broomsticks, candles, flashlights, baseballs, vegetables, and electric light bulbs. Most objects are cylindric. The largest documented object was a stone that measured 25 cm in diameter. Colorectal foreign bodies constituted 13.3% of all gastrointestinal foreign bodies in 1 series. [29] Rectal thermometers are the most common object in the pediatric population. Males predominate in most series by as much as 28:1.

Older literature on this subject stressed inpatient treatment, and reports were skewed toward complicated cases and resultant complications. Most recent series show that there is a high success rate for the outpatient management of rectal foreign bodies, if proper guidelines for evaluation and treatment are followed.

**Indications and Contraindications**

Diagnosis of a rectal foreign body can usually be made easily from the history. In unclear cases, digital examination, plain

radiographs, or radiography using contrast media may help establish the presence of a foreign body. If a foreign body with a sharp edge is suspected from the history, the physician is advised to forego digital examination. Once the diagnosis is made, general principles of management include foreign body identification, foreign body removal, and

assessment for associated intraperitoneal and extraperitoneal injuries.

All rectal foreign bodies should be removed once they are diagnosed. Although some objects may pass spontaneously, *it is safest to assume that extraction is necessary*. Delayed removal may lead to obstipation, pain, and infection, with or without rectal perforation. The majority of rectal foreign bodies can be removed successfully and safely in the emergency department.  A rational approach to the removal of foreign bodies in the emergency facility is outlined in Figure 48-12 .

The majority of foreign bodies can be removed uneventfully in the emergency department if the patient has no signs of perforation and the object is palpable on digital examination. Radiographs may occasionally be needed to identify the location and type of foreign body or to rule out perforation. The specific technique for removal depends on the size, location, orientation, and composition of a particular foreign body. These techniques are described in the subsequent section.

After removal, sigmoidoscopy or anoscopy to assess mucosal integrity and evaluate for possible perforation is recommended.  Patients with normal postextraction examinations without clinical evidence of perforation may be safely released home with precautionary instructions.

Indications for hospital admission or outpatient surgery with general anesthesia in certain circumstances include (1) the diagnosis or suspicion of perforation; (2) the presence of a rectal laceration beyond the superficial mucosa, significant bleeding, a nonpalpable foreign body, or broken glass or other extremely fragile foreign material; (3) the need for any procedure that would cause undue patient discomfort; or (4) failure of emergency department extraction after a reasonable period of time. [20]

Observation in the emergency department to allow small nonpalpable foreign bodies (above the rectosigmoid junction) to pass into the lower rectum has occasionally been successful. Enemas or cathartics should not be used, because they may increase the impaction of a foreign body or cause it to move higher into the colon. [31] In the unobstructed patient, the use of oral gastrointestinal preparation solutions (e.g., GoLYTELY), may be considered if immediate surgery is not planned.

As noted above, admission to the hospital or outpatient surgical unit for removal under general anesthesia or for postremoval care may be recognized before or after attempts at foreign body removal in the emergency department. In general, any attempt at rectal foreign body removal in the emergency department is contraindicated in the following situations: (1) a patient with severe abdominal pain or signs of perforation on presentation; (2) a nonpalpable (high-lying) foreign body; (3) a foreign body that is unusually difficult to remove (i.e., routine removal could result in perforation or other significant morbidity); or (4) when there is insufficient time, experience, or equipment for the procedure.

**Equipment**

As in any procedure, a private, well-lit room with a comfortable examination table is needed. Equipment items that should be available for most extractions include lubricant, rubber gloves, examining light, anoscope, proctosigmoidoscope, local anesthetic with appropriate syringes and needles, and suitable parenteral narcotics and sedatives.

Other equipment that may be used depends entirely on the specific technique chosen for foreign body removal. Most techniques use some type of speculum to aid in visualization and then some form of clamp or snare to grasp the object. Some of the more common methods of foreign body removal are detailed later and are listed in Table 48-1 .

## Techniques

### Preparation

The general principles of management of rectal foreign bodies include proper diagnosis, subsequent removal, assessment for rectal or colonic injury, and recognition and treatment of associated injuries. The indications for radiographs before the extraction procedure are controversial. Some authorities advocate routine radiographs for all patients with rectal foreign bodies. Reports demonstrating the safety of outpatient management of uncomplicated foreign bodies suggest that such films increase the expense but add little to patient management. The decision to order films should be based on clinical judgment. Films are clearly indicated when the patient has abdominal pain, peritoneal signs, suspected perforation, or fever or when the patient's clinical history is unclear or unreliable. The setting of anticipated sharp foreign objects also warrants radiographic evaluation.

The patient may be placed prone or in the Sims position, depending on patient comfort. The knee-chest position can be used but is difficult for the patient to maintain without a proctosigmoidoscopy table with knee support.

Because patient discomfort is common during the procedure, sedation and analgesia should be used.  A combination of fentanyl and midazolam, as outlined in Chapter 35 , is a useful approach. Even with parenteral drugs, supplemental local anesthesia using a field block technique (see Fig. 48-9) may be necessary to achieve optimal dilation of the anal sphincter. The perianal field block is performed after povidone-iodine application. Anesthesia is placed in a radial fashion around the anal sphincter, using 0.5% bupivacaine or 1.0% lidocaine with epinephrine 1:200,000 (unless contraindicated). One then extends the anesthetic submucosally, anteriorly, and posteriorly. [34]

### Procedure

Gentle abdominal (suprapubic) pressure often helps deliver the foreign body into the distal rectum, where the examining

| TABLE 48-1 -- Equipment Useful for Rectal Foreign Body Removal | | |
| --- | --- | --- |
| **Speculum** | **Grasping Tool** | **Example of Foreign Body** |
| Operative anoscope | Tenaculum forceps | Cucumber |
| Operative proctoscope | Ring forceps | Banana |
| Parks retractor | Tonsil snare | Pencil |
| Deaver retractor | Obstetric forceps | Apple |
| Vaginal speculum | Spoons | Vibrator |
| | Foley catheter | Small ball |
| | Endotracheal tube | Glass jar |
| | Suction dart | Light bulb |

finger helps the distal end of the smooth foreign body navigate to the anus. Quite often the foreign body lodges against the sacrum posteriorly and requires gentle redirection.

If one is unsuccessful at digital extraction, then another technique must be chosen. The specific technique used depends on several factors, including the nature, size, shape, orientation, and composition of the foreign body; the availability of equipment; and the experience and ingenuity of the physician. Numerous techniques have been discussed in the literature; the more commonly accepted ones are described here. In general, most techniques require adequate visualization, as with a speculum, and the use of a

grasping tool.

An operative anoscope, proctoscope, or vaginal speculum may be inserted to allow adequate visualization. The object may then be grasped with a forceps or tenaculum and gentle traction applied to remove both the speculum and grasping clamp simultaneously, because the object often has a greater diameter than the speculum. A Parks retractor was used successfully to aid in foreign body removal in 1 series of 100 patients (Fig. 48-13 *A- C*). [34]

A flexible sigmoidoscope with a polypectomy snare has been used for successful foreign body removal. [35] After the tube is inserted, air is gently insufflated to distend the rectum. The object is then grasped with a polypectomy snare. Advantages of this technique include (1) the ability to distend the bowel around the object, (2) the avoidance of general anesthesia, (3) the convenience of outpatient management, and (4) the reported ability of this technique to retrieve high-lying objects.

Other grasping objects that have been used successfully include a suction dart for a glass foreign body, [36] a de Pezzer catheter attached to wall suction to produce a negative-pressure traction, [37] cyanoacrylate (Super-Glue), tonsil snares, surgical instruments such as obstetric forceps, and even spoons (Fig. 48-14) . A Sengstaken-Blakemore tube has been inserted into a hollow glass foreign body, inflated, and traction applied, allowing for successful removal. [25]

Glass foreign bodies deserve special mention. Glass may be quite difficult to grasp and, if fragile, may break during attempts at removal. In addition, glass often creates a vacuum effect in the distal colon when traction is applied, making routine removal virtually impossible. To overcome this effect, Foley catheters or endotracheal tubes can be inserted past the foreign body to insufflate air and release any suction forces. When the balloons are inflated, traction may be used to allow the foreign body to be removed (Figs. 48-15 (Figure Not Available) and 48-16) (Figure Not Available) . [38]

Difficult-to-grasp glass objects have been removed successfully with clamps covered with rubber. [39] Plaster of Paris has been used to fill a hollow object; the plaster is allowed to harden around a handle such as a wooden tongue blade, and then it is removed with traction. Some clinicians frown on using this technique, especially with glass objects, because as the plaster hardens, the heat released could cause the glass to break or produce mucosal injury.

The great majority of objects can be removed safely transanally in the emergency department.  After removal, proctosigmoidoscopy is recommended to evaluate for perforation, bleeding, and mucosal trauma. Patients with signs of perforation, unstable vital signs, abdominal pain, deep mucosal lacerations, significant bleeding, or suspicion

of perforation should be admitted. Perforation commonly occurs at the initial point of rectosigmoid angulation, approximately 15 cm from the anal verge. Perforation may occur prior to medical evaluation; thus, even atraumatic foreign body removals can be problematic. [5]

The emergency physician should set a realistic time limit, such as 30 minutes, at which time referral to a consultant for treatment or admission to the hospital becomes appropriate if extraction of the foreign body has been unsuccessful.

Once the patient is admitted, a high-lying foreign body may advance over 8 to 12 hours of observation to become a low-lying foreign body that can be removed transanally. If this is unsuccessful, then regional or general anesthesia is used along with abdominal and rectal palpation to maneuver the foreign body. If these attempts fail, then laparotomy should be performed, in which the surgeon "milks" the object in the colon distally. Colotomy is only rarely necessary and is done as a last resort. [10]

**Complications**

The most feared complication of rectal foreign bodies is bowel perforation. The risk of perforation is related to the force of object introduction and the sharpness of the object. The duration of foreign body impaction may also be a factor for large objects. Perforation may occur during insertion or removal of the object.

Perforation above the peritoneal reflection leads to free air below the diaphragm with signs of an acute abdomen. In suspicious cases, water-soluble contrast material may be used to clarify the diagnosis. [33] If perforation is below the peritoneal reflection, symptoms may be insidious, and patients may present with signs of pelvic abscess or sepsis days after the injury. It is therefore commonly recommended that patients treated for foreign body removal undergo proctosigmoidoscopy to rule out associated complications. Other complications that occur include bleeding, mucosal laceration, a torn anal sphincter, or object fragmentation with retention of the foreign body.

When intraperitoneal or retroperitoneal perforation is diagnosed or suspected, the emergency physician should institute appropriate antibiotic coverage. Bowel anaerobes are the primary contaminant; therefore, the choice of antibiotic should reflect an appropriate broad-spectrum drug. Currently accepted antibiotic regimens include ticarcillin-clavulanic acid, ampicillin-sulbactam, piperacillin-tazobactam, imipenem, cefoxitin or cefotetan, or, alternatively, metronidazole or clindamycin together with an aminoglycoside. [40]

**Conclusions**

The majority of rectal foreign bodies can be removed safely in the emergency department. "Body packers" and removal of this class of foreign bodies are discussed in Chapter 45 in conjunction with bowel decontamination. The emergency

physician must be knowledgeable in the evaluation of patients with rectal foreign bodies, the techniques for removal, and the potential for complications.

# RECTAL PROLAPSE (PROCIDENTIA)

Rectal prolapse, also known as *procidentia,* is a relatively uncommon condition in which some or all of the layers of the rectum protrude through the external anal sphincter. Definitive treatment of rectal prolapse is usually surgical, but the acute prolapse may be reduced in the emergency department. Failure to reduce the prolapse can result in long-term complications of atonic anal sphincters with resultant incontinence, or, at worst, actual gangrene of the bowel wall.

There are three types of rectal prolapse:

> *Type I prolapse,* or false procidentia, involves protrusion of mucosa only. It is often associated with hemorrhoids, and protrusion is only 1 to 3 cm (Fig. 48-17 (Figure Not Available) *A*).

> *Type II prolapse* is a true intussusception of all layers of the rectum, without an associated cul-de-sac sliding hernia (see Fig. 48-17 (Figure Not Available) *B*).

> *Type III prolapse* is a sliding hernia of the cul-de-sac. The pouch of Douglas is the hernia sac, pressing on the anterior wall of the rectum, forcing the rectal lumen to protrude through the anus. This condition gives the appearance that the rectum has been turned inside out (Fig. 48-18) .

The diagnosis of partial (mucosal) prolapse may be made by digital examination. Palpation of the prolapse between the examiner's gloved finger and thumb reveals the absence

**Figure 48-17** (Figure Not Available) *A*, Type I procidentia (rectal prolapse). *B*, Intussusception of sigmoid colon beyond the anus. *(From Kratzer GL, Demarest RJ: Office Management of Colon and Rectal Disease. Philadelphia, WB Saunders, 1985, pp 221-222.)*

**Figure 48-18** Complete rectal prolapse.

of a muscular wall. Normal anal sphincter tone also suggests mucosal prolapse.

Other entities that may mimic a prolapse include hemorrhoidal tissue mass or a large rectal or polypoid lesion protruding through the anus.

Rectal prolapse may be either congenital or acquired. It is often idiopathic in children, although it has a high incidence in association with pinworms, paraplegia, and cystic fibrosis. [10] In adults, rectal prolapse can be associated with mental retardation, organic brain syndrome, polio, stroke, and chronic psychiatric and neurologic conditions.

**Indications**

Acute prolapse should be reduced in the emergency department, although all patients

should be referred for subsequent surgical evaluation.

**Technique**

Reduction of Type I prolapse usually can be easily accomplished manually with gentle pressure on the mass. In children who are anxious, sedation may be necessary. With the child on the parent's lap, firm manual compression is applied for 5 to 15 minutes. After reduction, a pressure dressing is applied, and the child is sent home. A stool softener is recommended. The condition is usually self-limited. Increased bulk in the diet may help to decrease straining at the stool.

Reduction of a complete prolapse in an adult may be more difficult. Parenteral sedation and analgesia are usually required for success and are recommended. The patient is placed in the prone position, and the buttocks are taped apart (see Fig. 48-7) . Steady, gentle pressure to the area is begun, starting with the least prolapsed part (Fig. 48-19) . It may be helpful to place 2 gauze pads on the prolapse at the 3 o'clock and 9 o'clock positions, as the bowel wall is quite slippery. The thumbs are placed near the bowel lumen, and the fingers grasp the exterior wall. Then with thumb pressure, the sides are gently rolled inward to force the prolapse back through the anus. Reduction is most likely to be successful with slow, gentle pressure in a relaxed, nonstraining patient.

Definitive therapy of rectal prolapse in the adult is surgical. Patients with unsuccessful reductions need urgent surgical referral. Those patients successfully reduced should have subsequent surgical referral arranged. [10]

**Complications**

Complications include ulceration and occasionally bleeding. Inability to reduce the prolapse can result in gangrene of the bowel wall. Atony of the anal sphincters with resultant incontinence may occur, and the incidence increases with delay in reduction.

## CONCLUSIONS

Anorectal disorders are not a rare problem facing emergency physicians. Anoscopy, excision of thrombosed external hemorrhoids, removal of rectal foreign bodies, and reduction of rectal prolapse are all procedures that the emergency physician may be called on to perform.

# Chapter 49 - Out-of-Hospital Splinting

**Mohamud R. Daya, Ronald J. Mariani**

## CERVICAL SPINE

Trauma patients frequently sustain injuries to the cervical spine. These injuries are particularly devastating when associated with injury to the spinal cord. In the United States, approximately 12,000 spinal cord injuries are incurred per year. [1] Most of these are a result of motor vehicle crashes. The remainder are due to falls, recreational activities (especially diving), and penetrating trauma. It is estimated that 40% of cervical spine injuries are associated with neurologic deficits and that up to 25% of such deficits are a result of improper handling during evaluation, extrication, and transport of the patient. [2] [3] Therefore, it is critically important that the cervical spine be immobilized early and effectively.

Because the minimum degree of motion required to cause spinal cord injury has not been defined, the goal of immobilization is to maintain the head in a neutral position with zero degrees of motion in all directions. [4] To immobilize the entire cervical spine, an orthotic device must fix the head, hold the occiput and mandible, and restrict motion at the cervicothoracic junction. [5] This approach follows the basic orthopedic principle of immobilizing the joint above and below a suspected injury. In addition, to be useful in the out-of-hospital setting, a cervical immobilization device must be portable and easy to apply and must allow access to the upper airway.

At present, the standard technique of spinal immobilization involves early manual stabilization of the head and neck relative to the long axis of the body. Manual stabilization is usually followed by the application of a cervical collar. Although its use is controversial, a cervical collar has value as an early adjunct in the sometimes complex process of immobilization and extrication. There are several types of collars, all of which provide various degrees of comfort and support, but even the ones with the best features provide less than adequate immobilization when used independently.

The choice of devices used in addition to cervical collars depends on the victim's position of origin and the complexity of the disentanglement process. If the victim is found in a sitting position, as in an automobile, some version of the short spine board is commonly used. The most effective of these are wraparound corset-type devices, designed to immobilize the neck and place handles on the patient.

Following the use of these intermediate spine immobilizers, the patient is usually fastened to a full-length spine board or similar rigid device in preparation for transport. Complete cervical immobilization must also incorporate lateral stabilization of the head in the form of lightweight bulky objects such as foam blocks, towel rolls, blanket rolls, or cushions and tape. This can also be accomplished using factory-made devices such as the HeadBed (Laerdal Medical Corp, Wappingers Falls, NY) or Bashaw Cervical Immobilization Device (CID) (Bashaw Medical, Inc, Pensacola, Fla).

**Background**

Since 1965, an entire industry aimed at out-of-hospital preservation of life and limb has evolved. Specially manufactured spinal immobilizers were developed for field use during the late 1960s and early 1970s. Many variations of these early devices have been developed to solve specific out-of-hospital problems. In addition, recent advances have produced more versatile plastics and stronger adhesives.

It should be stressed that although there is widespread agreement about the basic steps of spinal immobilization, out-of-hospital care is a setting only for people who can adapt. Because of the variety of circumstances that confront rescuers on a daily basis, flexibility is mandatory.

A pioneer in the use of a spine board for cervical immobilization after injury was J. D. Farrington. [6] Farrington emphasized that spinal fractures were frequently mishandled and made worse by rough or hasty movement at the scene. He outlined the use of a backboard, sandbags, and tape for the out-of-hospital extrication and care of patients with suspected spinal injuries. He fashioned extrication collars using universal dressings held in place by soft roller bandages and advocated manual traction during the extrication phase. [7]

Manual traction is no longer recommended, because it may aggravate an underlying spinal injury. [8] In addition, although sandbags are effective devices for lateral immobilization, they may cause significant movement of the neck if the board is suddenly tilted (e.g., to decrease the risk of aspiration in a vomiting patient). As a result, sandbags have been replaced by more lightweight devices such as foam blocks, the HeadBed or Bashaw CID. Despite these modifications, the original Farrington method of splinting the head and torso to a rigid object remains the preferred technique for effective spinal immobilization.

**Indications**

An extrication collar should be used as a primary adjunct in cases involving trauma to the head and neck. In the absence of visible trauma, patients should be immobilized whenever the mechanism for a potential cervical spine injury exists. The most common mechanism of injury involves sudden deceleration of an automobile, resulting in hyperflexion and hyperextension forces. Patients under the influence of alcohol or drugs lack the self-awareness to recognize their own spinal injury and should be immobilized routinely. Likewise, every unconscious trauma patient should be immobilized to avoid aggravating an underlying spinal injury. Any awake and alert trauma patient who complains of spine pain, paresthesia, weakness, or absent movement should be immobilized carefully to avoid secondary injury to the spinal cord.

It is not uncommon to encounter patients in the field who are both conscious and oriented but unaware of their own neck injuries. The sensory impact of having one's automobile forcibly disassembled by rescue personnel who are using modern tools that

produce noise in excess of 80 dB may result in considerable distraction. In addition, the presence of other painful injuries or concern over other victims can easily mask the manifestations of an occult cervical spine injury.

Serious cervical cord injuries can also occur in the absence of demonstrable fractures. Spinal cord injury is common in elderly patients with cervical spondylosis, in whom an arthritic osteophyte may sever a portion of the cord as permanently as a fracture or dislocation. In such cases, there is often little or no subjective pain, and the mechanism of injury may appear seemingly minor. [9]

Therefore, a high index of suspicion for possible spinal injury must be maintained at all times. In all cases, the medical record must include documentation of the neurologic examination before and after spinal immobilization.

The purpose of an extrication collar is to assist in splinting the head and neck either therapeutically or prophylactically in a neutral position. [5] The collar is useful for the following reasons:

1. It provides airway protection by limiting flexion in a patient whose unsupported jaw and neck position threatens patency.
2. It helps reduce cervical spine motion, especially flexion, but also rotation, lateral bending, and extension. In this regard, however, it serves only as an adjunct.
3. If properly chosen, it can support the weight of the head while the patient is sitting and help to maintain the alignment of the cervical spine once the patient has been moved to a supine position.
4. An equally important function is to serve as a reminder to the patient and rescuers that the integrity of the basilar skull and cervical spine are suspect because of the mechanism of injury.

The cervical collar does not provide complete immobilization of the head and neck. The collar was designed as an adjunct and was never intended to provide definitive immobilization in itself. Complete immobilization is not possible until the patient is properly secured to a long backboard-type device. Nonetheless, the collar should go on first and remain in place during the entire procedure.

**Contraindications**

There are few circumstances that preclude the use of an extrication collar. The presence of a surgical airway (cricothyroidotomy or tracheostomy) may require modification of the cervical support technique.

In addition, cervical dislocation with fixed angulation may prevent effective application of factory-made collars. This situation is rarely encountered and can be managed with an improvised cervical immobilizer, such as a version of a horse collar or prolonged manual positioning without traction. [10]

A third circumstance that could preclude the use of a collar is massive cervical swelling

(e.g., secondary to hemorrhage or tracheal injury). In these circumstances the compressive effect of a collar may impede air exchange, decrease cerebral perfusion, or increase intracranial pressure. [11]

Finally, the presence of an impaled foreign body such as a knife, a piece of glass, or metal can also make cervical spine immobilization using extrication collars difficult.

Note that improvised cervical support may work better than a manufactured collar, depending on the size and shape of the patient. This is particularly true in the pediatric population, as collars come in limited sizes and may not be well tolerated. In such circumstances, prolonged manual in-line stabilization is often necessary. Adaptability is a trait of well-trained and experienced field personnel.

## Equipment

Traditionally, cervical collars have used 4-point support structures for the bottom of the collar: namely, at the 2 trapezius muscles posteriorly and at the 2 clavicles anteriorly (Fig. 49-1) (Figure Not Available) . Newer rescue collars also use the sternum as a fifth support structure (Fig. 49-2) (Figure Not Available) . Current collar designs support the head with wing-like flaps on the collar's upper posterior edges. Anteriorly, the collar supports the mandible. The collar's flaring design generally prevents compression of the thyroid cartilage and cervical vessels, even when the collars are applied firmly.

Soft collars, although comfortable, have no role in spinal immobilization, because they provide minimal support and do not reduce cervical motion to any significant degree. [3] [12] However, the semirigid collar must be comfortable to ensure patient compliance. Dick and Land described the subjective characteristics of an "ideal collar." [13] These features included the following:

1. It should support the weight of the head in a neutral position.
2. It should prevent lateral, rotational, and anteroposterior movement of the head.
3. It should be comfortable, translucent on radiographs, and compact.
4. It should be easy to apply.
5. Its price should be such that it can be carried in sufficient numbers in various sizes by any ambulance.
6. It should not interfere with the position or function of important airway structures, nor adversely affect cerebral circulation in any way.
7. Simplicity of design should permit its application by 2 rescuers in less than 60 seconds, in darkness, rain, or cold weather, without manipulation of the head or neck.
8. It should be available in the smallest number of sizes possible.

Other investigators have attempted to evaluate cervical collars in an objective fashion. The accepted gold standard for comparison is the halo brace, which restricts motion to 4% flexion-extension, 1% rotation, and 4% lateral bending. [5] A number of studies have evaluated neck motion in volunteers immobilized supine on a backboard with various collars in place. Overall, these studies merely confirm the fact that cervical collars alone are inadequate to immobilize the cervical spine completely. At the present time, there is

no out-of-hospital device or technique that approaches the halo in its ability to immobilize the cervical spine. [5]

Little information is available regarding the proper selection and application of spinal immobilization devices for children. Half of the total growth in head circumference is achieved by the age of 18 months, giving children a disproportionately large head compared with the rest of the body. Prior to age 8 years, these anatomic and developmental differences result in a higher incidence of upper cervical spine injuries (C1-2). Because injuries in this area are frequently unstable, proper cervical immobilization in the neutral position is critically important. Because the head is large, positioning the child's body on a standard backboard may force the neck into flexion or a relative kyphosis. The clinical significance of this is currently unclear, but theoretically, adult-type immobilization devices may be hazardous for use in young children. The standard backboard may be modified to adapt to the child's larger head size. As a rough guide, the external auditory meatus should be on the same level as the mid-shoulder. Suggested modifications include a cutout in the backboard that accommodates the occiput or a pad under the back at the level of the chest (Fig. 49-3) (Figure Not Available) . If not modified, the standard backboard in conjunction with the disproportionately large head of a child may force the neck into hyperflexion, possibly aggravating a cervical spine injury. [20] Nypaver and Treloar showed that all children required elevation of the back (mean height, 25.4 ± 6.7 mm) for correct neutral position on a spine board. Children <4 years old required more elevation than those 4 years old. [21]

In the only study addressing pediatric cervical spine immobilization, Huerta and coworkers evaluated the performance of infant and pediatric collars using a mannequin model. [22] They concluded that the high-cut collar (Fig. 49-4) (Figure Not Available) in conjunction with a rigid backboard and lateral neck stabilizers provided the most effective spinal immobilization. In some children, these devices may provoke struggling because of fear, which has the potential to aggravate an underlying injury. In these situations, manual immobilization may be more effective and less frightening for the child.

**Procedure**

Application of an extrication collar is a straightforward procedure (Fig. 49-5) (Figure Not Available) . A collar should be treated as a splint. The normal axiom in splinting is to immobilize the joint above and below the area of injury. Because no collar performs this function perfectly, a rescuer should be charged with maintaining manual cervical stabilization in the neutral position during collar application and until the patient can be fully immobilized in a corset-type device or on a full backboard. The rescuer's intentions should be thoroughly explained to the patient throughout the procedure.

The neck should be examined before application of the collar for swelling, ecchymosis, deformity, or penetrating wounds. Once the collar is in place, a conscious patient should be cautioned repeatedly against movement of the head. Any persistent complaints of pain or dyspnea by the patient should be investigated by removal and possible replacement of the device while manual stabilization is maintained. The collar size

should be determined using the manufacturer's suggested guidelines. For example, the Stifneck collar (Laerdal Medical Corp, Wappingers Falls, NY) is available in various sizes and uses the distance from the top of the shoulder to the chin to determine size (Fig. 49-6) (Figure Not Available) . The tallest collar that does not cause hyperextension should be used. For extremely short necks, a special extrication collar such as the No-Neck by Stifneck is recommended (Fig. 49-7) (Figure Not Available) .

In cases in which an extrication collar of the proper size is not available, an improvised device should be made from available materials (Fig. 49-8) (Figure Not Available) . Once the patient is in the supine position on the backboard, lateral stabilization should be added, using foam blocks and tape or a factory-made lateral neck stabilizer (Fig. 49-9) (Figure Not Available) . Because immobilization on a flat backboard has been shown to place most adult patients into relative cervical extension, it is recommended that occipital padding (mean, 3.8 cm) be added to restore neutral position in adults. [24]

## Complications

Improper application of an extrication collar can occur if the wrong size is used or too little care is exercised during placement. The best means of preventing either error is strong physician involvement in the training and continuing education of rescue crews, with vigorous feedback in cases of correct and incorrect application.

A collar that is too small for a patient may be either too tight for the girth of the neck (with obvious complications) or too short to provide adequate immobilization. Too large a collar commonly results in hyperextension, which can exacerbate a pre-existing spinal injury.

Improper or prolonged application of an extrication collar may impede venous return and raise intracranial pressure. This is sometimes manifested by facial flushing and is more common with the short collar types. [11] The clinical significance of the elevation of intracranial pressure produced by cervical immobilization is unknown; a collar was found to elevate intracranial pressure by only 2 cm $H_2O$ in 1 study. [25]

The long-term use of the Philadelphia extrication collar as part of the treatment plan for an underlying cervical spine injury has been associated with pressure ulcers of the scalp. [26] Because some collars (e.g., Philadelphia [Philadelphia Cervical Collar Co, Westville, NJ] and Stifneck) have been shown to exert higher capillary closing pressures at contact points, it is suggested that collars with favorable skin pressure patterns and superior patient comfort (e.g., NECLOC, Jerome Medical, Moorestown, NJ) be used in these settings.

One final complication should be mentioned. The patient who, for whatever reason, actively resists placement of an extrication collar or other splint should not be forced to wear it. Immobilization of the resisting patient cannot be accomplished without considerable muscular exertion not only by rescuers but also by the patient. If fractures do exist, it is possible that this kind of struggling can cause further damage. If the patient

permits manual stabilization, this should be maintained as an alternative.

**Summary**

The use of a good cervical collar is an important first step in the immobilization of patients with potential cervical spine injuries. The use of a cervical collar does not constitute complete immobilization. Full support must be provided by means of a full backboard and lateral support for immobilization of the head and neck.

# THORACOLUMBAR SPINE

Adequate full-body thoracolumbar spinal immobilization is probably best done by means of a full-length spine board (also called a *backboard*). Full-body spinal immobilization should include early application of a cervical collar, lateral immobilization of the head and neck, and ample belting of the entire body to the backboard. Proper belting minimizes movement whenever the backboard is used to transport the patient over long distances. In addition, this will help limit spinal movement associated with backboard tilting, which may be necessary in the likelihood of emesis or during transport of pregnant females in the second and third trimester.  Transferring a victim from a location and position of origin to a backboard may require the use of an intermediate spine immobilizer. This can be done using a short spine board or a corset-type device. Corset-type devices have extensions that engage the head and neck and are equipped with weight-bearing loops that allow easier handling of the patient. Intermediate immobilizers or extrication splints should be used when a patient must be removed from a confined environment or when circumstances require movement in or from a sitting position.

In some circumstances, a threatening environment (fire, hazardous material incident, extreme weather) or patient condition (compromised airway, shock) may necessitate rapid extrication using a manual technique. Rapid manual extrication may also shorten on-scene times and has been encouraged by out-of-hospital trauma care courses. [1]

When extrication is not required by a patient's location, position of origin, or route of egress, the patient is most often found lying at ground level. With an extrication collar in place and in-line manual cervical immobilization, the patient can be logrolled onto a backboard. Visual inspection of the back should be carried out during the logrolling process while the body is kept in a single plane.

McGuire and colleagues evaluated the safety of the logroll maneuver in three different settings: a volunteer with a stable spine, a cadaver with a surgically created unstable thoracolumbar injury, and a patient with a T12-L1 fracture-dislocation. [27] Radiographic studies on the volunteer demonstrated mild lateral movement during logrolling. The cadaver demonstrated a 2.1-cm anteroposterior displacement and a 5-mm lateral displacement during the logroll. The injured patient demonstrated no anteroposterior displacement but did experience a 7-mm lateral displacement when logrolled onto the side. Suter and coworkers studied variations of the logroll maneuver in healthy volunteers to determine the effect of axial traction and subject arm positioning on

thoracolumbar movement. [28] Their study demonstrated significant thoracolumbar movement during the logroll maneuver that was consistently minimized by positioning subjects with the arms extended at the sides and the palms resting on the thighs. These studies raise concern about the safety of the logroll maneuver and suggest that extreme caution be used during transfer of patients with suspected spinal injuries.

An optional but effective means of moving a supine patient is provided by a type of stretcher that breaks apart longitudinally and can be slid beneath a victim without disturbing their position. The halves of this scoop stretcher are anatomically contoured to enhance comfort and limit lateral movement of the immobilized patient. Unfortunately, visual inspection of the back is not possible with the use of a scoop stretcher.

A third means of both immobilizing and moving a trauma victim consists of a specially designed full-body splint, complete with factory-made straps or harnesses. There are several such immobilizers, most of which are highly effective and provide good lateral stability as well as anatomic conformity. One such device that is popular in European countries is the vacuum stretcher. A full-body splint that has recently been marketed in the United States (Evac-U-Splint [Hartwell Medical, Carlsbad, Calif]) offers fast, full-body immobilization that supports the entire patient without creating pressure points (Fig. 49-10) (Figure Not Available) . In general, the patient must either be logrolled or lifted, using a scoop stretcher or full backboard, onto one of these devices.

## Background

As recently as 1965, the principle of "rapid transportation above all" held widespread acceptance among rescuers, who had little or no orthopedic training, and among physicians whose emergency care experience, by modern standards, was just as limited. The most commonly agreed-on means of getting a sitting patient out of a wrecked automobile was to use some version of a chair-carry. If the patient originated in a position other than the sitting position, the patient was first placed into a sitting position and *then* moved by means of a chair-carry or simply dragged out of the vehicle.

Col. Louis Kossuth, commander of the U.S. Air Force's Medical Service School at Gunter Air Force Base in Alabama, made note of several automobile crashes in which he thought patients were handled roughly by bystanders who were trying to help. [29] Finding the medical literature at that time lacking, he made recommendations regarding how these victims should be handled. He experimented with a set of canvas splints reinforced with semirigid steel stays as slats, similar to the modern Kendrick extrication device (KED) (Ferno Model 125, Ferno-Washington, Inc, Wilmington, Ohio). In addition, Kossuth probably developed the first modern-type spine board. [7]

In 1967 and 1968, Farrington wrote 2 classic articles that showed the use of an extrication collar, spinal traction, 9-foot webbing straps, and both short and long spine boards to remove people in every conceivable position from automobiles. [6] [7] Much of today's extrication theory is essentially identical to what was taught by Farrington.

In 1967, the Committee on Trauma of the American College of Surgeons listed the minimum amount and type of equipment that should be carried in ambulances. This list

included both short and long spine boards, with accessories. [30] A similar document published the following year by the National Academy of Science also provided a list of medical requirements for ambulance equipment and recommended short and long spine boards. [31]

In 1969, St. Louis surgeons Klippel and Conrad described the use of a full-length spine board that was capable of being broken down into separate components for the upper and lower halves of the body. The lower portion included a simple traction device that could be used to simultaneously splint a fractured femur of either lower extremity. [32]

## Indications and Contraindications

Any mechanism capable of causing injury to the cervical spine should prompt rescuers to immobilize not only the head and neck, but also the entire body. The motion of any vertebral joint is impossible to isolate. For complete spinal immobilization, the head, neck, and torso must be fastened into a single common plane. Extrication devices (e.g., short spine boards) will accomplish this to some extent, although movement of nonimmobilized lower extremities can lead to secondary movement of the pelvis and lumbar spine. Movement of the lumbar spine may also induce thoracic movement to some extent. Considering the fact that the most feasible position for transport is the supine position, fullbody

**Figure 49-10** (Figure Not Available) The Evac-U-Splint mattress. *(Courtesy of Hartwell Medical, Carlsbad, Calif.)*

immobilization is best achieved using the long spine board.

Mechanisms that arouse suspicion of injury to the thoracolumbar spine should also prompt full-body spinal immobilization. These include penetrating and blunt injuries to the thorax, abdomen, pelvis, and spine. Full-body immobilization should be considered whenever the mechanism for spinal injury exists, even in the absence of signs and symptoms. In such cases, the possibility of occult injury is best ruled out through clinical and/or radiographic examination at the hospital.

The only contraindication to full-body immobilization of a patient whose mechanism of injury suggests spinal injury is *the existence of a greater threat*. The threat to a patient's life may exceed the threat of *possible* spinal injury under the following circumstances:

> *Hazards on the scene.* Problems with traffic control in the direct vicinity of the patient's location such that the patient or rescuers are likely to be injured. Such hazardous situations include leaks of fuel or other flammable substances, fire, hostile crowds, partially collapsed structures, or unstable vehicles.

> *Ongoing gunfire at the scene.* Gunfire is always considered an indication for rescue personnel to stay away from a scene.

> *Overwhelming casualties.* Rescuers may have to improvise in cases in which

casualties exceed available resources. In such cases, proper spinal immobilization may merit a lower priority than usual.

*Weather extremes.* Under conditions of extremely adverse weather, the urgency to move the victim may supersede the priority of normal treatment, including immobilization.

*Patient noncompliance.* A competent rescuer can do much to make an immobilization device comfortable by means of padding and reassurance. If this fails, immobilization that is applied by force may cause more harm than no immobilization at all.

## Equipment

### Cervical Extrication Splints

There are a large variety of short spine boards available for out-of-hospital use (Fig. 49-11) (Figure Not Available) . Although these devices have not been compared scientifically, some useful and important features of cervical extrication splints have been identified.

The device should not produce jostling or change the position of the head, neck, shoulders, or torso during application. In conjunction with a good cervical collar, a properly applied cervical extrication splint should effectively limit lateral, flexion, extension, and rotational motion of the head, neck, and torso. A cervical extrication splint should be comfortable, since it may be left on a patient for prolonged periods. Simplicity of design should allow rapid placement of the device by 2 rescuers in any situation, without causing movement of the head, neck, or torso.

The cervical extrication splint should be compact enough to allow its use in many types of extrication situations. Cost should be reasonable, so that at least 1 device can be carried in every ambulance. Ideally, the device should also be translucent so that radiographs can be readily obtained in the emergency department (ED). Lastly, the cervical extrication splint should be designed to allow for repeated sanitation.

One commonly used device that meets all of these criteria is the KED (Fig. 49-12) (Figure Not Available) . The KED was developed by Rick Kendrick, an emergency medical technician (EMT)-firefighter from El Cajon, Calif, as a result of the frustration of rescuers who experienced difficulty when removing victims from wrecked race cars. The device consists of 2 layers of nylon mesh impregnated with plastic and sewn over plywood slats to provide rigidity. It has a nylon loop behind the patient's head that is continuous with the pelvic support straps for additional strength. Part of its anterior thoracic panels can be folded backward to fit the obese, pregnant, or pediatric patient. Properly applied, the KED is a snug-fitting, highly adaptable immobilizer that can be used under even the most adverse of circumstances.

**Full-Body Spinal Immobilizers**

There are 3 basic classes of full-body spinal immobilizers, each with its own advantages and disadvantages. [33] It should be stressed that it is more important for a rescuer to achieve results than to be particular about using a specific piece of equipment.

**Full-body spine boards (backboards).**

Backboards are made of wood or plastic and can be either rectangular or tapered in shape (Fig. 49-13) (Figure Not Available) . Most rescuers prefer the tapered type, because it takes up less horizontal room when angled into a narrow doorway (such as that of an automobile). In addition, the slight narrowing of these boards on either end enhances the effectiveness of strapping.

Many boards also feature runners, usually about 2.5 cm thick, on their underside that serve both as stiffeners and as spacers. These raise the board slightly off the ground so that rescuers can get their fingers under the board during lifting. The runners, however, may make it more difficult to slide a patient onto the board.

Advantages of boards over full-body splints include their ease of storage, low cost, and extreme versatility. The backboard can be used to slide a victim out of an automobile or to protect a victim during removal of a windshield. Boards provide insulation against electrical hazards and can be used as improvised shelter in bad weather. Finally, they may be useful as ramps in muddy conditions.

Disadvantages of boards as immobilizers, although few, deserve mention. Board-like splints, as a class, are the least comfortable of all immobilizers. One prospective study demonstrated that standard spinal immobilization (hard backboard, rigid cervical collar, lateral immobilization device) of healthy volunteers, was associated with a variety of symptoms, including headache, backache, and jaw pain. [34] These findings may have clinical implications, since pain generated by the application of a backboard can be difficult to separate from other sources of pain in the trauma patient. Discomfort may be minimized by using padding at points of contact between a bony prominence and the board or the cervical collar. Alternatively, patient discomfort can be reduced, without compromising cervical spine immobilization, with the use of a pad (1.3 cm closed-cell foam padding) along the entire board. [35]

Backboards have also been shown to produce higher sacral interface pressure than a conforming support surface such as the vacuum-type splint. [36] The duration of contact pressure and the degree of contact pressure are considered important determinants of pressure necrosis in spinal cord injuries. [37] A further disadvantage of the backboard is its slippery surface, which makes it an unstable carrying device in rough terrain.

**Scoop stretchers.**

If a trauma victim has to be slid out of a tight location, a smooth backboard is probably

the best immobilizer. If the victim is not in a tight location, the scoop stretcher is an ideal field immobilizer (Fig. 49-14) (Figure Not Available) . The scoop stretcher is comfortable, rigid, adaptable to patients of various lengths, and provides unobstructed radiographic transparency of the entire spine. If necessary, it can be almost instantly applied or removed without disturbing the position of the victim. The stretcher also provides good lateral stability due to the trough-like shape of its top surface, and it is stable enough to be used for carrying purposes. For optimal protection of a potential spinal injury, the scoop stretcher should be placed on a backboard before moving the patient.

The scoop interferes slightly with the ischial section of a half-ring traction splint but works well with Sager-type devices. The scoop has no adverse effect on other immobilizers and does not interfere with cardiopulmonary resuscitation (CPR). The Ferno-Washington model 65 scoop (Ferno-Washington, Inc, Wilmington, Ohio) is the most widely used stretcher of this type.

**Full-body splints.**

There are various devices that take the concept of full-body immobilization 1 step further than the spine board. Los Angeles County Fire Department paramedic Larry Miller designed a narrow spine board shaped like the human body, with handles on both long edges and a system of harnesses to provide immobilization. The Miller body splint consists of a polyethylene shell injected with a closed-cell foam that is radiographically translucent and provides buoyancy in water (Fig. 49-15) (Figure Not Available) . The full-body splint features a removable head harness, a thoracic harness, and pelvic as well as lower extremity belts. The space between its lower extremities facilitates wrapping with bandage material in the event of fractures. In addition, it is shaped so that it can easily fit into a basket-type rescue stretcher such as the Stokes.

A recent innovation in the area of spinal immobilization in the United States is the vacuum mattress splint (see Fig. 49-10) (Figure Not Available) . It consists of a vinyl-coated polyester envelope filled with thousands of 1.1-mm diameter polyester foam spheres. A manual or electric vacuum pump is used to evacuate the interior to a pressure of about one-fourth atmosphere. This reduction in internal pressure causes the mattress to conform to the contours of the patient's body. Vacuum splints are more comfortable, quicker to apply, and allow less slippage on lateral tilting than wooden spine boards. [38] [39] The vacuum splint has also been shown to produce lower sacral interface pressure and may be the preferred immobilization device in patients with spinal cord injuries. [36]

**Lateral neck stabilizers.**

Sandbags, which were used as lateral neck stabilizers for years, are no longer recommended for use in the field. Lighter objects such as blocks (10 cm × 10 cm × 15 cm) made of medium-density foam rubber are commonly used. Foam blocks are lightweight, inexpensive, and disposable and do not slip on the backboard. More recently, cardboard devices that have the same advantages as foam blocks have been

developed to provide lateral stabilization (Fig. 49-16) (Figure Not Available) .

Another commercial device is the Bashaw CID. It is a lateral neck stabilizer designed to quickly and easily fasten the patient's head to a scoop stretcher or spine board (Fig. 49-17) (Figure Not Available) . The CID is made of a Herculite nylon and polyethylene foam platform that is fastened to the stretcher, either by elastic belts or by nonelastic belts with buckled closures. Its pillows are then attached to the nylon platform by means of large Velcro interfaces. A similar device called the Universal Head Immobilizer is also manufactured by Ferno (Fig. 49-18) (Figure Not Available) .  Mosesso and colleagues compared 6 out-of-hospital cervical immobilization devices and concluded that the devices were similar in their ability to immobilize the cervical spine, although the Bashaw CID was less effective than the other 5 devices in limiting rotational movement. [40]

## Procedure

Despite the presence of a field cervical collar, manual cervical stabilization should be continued until the patient is fully immobilized in either a cervical extrication splint or a full-body splint (e.g., a backboard or vacuum stretcher). The splinting technique used will depend on the patient's position of origin.

### Sitting Position

The extrication splint should be stored so that its straps are secured in their individual retainers to reduce their likelihood of becoming entangled during application. At least 2 rescuers should be used to apply an extrication splint to a sitting patient.

When used, the device is opened, butterfly style, and gently slid behind the victim with a rocking motion. If necessary, the patient can be very carefully rocked forward a few degrees to facilitate placement of the splint.

Once behind the victim, the splint's pelvic support straps should be freed from their retainers and allowed to dangle at the patient's sides. Next, the lateral thoracic panels are brought around the chest just beneath the patient's shoulders. While grasping these panels, a rescuer *slides the splint upward until the top edges of the panels firmly engage the patient's axillae*.

Now the thoracic straps can be used to secure the splint, beginning with the middle strap, then the bottom strap, and finally the top strap. The straps should be fastened snugly but not so tightly as to interfere with respiration.

The pelvic support straps are fastened next. They can be slipped, 1 at a time, beneath the patient's lower extremities and *brought directly beneath the pelvis using a back-and-forth motion*. If the pelvic straps are not applied properly, they allow considerable slippage when the patient is lifted. The free end of each of these straps mates with a buckle located at the patient's hip on the outside of the splint. Once a strap is ready to be buckled, it can either be attached to the buckle on its own side or moved

across the patient's lap and engaged with the opposite buckle. The latter method is preferred by most people, because it allows the patient's knees to remain together without discomfort to the patient.

Next, the head is fastened. When using the KED, the head panels are wrapped snugly around the head and neck by 1 rescuer while another rescuer applies the diagonal head straps. It may be necessary to place padding behind the head to maintain a neutral position. The forehead can be used as a point of engagement for 1 strap, and the cervical collar itself can be used for the other.

Finally, all buckles should be tightened until the entire splint is firmly in place, while avoiding respiratory discomfort. The patient can now be moved. If the patient is to be lifted from a vehicle, the ambulance cot, with a spine board on it, should be brought as close as possible. While 1 rescuer supports the patient's knees, the other rescuer uses the handholds on the splint to lift the patient. The patient should be rotated and laid in a supine position onto a backboard.

The pelvic straps should then be loosened to allow the legs to be lowered onto the backboard. The legs can then be extended and secured to the backboard or left in the flexed position with a pillow placed under the knees for support.

Some type of lateral immobilizer should be applied for the head and neck, and the body should be belted into place on the board. Once the patient is on the board, the thoracic straps of the cervical extrication splint should be loosened.

**Recumbent Position**

A patient who is found in a recumbent position should be placed in a supine position, if not already in one. If repositioning is necessary, the back should be examined in the process. Physical examination, spinal immobilization, airway management, and transport are easier to accomplish with the patient in the supine position.

Patients who are found supine do not require the use of a cervical extrication splint. They should, however, receive initial manual cervical stabilization and an extrication collar. After that, they are usually fastened to a full-body spinal immobilizer, such as a scoop stretcher, a backboard, or a full-body splint.

**Scoop stretcher.**

A patient who is in a supine position can be moved by means of a scoop stretcher. In the conscious patient, rescuers should explain to the patient that they are about to apply a scoop-type stretcher, which may be cold to the touch, beneath his or her body. An extrication collar is applied, and manual cervical stabilization is maintained until the patient is completely secured to the stretcher. Another rescuer places the scoop on the ground next to the patient and opens the latches that regulate its length. The length should be adjusted so that the scoop stretcher fits the full length of the patient's body.

The latches that regulate the length of the device should then be engaged.

Next, the latches at each end should be released, allowing rescuers to separate the stretcher into 2 halves. Each half is then placed next to the patient. One rescuer then gently pushes half the stretcher under 1 side of the patient. In some cases it may be necessary to have another rescuer rock the patient to allow proper positioning. The same procedure takes place with the opposite half of the scoop until both halves are aligned beneath the patient. The latch at the head of the device should be engaged first. The lower end of the stretcher is then brought together, and the foot latch is engaged to complete the integrity of the stretcher. The patient's torso should be strapped into place and the head immobilized using a suitable lateral neck stabilizer. The patient can then be lifted onto another device (e.g., Stokes stretcher or backboard) for transport. After placement on another device, the scoop stretcher can be removed without disturbing the patient's position, if necessary.

**Full-body spine boards (backboards).**

There are several ways of placing a patient onto a spine board. The precise technique used will depend on the space available and the position of the patient within that space.

For *lengthwise extrication*, as from an automobile seat, the patient can be slid, either feet first or head first, onto the backboard. It is important that the patient be moved as a unit during this process.

The end of the backboard should first be placed on the seat or doorsill of the automobile. One rescuer should then stabilize and maintain the backboard level at its opposite end, while other rescuers (at least 2) lift and slide the

body onto the board. Manual cervical stabilization should be maintained throughout the procedure, and rescuers should avoid spinal compression or traction. Once the patient is completely on the board, the board can be slid out and placed on a waiting stretcher.

When space permits, *lateral extraction* is preferred. With the patient in the recumbent position, rescuers may logroll or slide the patient onto the board. As mentioned previously, extreme caution should be used with the logroll maneuver. The logroll maneuver requires the presence of at least 3 rescuers. One rescuer is positioned at the patient's head and applies manual cervical stabilization. It will be this person's responsibility to oversee and direct body movement throughout the procedure. The backboard is then positioned next to the body. To minimize thoracolumbar movement, the patient's arms should be extended at the sides with the palms resting on the lateral thighs. [28] If 1 arm is injured, the backboard should be placed against this side, so that the patient can be rolled onto the uninjured extremity. The other rescuers should be positioned on the side that the patient will be rolled *toward,* with 1 rescuer at the midchest and the other at the legs. The rescuer at the chest should reach across the victim, taking hold of the shoulder and hips, while the other rescuer grasps the hips and lower legs. When everyone is ready, the rescuer at the head gives the command to roll the patient. The patient's back should be examined at this point. The backboard is then

slid under the patient, and when everyone is ready, the rescuer at the head gives the command to lower the patient onto the board. The patient should then be centered and securely strapped to the board.

Alternatively, during lateral extraction, a recumbent patient can be slid sideways onto the spine board. This improvised technique also requires the presence of 3 or 4 rescuers, 1 of whom can maintain control of the patient's head and neck.

Various techniques can be used to secure a patent onto the backboard. The effectiveness of 4 different strapping techniques in reducing lateral motion of volunteers restrained on a backboard was studied by Mazolewski and Manix. [41] These investigators found that the addition of an abdominal strap significantly reduced lateral motion without compromising respiration. Aside from this study, there is little scientific basis for the various strapping techniques, and in many cases the methods used are determined by local protocol.

When strapping a patient to the board, the first step is to secure the body in place. One method involves using 2 straps crossed over the chest to secure the thorax in place. This can be augmented by a third strap placed just under each axillae and across the chest. Additional straps are then placed at the hips and lower legs (Fig. 49-19) . After the body has been strapped to the board, the head can be secured. If necessary, padding should be placed under the occiput to maintain the head in neutral position. A lateral neck stabilizer (e.g., foam blocks or a Headbed device) is then applied and the head secured in place using tape or straps. Most taping techniques involve the use of 1 piece across the forehead and 1 piece across the cervical collar. Note that this method of securing a patient to a backboard is designed for horizontal lifting only.

### Standing Position

The standing patient with a potential spine injury must be immobilized and placed in the supine position. One technique for placing these patients on a backboard that is quick, safe, and effective is presented here (Fig. 49-20) (Figure Not Available) . [42] The tallest rescuer should be positioned behind the patient to manually stabilize the head while a second rescuer applies an extrication collar. The first rescuer must maintain manual cervical stabilization until the patient is completely secured to the board. The backboard should be centered behind the patient between the arms of the rescuer who is stabilizing the neck. Facing the patient, 1 rescuer on each side should each reach under the patient's arm and grab the backboard by a handhold at or above the patient's axillae. The patient's elbows are then brought closer to the body. If an additional rescuer is available, this rescuer should be positioned at the feet to prevent the board from sliding out, particularly on slippery surfaces. The patient should be slowly tilted back by lowering the head of the backboard. The rescuer at the head should step back during this process while maintaining the patient's head and neck in neutral alignment. When the backboard is completely horizontal, the patient can be secured to the backboard in the normal fashion.

## Complications

In general, complications are more likely to occur as a result of failure to immobilize spinal injuries before movement than from the technique of immobilization. When complications do arise, they may be related to improper choice or use of equipment. The wide variation of situations that are encountered routinely in the field call for a common-sense approach to spinal immobilization.

Victims are generally belted in place on a spine board to prevent sliding during transport. If too few straps are used or if the straps are loosely applied, motion during transport can occur. Patients who are strapped too firmly in place may complain of extreme discomfort and even panic. Excessive strapping can interfere with respiratory function in both children and adults.

Once strapped into place, an unresponsive patient who vomits should be protected from aspiration. The traditional means is to logroll the board and patient as a unit to the side. Although this procedure may be associated with some spinal movement, the airway takes higher priority.

**Summary**

The wide variety of circumstances in which a traumatized patient is likely to be involved mandates the need for many approaches to full-body spinal immobilization. The overall goal of rescuers is to fasten the victim to a board-like full-body immobilizer. After early application of an effective cervical *collar*, this process involves the following steps:

1. Application of a cervical *extrication splint* that serves to immobilize the cervical and thoracic spine. This sort of device is often used when the patient is first encountered in a confined environment, such as a wrecked automobile or bathtub.
2. Placement of the patient on a full-body spine board, a scoop stretcher, or a factory-designed full-body immobilizer.
3. Application of lateral immobilization support devices for the head.
4. Use of straps to fasten the patient securely to the backboard.
5. Placement of the immobilized patient into a rescue litter, such as a Stokes basket stretcher, if complex terrain must be traversed.

## UPPER EXTREMITY SPLINTING

Fractures and dislocations of the upper extremity are extremely common injuries. Although upper extremity injuries are rarely life threatening, it is important to assess and manage these injuries properly. Appropriate splinting of a minor fracture or dislocation, in addition to decreasing pain, reduces the incidence of serious complications and the risk of permanent disability.

The rescuer must not let obvious injuries to the extremities be a distraction to the care of more life-threatening injuries. In some situations it may be necessary to rapidly secure the patient to a long backboard that supports and splints every bone and joint of the body in one efficient step. [1] Injuries to nerves or blood vessels are a frequent

complication of upper extremity trauma. Circulation, motor function, and sensation distal to the injury must be assessed early and monitored closely.

The purpose of splinting is to prevent motion of broken or dislocated bone ends. Carefully applied splints decrease pain while minimizing further damage to muscles, nerves, and blood vessels. Splinting also reduces the risk of converting a closed injury to an open one. [46]

## Indications and Contraindications

Indications for splinting an extremity are usually clear. Pain with or without deformity following trauma should arouse suspicion for underlying bone or joint injury. Other signs include swelling, discoloration, deformity, crepitus, or loss of neurovascular function. The absence of these findings does not always rule out an underlying fracture or dislocation. Whenever a musculoskeletal injury is suspected, a prophylactic splint should be applied and maintained. The old axiom, "if in doubt, splint," should be followed.

There are no contraindications to splinting suspected upper extremity fractures or dislocations. However, in the setting of multisystem trauma with life-threatening injuries, rapid transport may be more important than extremity splinting. Averting loss of life takes precedence over averting loss of limb.

## Equipment

Various splints are currently available for immobilizing upper extremity injuries. They can be divided into 2 basic types: rigid and soft. [1]

*Rigid splints* are made of many different materials, including cardboard, plastic, aluminum, wire, and wood. These splints must be fastened to the injured extremity using tape, gauze, cravats, or Velcro straps. They are nonflexible and, when applied, immobilize the limb in a rigid fashion to maintain stability. Although some rigid splints are prepadded, many others require the use of cotton or some other soft material. When applying rigid splints, the fingertips should be left exposed so that distal circulation can be monitored.

Cardboard splints are excellent for long-bone fractures of the upper arm. They can be formed into any desired shape and are inexpensive. Plastic, aluminum, wire, and wood splints, although less malleable, are also good choices. An inexpensive aluminum splint that is popular in wilderness medical kits is the SAM splint (The Seaberg Company Inc, South Beach, Ore). The SAM splint is foam padded for comfort, water resistant, lightweight, radiolucent, and reusable. These characteristics also make it an ideal tool for disaster medicine. [47]

Vacuum splints (Fig. 49-21) (Figure Not Available) are a special type of rigid splint in which the air is evacuated from a closed bag containing tiny foam beads. This compresses the contents into a solid mass, resulting in a rigid splint. Injuries can be

encased and immobilized in the position in which they are found, thereby reducing patient discomfort. Flexibility of the splint before removal of air allows molding of the splint to conform to the patient's position. Vacuum splints are radiolucent and do not apply external pressure to the injured extremity.

*Soft splints* include air splints, pillows, slings, and swaths. Immobilization with pillows, slings, or swaths alone is usually inadequate, because they allow significant flexibility and motion. These splints are therefore most effective when used with some form of a rigid device.

Air splints are soft splints that become rigid when inflated. Besides providing immobilization, they help compress underlying soft tissue to reduce local hemorrhage. These devices are sensitive to differences in atmospheric pressure and temperature. Therefore, their inflation must be constantly monitored to ensure that the underlying tissue is not subject to pressure-induced ischemia. One study suggests a maximum splint pressure of 15 mm Hg to reduce the risk of ischemia. [48] With long ambulance transports, the splint should be deflated for 5 minutes every 1.5 hours. [49] A significant disadvantage is that pulses can no longer be monitored once the air splint is in place. Air splints are designed to conform to a specific shape when inflated and should not be used on angulated fractures. In addition to being radiolucent, some types can be inflated with a refrigerant to provide concurrent cooling.

Pillow splints (Fig. 49-22) can be fashioned from any soft bulky material and are excellent choices for hand or wrist injuries. These splints are extremely comfortable and can be easily applied.

Slings and swaths are usually used in combination with a rigid or soft splint. When used alone, they can effectively immobilize injuries to the shoulder, clavicle, or humerus.

**Procedures**

To apply a splint properly to an injured extremity, several general rules must be followed. Communication is important to ensure that the patient understands what is being done at all times. If necessary, clothing should be removed to adequately visualize the injured extremity. Manual stabilization of the fracture site helps limit unnecessary movement and prevent further injury. The neurovascular status (i.e., pulse, motor, and sensation) should be checked before and after the application of a splint. With a severely angulated extremity, traction not exceeding 10 lb of pressure may be applied to reduce the deformity. If resistance or pain is encountered, the extremity should be splinted in the position found. Open wounds should be covered with a sterile dressing before a splint is applied. The splint (Table 49-1) should be applied using the orthopedic principle of immobilizing the joint above and below a suspected fracture site. Cooling and elevation of the injured area may help reduce local swelling. Once the splint has been applied, the distal neurovascular status should be assessed frequently. Any deterioration requires immediate evaluation of the splint to determine if excess pressure is being applied.

**Rigid Splints**

To apply a rigid splint, an assistant should provide support and gentle traction above and below the injury. The splint is then applied on the side of the extremity away from any open wounds. The splint should be large enough to immobilize the joint above and below a fracture or the bone above and below a dislocation. The splint should be well padded

| TABLE 49-1 -- Management of Specific Upper Extremity Orthopedic Injuries | | |
|---|---|---|
| **Site** | **Injury** | **Suggested Immobilization Techniques** |
| Clavicle | Fracture | Sling and swath |
| Shoulder | Dislocation | Sling and swath as it lies |
| Humerus | Fracture | Cardboard or vacuum splint with sling and swath |
| Elbow | Fracture or dislocation | Cardboard or vacuum splint as it lies |
| Forearm | Fracture | Cardboard, malleable metal, air, or vacuum splint with sling and swath |
| Wrist | Fracture | Pillow, cardboard, malleable, metal, or vacuum splint applied in position of presentation |
| Hand | Fracture | Pillow splint in position of function |

| Finger | Fracture | Tongue depressor or small malleable metal splint |
|---|---|---|

to reduce the risk of pressure necrosis. The splint is then secured to the extremity using gauze or tape (Fig. 49-23) .

Vacuum splints are applied in much the same manner as other rigid splints. While an assistant stabilizes the injured site and applies traction, the splint should be wrapped around the extremity and secured with the attached straps.  The air is then evacuated from the splint by means of a hand pump until the splint becomes rigid.

**Soft Splints**

The application procedure for an air splint depends on whether the splint is equipped with a zipper. If the splint does not have a zipper, it must first be placed on the rescuer's arm until the bottom edge lies above the wrist. Next, the rescuer grasps the hand of the patient's injured extremity, while the free hand is used to provide support and gentle traction above the injury (Fig. 49-24 *A*). An assistant should then slide the splint onto the patient's arm (see Fig. 49-24 *B*). After making sure that the splint is not wrinkled, it should be inflated until finger pressure makes a slight dent (see Fig. 49-24 *C*). Zippered air splints should be opened and placed around the injured area. The zipper should be closed and inflation accomplished as discussed previously. With air splints that completely enclose the hand, distal circulation must be assessed by checking capillary refill at the nailbeds.

Pillow splints are applied by encasing the injury in the pillow and securing with tape, cravats, or gauze (see Fig. 49-22) . If possible, the nailbeds should remain exposed to allow for assessment of circulation.

To apply a sling, an assistant should support the injured arm in a flexed position across the patient's chest. The long edge of the triangular bandage should then be placed lengthwise along the patient's side opposite the injury, with its tip over the uninjured shoulder (Fig. 49-25) . The other tip is then brought over the injured shoulder to enclose the arm in the sling. The sling should be adjusted so that the arm rests comfortably with the hand higher than the elbow. The sling is then tied together at the side of the neck, and the knot is padded for patient comfort. The point of the sling at the elbow should be drawn around to the front and pinned. With the sling properly applied, the patient's arm rests comfortably against the chest with the fingertips exposed (Fig. 49-26) .

To apply a swath, a cravat of sufficient length should be placed under the uninjured arm and over the injured arm at the level of the midhumerus. This should then be fastened circumferentially around the thorax so that the injured extremity is secured snugly to the chest (Fig. 49-27) . In adults, 2 cravats may have to be tied together in an end-to-end fashion to produce a swath of sufficient length.

**Complications**

Potential complications of upper extremity splinting include pressure necrosis, conversion of a closed injury into an open one, and loss of neurovascular function. With the use of air splints, there is the additional risk of pressure-induced tissue ischemia and compartment syndrome.

**Summary**

Injuries to the upper extremities, although not life threatening, can have significant immediate or long-term effects. A high index of suspicion for underlying neurovascular injury should always be maintained. Neurovascular status must be checked before and after application of splints and monitored frequently throughout transport.

| **TABLE 49-2** -- Management of Specific Lower Extremity Orthopedic Injuries | | |
|---|---|---|
| **Site** | **Injury** | **Suggested Immobilization Techniques** |
| Pelvis | Fracture | Pneumatic anti-shock garment (PASG), long backboard |
| Hip | Fracture | Traction splint and long backboard, or secure injured leg to uninjured leg |
|  | Dislocation | Long backboard with limb supported by pillows |
| Femur | Fracture | Traction splint or PASG |
| Knee | Fracture or dislocation | Cardboard or vacuum splint in position found |

| | | |
|---|---|---|
| Tibia/fibula | Fractures | Cardboard, air, or vacuum splint |
| Ankle | Fracture or dislocation | Pillow or air splint |
| Foot | Fracture | Pillow or air splint |
| Toe | Fracture | Tape to adjacent toe |

## LOWER EXTREMITY SPLINTING

Many of the principles, techniques, and complications discussed with upper extremity splinting also apply to injuries of the lower extremity (Table 49-2) ; the pillow splint is 1 example (Fig. 49-28) . In addition, the pneumatic anti-shock garment (PASG) can also be used as an effective splinting device for lower extremity injuries (see Chapter 30) . Prolonged use of the PASG as a splinting device is discouraged, since it has been associated with an increased incidence of compartment syndrome. [50] The following discussion is limited to lower extremity traction splinting.

The use of traction and counter traction for the alignment and reduction of fractures dates from the time of Hippocrates. [51] In the late 1800s, Sir Hugh Owen Thomas developed the first full-ring traction splint for the definitive management of fractured femurs. [51] Since the Thomas full-ring splint was not an emergency treatment device, it was later modified by his nephew, Sir Robert Jones, and other surgeons to a half-ring splint that made it easier to apply in the field. During World War I, the modified splint was credited with reducing the mortality rate associated with fractured femurs from 80 to 15%. [52] Since then, several additional modifications that carry the name of their inventors (e.g., Glenn Hare, Joseph Sager, Allen Klippel) have furthered the development of lower extremity traction splints.

In the setting of a fractured femur, muscle spasm and fragment overlap may cause the thigh to lose its cylindrical shape and adopt a more spherical appearance. [51] The resultant decreased tissue pressure and increased volume may allow 1 to 2 L of blood to accumulate at the fracture site. Traction splints are designed to restore the cylindrical shape of the thigh, which in turn increases tissue pressure and inhibits further hemorrhage.

**Indications and Contraindications**

Application of a lower extremity traction splint is indicated whenever a fractured femur is suspected. This should be clinically suspected when there is shortening, angulation, crepitus, swelling, or ecchymosis of the thigh with pain. The use of a traction splint will help align the fracture fragments, relieve pain, prevent damage to neurovascular structures, and reduce blood loss.

It is controversial whether traction should be applied to an open femur fracture. Concern has been expressed that the use of traction may allow contaminated bone fragments to retract into the wound. Should traction produce bony retraction into the wound, this information must be relayed to the receiving physician. A workable compromise is to use the splint to apply sufficient traction to achieve stabilization without retraction. Alternatively, a vacuum splint or the PASG can be used to immobilize the bony fragments in the position of presentation. In any case, stabilization of the fracture site to prevent further hemorrhage, neurovascular damage, or soft tissue injury should take precedence over the theoretical risk of increased contamination.

**Equipment**

Although the lower extremity traction splints currently in use have been updated slightly, the product comparison by Dick is useful (Fig. 49-29) (Figure Not Available) . [53] Except for Sager-type devices, most traction splints produce flexion at the hip joint because of their half-ring design. This flexion of up to 30° does not allow complete fracture alignment, unless the patient is in a reclining position about 30° from horizontal or the injured extremity is elevated to create the same angle.

**Procedure**

When possible, the splinting procedure should be explained to the patient. There is always pain associated with the application of a traction splint, but the patient should be reassured that the resultant stabilization of the fracture site will help reduce subsequent discomfort. The area of injury should be exposed and the distal neurovascular status assessed before application. Open fractures should be managed as discussed previously. If the injured leg is markedly deformed, it should first be straightened using manual traction and maintained in that position by an assistant until a splint has been applied.

If the splint has an adjustable bar, the noninjured extremity can be used for length adjustment. The splint should extend approximately 25 to 30 cm beyond the heel. With the extremity slightly elevated, the *half-ring splint* (e.g., Hare splint [Dyna-Med, Carlsbad, Calif]) is placed under the injured leg and brought to rest firmly against the ischial tuberosity. *Sager-type devices* (Sager splint [Minto Research and Development, Redding, Calif]) should be placed either against the symphysis pubis or positioned laterally against the greater trochanter of the femur (Fig. 49-30) . When the padded end of a Sager-type device is placed in the groin, one should ensure that the genitalia are carefully protected. Once the splint is properly positioned, the thigh strap should be

firmly secured.

A harness is then placed around the ankle immediately above the medial and lateral malleoli and attached to the distal end of the traction splint. Traction is then applied gradually to approximately 10% of body weight or a maximum of 22 to 25 lb (10 to 12 kg). For the *Sager-type devices*, the inner shaft of the splint must be extended until the desired amount of traction is achieved (see Fig. 49-30) , whereas the *Hare splint* uses a ratchet mechanism to apply traction to the ankle strap.

Before movement, supportive straps are applied around the thigh, knee, and distal leg to vertically stabilize the extremity. After application of the splint, the distal neurovascular status should be rechecked. The loss of pulses with application of a traction splint requires that the position of straps and the amount of applied traction be reassessed immediately. Once the splint is applied, the position of the splint and the patient's neurovascular status should be rechecked following any patient movement. Removal of a traction splint should be done in reverse order of application.

### Special Considerations

The traction splints in common use can be applied before application of the PASG. Except for Sager-type devices, application of the PASG over a traction splint is awkward and associated with uneven pressure distribution. In addition, Sager-type devices are advantageous in that they can be applied *before* or *after* application of the PASG.

Unless the patient is found in an extremely cold environment, the shoe on the injured extremity should be removed before splinting to allow for assessment of neurovascular status. Furthermore, this facilitates monitoring of changes in color, temperature, or pulse distal to the injury en route to the hospital. The shoe can be removed by cutting the shoelace and pulling forward on the tongue of the shoe. The underlying sock can then be removed using scissors to prevent movement of the fracture site. Removal of the shoe and sock of the noninjured foot will allow comparison of the distal extremities.

The use of Sager-type devices is not recommended in the presence of an associated distal tibia-fibula or ankle fracture in the same extremity. In these circumstances, the amount of traction required to realign the fractured femur can distract the distal fracture site. Similarly, splints that incorporate part of the foot in their design may not be usable in the presence of an unstable foot fracture in the same extremity. A cardboard splint or the PASG should be considered in these settings.

### Complications

Complications are generally the result of incorrect application and include ongoing hemorrhage, perineal injury, movement at the fracture site, or neurovascular compromise. Once the fracture site is stabilized, additional traction is unnecessary and potentially dangerous.

**Summary**

A properly applied traction splint will limit hemorrhage and movement associated with femur fractures. Careful monitoring of distal neurovascular status is imperative with the use of these splints.

# HELMET REMOVAL

Although originally developed for protection of the head during combat, helmets are commonly used by athletes (e.g., football and hockey players) and motorcyclists. The use of helmets has been shown to reduce the incidence and severity of head injuries associated with motorcycle crashes. Increasing use of helmets in other sporting activities (e.g., kayaking, lacrosse, and roller blading) and by bicyclists and moped riders will further increase the frequency of appearance and the spectrum of injuries associated with helmet use in the out-of-hospital setting. Most modern helmets consist of an inner layer of foam material covered by a hard plastic shell. Helmets can be modified by additional padding so that they conform tightly to the individual's head. Although earlier studies suggested that the use of motorcycle helmets may be associated with an increased incidence of cervical spine injury, this concern has not been substantiated in the recent literature.

Careless removal of a helmet must be avoided, as this may exacerbate an underlying cervical spine injury. In 1980, the American College of Surgeons endorsed the helmet removal technique discussed in this chapter. [59] As the rapid transport of injured patients from the out-of-hospital setting to the hospital is stressed, the desirability of helmet removal in the field setting is likely to receive greater scrutiny.

**Indications and Contraindications**

There are 2 schools of thought with regard to helmet removal in the out-of-hospital setting. One theory suggests that removal of helmets is rarely necessary, since the helmet itself provides for excellent spinal immobilization. The other philosophy recommends routine removal of the helmet so that the patient is completely exposed and the rescuer has full access to the head and neck for airway management, hemorrhage control, and cervical spine stabilization. Since it is difficult to monitor the airway with a helmet in place, most rescuers prefer to remove the helmet whenever possible.

If local protocols do not allow for helmet removal, the airway may be accessed by lifting away the visor or by removing the face guard using bolt cutters or a screwdriver. Cervical spine immobilization can usually be maintained with a properly fitting helmet by using tape, foam blocks,

and a backboard. In these cases the helmet will have to be removed in the ED to allow for complete examination of the head and neck and to help facilitate ancillary studies.

The only absolute contraindication to helmet removal is neck pain associated with the procedure. Other relative contraindications to helmet removal include unfamiliarity with the technique and lack of sufficient assistance. [57]

## Procedure

Proper removal of a helmet requires at least 2 individuals along with manual stabilization of the cervical spine throughout the procedure.  Although a 1-person technique of helmet removal has been described, it is best for 2 rescuers to remove the helmet, especially in the setting of an unconscious or uncooperative patient.

One rescuer establishes neutral position by placing his or her hands on each side of the helmet with the fingers on the victim's mandible (Fig. 49-31) (Figure Not Available) . The second rescuer then cuts or releases the chin strap and assumes manual stabilization by placing 1 hand on the patient's mandible and the other hand on the occiput. The first rescuer then gently removes the helmet by grasping it at the base and expanding it laterally to clear the ears. Some helmets may need to be tilted backward to allow the chin guard to clear the nose. The second rescuer must be prepared to assume the weight of the head as the helmet is removed. The first rescuer then reassumes the responsibility for manual stabilization and neutral positioning while the second rescuer applies a rigid extrication collar. Both rescuers should *avoid in-line traction* during helmet removal to reduce the risk of subluxation or distraction of an unstable cervical spine. [60]

In a volunteer study, Meyer and Daniel have shown limited motion of the cervical spine with this removal technique. [61] The presence of a folded sheet or jacket placed behind the patient's shoulders was also found to limit any cervical motion associated with helmet removal. [61] In contrast, Aprahamian and colleagues demonstrated that manual helmet removal adversely affected a surgically created unstable cervical spine injury in a cadaver model. They recommended the use of a cast-cutter saw to divide the helmet into 2 pieces in the coronal plane and thereby facilitate

removal. Following division of the helmet shell, the inner foam material may be cut with a scalpel or other sharp blade. Although this approach does provide an alternate method of removing the helmet, the intense vibrations produced during use of the cast cutter may exacerbate an underlying spinal injury. In addition, the technique would be slow and difficult with modern, well-fitting, high-quality helmets. [57] Alternatively, immediate removal of the chin piece using a cast-cutter or other saw may permit rapid airway access and allow for safer removal of the entire helmet.

## Complications

Underlying cervical spine injuries may be exacerbated by failure to adhere to proper helmet removal techniques. Although no controlled studies have demonstrated the safety of manual cervical stabilization, its efficacy during endotracheal intubation of the

trauma patient suggests that little movement is likely during helmet removal.

**Summary**

Airway management and cervical spine stabilization may be complicated by the presence of a helmet. Out-of-hospital helmet removal can be accomplished in a safe and effective manner using a 2-person technique.

# Chapter 50 - Management of Amputations

**William C. Dalsey**

"Injury occurs to the hand more frequently than to any other part of the body and the first person caring for an injured hand will probably determine the ultimate stage of its usefulness." [1] Rapid and appropriate emergency care of a patient with an amputated part is crucial to the salvage and preservation of function. This chapter discusses the acute care of amputated parts before they are replanted and specifically addresses the management of distal digit amputations and dermal "slice" wounds.

Amputation may be partial or complete. Injuries with interconnecting tissue between the distal and proximal portions, even if there is only a small piece of bridging skin, technically are considered incomplete, or partial, amputations. Complete amputations are replanted, whereas partial amputations are revascularized. This distinction is arbitrary; for emergency physicians, treatment for both injuries is very similar. The prognosis and outcome of partial and complete amputations are similar, although partial amputations often have better venous and lymphatic drainage, and functional recovery may be more complete if there is less anatomic damage.

The peak incidence of traumatic amputations occurs between the ages of 20 and 40 years, [2] [3] and men predominate over women at a ratio of 4:1. Local crush injuries are the most common mechanism of injury, and sharp guillotine amputations are the least common. [4] [5] [6] [7] Partial amputations occur as often as total amputations. [8] Power saws and lawn mowers are frequently the instruments of destruction. [8] Proximal amputations are less common than distal amputations.

The media have exaggerated somewhat the success of replantation and have often generated unrealistic expectations from the public. The technical limitations of successful repair of vessels that are <0.3 mm in diameter usually preclude replantation of digits distal to the distal interphalangeal joint. [9] Successful revascularization of amputated parts often ensures viability, but neurologic, osseous, and tendinous healing are critical for ultimate function. If there is incomplete neurologic recovery, limited range of motion, and intolerance to cold, the replanted part may have little functional value for the patient. Rehabilitation from replantation surgery may be prolonged, often requiring >1 year and repeated surgical procedures. The emergency physician should be aware of the limitations of replantation surgery and should not encourage unrealistic expectations in injured patients or their families.

## BACKGROUND

The possibility of restoring viability and function to traumatically severed parts has fascinated physicians for centuries. Physicians have attempted to replant parts with little more than a few sutures and secure bandaging and occasionally have had spectacular results. One of the earliest medical reports was by Fiorvanti, who in 1570 reported the successful replantation of a soldier's nose, which was severed by a saber, after first cleansing it with urine and then carefully bandaging it.  In 1814, Balfour reported the

successful replantation of a finger, which was severed by a hatchet, [12] using only meticulous alignment and secure bandaging. The ability to consistently replant amputated parts awaited the development of modern microvascular surgical techniques. The first reported successful upper limb replantation was by Malt and McKhann in 1962. [13] Later that year, a successful replantation of a hand and arm was reported by Chen and Pao. [14] Developments in microsurgical techniques, advanced optics, and microsurgical instruments have created the ability to consistently replant amputated parts with a high degree of success. Since 1965, when Kleinert and Kasdan reported the first successful microvascular anastomosis of a digital vessel, [15] there have been several large series of replantations, with success rates ranging from 70 to 90%. [16] To the original pioneers in replant surgery, survival of the replanted tissue was the criterion for success, but with further refinements, today's surgeons emphasize functional recovery as well as viability. The replantation of a part that is painful or useless or that interferes with function is a disservice to the patient and is less desirable than early restoration of function without replantation.

## INDICATIONS

Preservation of the amputated part is generally indicated when replantation or revascularization is a potential therapeutic method for care of the injured part. Revascularization and reanastomosis of partially and completely amputated parts should be provided when there is hope of preservation or restoration of function. Aesthetic considerations, patient avocations, and occasionally religious or social customs may also influence the decision to proceed with surgery. [29] [30] [31] Ultimately, the decision must be reached by both the operating microsurgical team and the patient after a rational explanation of potential results and successes.

Indications for replantation of fingers and hands have been proposed and are generally accepted, although they should not be applied rigidly to all circumstances. Successful functional recovery is more likely in distal than in proximal extremity amputations and in multidigit amputations, single-digit thumb amputations, or transmetacarpal amputations. [32] [33] Generally these are indications for replantation (Table 50-1) . Single digits proximal to the distal interphalangeal joint and distal to the flexor digitorum superficialis may be replanted successfully, with good functional recovery.

Successful replantations have been reported in patients from the ages of 1 to 84 years. There are no fixed age limits for replantation, although particularly good results have been reported in children owing to their regenerative capacity and adaptability to rehabilitation. The decision to replant is made on a case-by-case basis by the microsurgical team, which must weigh all the factors involved.

## CONTRAINDICATIONS

There is no contraindication to managing the amputated part and stump as though replantation were going to occur, even

| TABLE 50-1 -- Replantation of the Amputated Extremity |
|---|
| **Indications** |
| Young stable patient |
| Thumb |
| Multiple digits |
| Sharp wounds with little associated damage |
| Upper extremity (children) |
| **Absolute Contraindications** |
| Associated life threats |
| Severe crush injuries |
| Inability to withstand prolonged surgery |
| **Relative Contraindications** [*] |

| |
|---|
| Single digit, unless thumb |
| Avulsion injury |
| Prolonged warm ischemia (12 hr) |
| Gross contamination |
| Prior injury or surgery to part |
| Emotionally unstable patients |
| Lower extremity |

\* If the victim is a child or if there are multiple losses, salvage replantations are attempted, and the relative contraindications are ignored.

when replantation is considered unlikely by the emergency physician. The care of the entire patient must take precedence over that of the amputated part, although the requirements of the amputated part and stump can often be handled by ancillary personnel during resuscitation and transportation of the patient. Contraindications to replantation are listed in Table 50-1 and are discussed in the following sections. Note that even when replantation is contraindicated, tissue (skin, bone, tendon) from the amputated part may be useful in restoring function to other damaged parts. Never discard amputated tissue until all possible uses of the severed parts are considered. For example, even though an amputated fingertip is not suitable for replantation, the skin may be an ideal donor source for a skin graft to the stump.

## GENERAL CONSIDERATIONS

### Mechanism of Injury

The potential for successful replantation in terms of survival as well as useful function is directly related to the mechanism of injury. Guillotine injuries, which are sharp, are the least common but have the best prognosis owing to the limited area of destruction.

Crush injuries, which are the most common, produce more tissue injury and therefore have a poorer prognosis. The avulsion injury has the worst prognosis, because a significant amount of vessel, nerve, tendon, and soft tissue injury invariably occurs.

**Ischemia Time**

The time that an amputated part can survive before replantation has not been determined. After 6 hours, additional delay may decrease the success rate of revascularization and lead to diminished function. Skin, bone, tendons, and ligaments tolerate ischemia much better than do muscle and connective tissue. Therefore, as a general rule, the more proximal the amputation, the less ischemia time the amputated part can tolerate. Attempts to extend viability during ischemia have shown that the most important controllable factor is the *temperature* of the amputated part. Warm ischemia may be tolerated for 6 to 8 hours. [41] When the part is cooled properly to 4 °C, 12 to 24 hours of ischemia may be tolerated with distal amputations.  There is a report of a successful digital replantation after 33 hours of cold ischemia. [42] The way in which hypothermia protects tissue from ischemia is currently under investigation. It has been postulated that hypothermia may limit metabolic demand, thereby preserving intracellular energy.  Other investigations suggest that the effect is due to the retardation in development of an acidotic pH. [47] Hypothermia may also prevent the no-reflow phenomenon that can follow low-flow states.

Delay in replantation of proximal arm and leg amputations containing significant amounts of muscle tissue can lead to the buildup of toxic products. In such cases, when blood supply is restored, the absorbed toxins have been reported to cause respiratory failure, renal failure, cardiovascular collapse, and even death.

Perfusion techniques such as those used in organ transplants to extend anoxic time have not yet been developed. In the past, surgical teams used intraoperative perfusion as a technique to help cool the amputated part. The benefits of intraoperative perfusion with cold hypertonic solutions are

currently being investigated. Perfusion should not be attempted by emergency physicians. The risk of damage to vessels and the potential delay in care and rapid transport of the patient and the amputated part override the theoretic benefits of emergency department (ED) cold perfusion at this time.

## SPECIAL CONSIDERATIONS

**Hand Function**

Hand function is often determined in part by pinch and grasp functions. If the index finger is removed, the pinching function of the index finger is adequately provided by the middle finger. Power in grasping and gripping is mainly considered an ulnar function of the fourth and fifth digits. An effective grip that provides the ability to hold a variety of objects is a central function of the ring and middle fingers. In addition to its function in pinching, the thumb is the major opposing force for successful grip and grasp. The

thumb is the most important digit for adequate hand function, and its loss results in 40 to 50% disability. Such disability requires aggressive attempts to replant amputated thumbs. If this is impossible or unsuccessful, pollicization of other digits or toe transfers are secondary alternatives.

## Lower Extremity Amputations

There are few reports of successful replantation of amputated parts of the lower extremity. [63] [64] Indications for replantation of amputated parts of the lower extremity are different from those for replantation of amputated parts of the upper extremity. The goal of all replantation is restored function. If this cannot be achieved, a patient is substantially better off with a prosthesis.

The lower extremity is primarily used for weight bearing and allows the individual to ambulate. Lower limb prostheses, especially those used below the knee, are well tolerated and functional. Prostheses provide a secure stance and permit locomotion. Lower extremity replantation generally requires skeletal shortening, and distal nerve regeneration is often imperfect. Both deficits may produce dysfunction. A patient with a replanted lower extremity with significant shortening and without sensation functions better with a prosthesis. This is not necessarily true of someone with an upper extremity replant. For these reasons, lower limbs are not generally replanted except under ideal circumstances, usually in children, although there are documented cases of successful lower extremity replantation in adults. [63] [64] The final decision regarding replantation should be left to the replantation team.

## Fingertip Amputations and Dermal "Slice" Wounds

Proper treatment of distal fingertip injuries is controversial. Fingertip amputations often heal by normal wound contracture, but occasionally this practice may result in the loss of functional ability to palpate. The basic goals of treatment are to provide tissue coverage, an acceptable cosmetic result, and an early functional recovery. In distal amputations in which the wound area is <10 mm [2] , this is not a problem (Fig. 50-1) (Figure Not Available) . Larger dorsal wounds also heal well by secondary intention. The challenging problem of fingertip injuries occurs when loss of skin and soft tissue from the fingerpad is significant. Volar skin is unique in its combination of toughness and sensitivity. Wounds with significant volar tissue loss frequently require additional treatment. Children, with their regenerative capacity, often progress very well when significant volar wounds are allowed to heal primarily. For older people and for amputations that involve a more significant amount of the distal digit, a wide variety of techniques for managing the injured fingertip have been advocated, including partial-thickness skin grafts; full-thickness skin grafts; V-Y, Kutler, Kleinert, and island advancement flaps; and various local and distal flap coverage techniques. These procedures are designed to preserve length and to provide soft tissue coverage of exposed bone and sensation to the fingerpad. Each of these procedures has its own indications, complications, and limitations. [70] Discussion of these procedures is beyond the scope of this chapter. Most of these techniques are best performed by a specialist in the operating room as primary procedures under ideal circumstances or as delayed

procedures when necessary.

In most complete fingertip amputations distal to the distal interphalangeal joint, the emergency physician can provide adequate care initially with conservative wound management. Although thinking has changed significantly over the years, many hand surgeons still advise skin grafting to shorten the time for wound healing. Although complete transverse amputations could be handled conservatively, wound healing may take several weeks, and these patients may benefit from operative treatments. Patients with complete transections should be referred for consultation to coordinate their initial care and subsequent follow-up.

Incomplete transections and small distal amputations without significant soft tissue loss may heal well with conservative therapy started by the emergency physician. Nonoperative treatment in selected patients provides excellent functional and cosmetic results, minimizes recovery time, and has few complications. Children have excellent regenerative capacity and also respond extremely well to conservative treatment. Necrotic or grossly contaminated tissue should be debrided, and the wound should be irrigated thoroughly. If bone is left exposed without soft tissue coverage, the patient will need an operative procedure; alternatively, the bone may be rongeured (shortened) to allow soft tissue coverage and primary healing with better functional recovery. The nailbed tissues should be preserved, because the presence of a nail affects the cosmetic result. After cleansing and cautious debridement, an occlusive dressing is placed directly over the wound. Tetanus prophylaxis, wound dressings, and bandages, along with placement of a protective splint, complete the initial management of these injuries. Amputations that involve the distal phalanx are frequently treated as contaminated open fractures, with IV antibiotics given initially, followed by a course of oral antibiotics. [75] Wounds managed conservatively must have serial dressing changes and cleansing. Soaking wounds, cleansing, and replacement of dressings help provide superficial debridement, which may aid healing and minimize the chance of secondary infection. Wound contraction and healing usually result in acceptable cosmetic and functional recovery in 2 to 3 weeks. Patients should have appropriate follow-up to ensure adequate healing and recovery.

Partial fingertip amputations distal to the distal interphalangeal joint can also be managed successfully by the emergency physician. These wounds are treated in a manner similar to that for complete amputations. However, when the amputation has substantial undamaged tissue connecting the fingertip, careful alignment and stabilization are provided by sutures or bandaging and protective splinting. Partially amputated fingertips, especially in children, may occasionally survive and regain vascularization and sensation. If the distal tissue becomes ischemic and necrotic, the amputation becomes complete.

Injury to the nailbed requires special attention to ensure proper alignment. If the nailbed tissues are not aligned properly, permanently disfigured nails may result. Removal of the nail helps provide adequate visualization for the placement of sutures and also minimizes the risk of contamination of the wound.

Dermal "slice" wounds (type 1 in Fig. 50-1) (Figure Not Available) are managed by

gentle wound cleansing and application of an antibiotic ointment and a nonadherent dressing, followed by a pressure dressing (e.g., tube gauze). At a dressing change 48 to 72 hours after the initial treatment, the patient can be instructed on daily changes of nonadherent dressings for 10 to 14 days until functional epithelialization of the wound occurs. A protective finger splint or guard also minimizes the risk of further injury and pain from trauma to the sensitive wound area. Protection allows an earlier return to function and employment. Wounds larger than 10 mm [2] and those with deep loss of digit pulp tissue may be candidates for skin grafting (see Chapter 38) .

### Penis, Ear, and Nose Amputations

Replantation of the penis, ear, and nose generally results in better function and cosmesis than a prosthesis or reconstructive surgery. The amputated parts and wounds should be handled as they are for digital replantations.

Penile amputations are an uncommon problem. Most cases result from self-inflicted trauma in patients who are severely psychologically disturbed. Successful replantation has been reported using microsurgical techniques. Preservation or reconstruction of the urethra to maintain a competent urinary stream is critical for success.

Ears and noses frequently are partially amputated and occasionally are totally amputated. Whenever possible, these body parts should be replanted unless they are severely traumatized and there is gross contamination. These wounds frequently heal well, and patients with such wounds have a high survival rate and a low incidence of total necrosis. Replantation of these parts requires good suture technique and careful placement but does not necessarily require skill in microsurgical techniques.

## ASSESSMENT OF THE PATIENT

The initial care and treatment of the patient who has had a body part amputated are the same as those for any trauma patient. The physician must not be distracted by the amputated extremity or the excitement of others from assessing and stabilizing the patient's airway, breathing, and circulation

. Amputations are generally not life-threatening injuries, and other potentially more serious injuries must first be assessed and treated. Hemorrhage from amputated limbs is often limited by the retraction and spasm of severed vessels. Therefore, partial amputations may result in more serious hemorrhage than if the vessels were totally severed. Usually hemorrhage can be controlled adequately with direct pressure and elevation. Vascular clamps and hemostats have no role in the ED management of these injuries and may cause additional injury that may make replantation impossible. A proximally placed blood pressure cuff inflated 30 mm Hg above systolic pressure can be used for short periods of time (<30 minutes) to control severe bleeding, if necessary.

After the initial primary assessment and treatment and subsequent stabilization of the patient, care of the stump and amputated part can be initiated safely. In addition to the general history obtained from all trauma patients, particular attention should be focused

on the exact mechanism of injury, the time and duration of injury, handedness, allergies, medications, illness, prior injury to the affected part, care of the stump and amputated part before arrival in the ED, occupation, avocations, and tetanus history.

Tetanus prophylaxis and broad-spectrum systemic antibiotic therapy (e.g., cephalosporins) should be initiated. Analgesic medications may be necessary, especially with crushing injuries, for managing patient discomfort. The dose of IV opioids should be titrated to the clinical condition. In fingertip amputations, digital or regional nerve blocks are ideal for pain relief but may make functional and neurologic evaluation by a consultant impossible. Some physicians recommend the early use of aspirin, low-molecular-weight dextran, or both for amputation patients, but such attempts to maintain small vessel perfusion are controversial and have not proved efficacious in preoperative treatment.

Amputation patients often experience denial, shock, disbelief, and feelings of hopelessness about their injury; some have even become suicidal. Patients should be treated with supportive and realistic reassurance, but unrealistic medical promises should be avoided. It is important that the emergency physician (or other nonreplantation specialist) not speculate on the specifics of the ultimate prognosis.

Examination of the stump may be brief and should primarily be an assessment of the degree of damage to the surrounding tissue. Gross contamination can be removed by irrigation with normal saline. Local antiseptics, especially hydrogen peroxide or alcohol, should *not* be used, because they may damage viable tissues. Similarly, tissues should not be manipulated, clamped, tagged, or further traumatized in any way. It is important to assess the degree of contamination, the level of injury, and any concomitant injury, such as crushing or multiple levels of injury or amputation. The amputated part should also be examined for the degree of tissue injury, contamination, and possible distal injuries. Radiographs of the amputated part and proximal stump to the level of at least 1 joint proximal to an extremity injury should be obtained. Preoperative laboratory studies and IV access in an uninjured extremity should also be initiated.

The neurologic status of the stump or distal extremity in partial amputations should be assessed by pin prick and 2-point discrimination tests. The presence of sweat may indicate autonomic-neurologic functioning. Vascular competence can be assessed by noting the color, temperature, capillary refill, and presence of pulses. An Allen test at the wrist or a modified Allen test at each digit may aid in determining the existence of an arterial injury (see Chapter 19) . The neurovascular status should be carefully and clearly documented in the medical record. Motor and tendon function should be evaluated immediately. The regional microvascular resource center should be contacted as soon as possible to arrange transportation and to provide adequate time for mobilization of the replantation team.

## CARE OF THE STUMP AND AMPUTATED PART

The stump can be evaluated, and primary care can be rendered during the secondary assessment of the trauma victim (Table 50-2) . If replantation is proposed, the goals of initial care include control of hemorrhage and prevention of further injury or

contamination. All jewelry should be removed. The stump should be irrigated with normal saline to remove gross contamination. Debridement and dissection should be done by a specialist. Do not clamp arterial bleeders. The stump wound should then be covered with a *saline-moistened* sterile dressing to prevent further contamination and to limit damage from desiccation. The stump should be splinted for protection and for the prevention of further injury from concomitant fractures or compromise of blood flow owing to change in position. Splinting and elevation may reduce the extent of edema and help control bleeding.

Care of the amputated part follows the same general guidelines as that for the stump. Gross contamination can be eliminated by irrigation with saline. All jewelry should be removed. The amputated part should be handled minimally to prevent further damage and should be wrapped in a saline-moistened sterile dressing. *Direct immersion in saline or hypotonic fluids should be avoided*, because it may cause severe maceration of tissue and may make replantation more difficult technically. The amputated part should be cooled as soon as possible. The ideal temperature is 4 °C. Care must be taken to prevent the freezing of tissues. Amputated parts should not be placed directly on ice, because tissue that is in direct contact with the ice may freeze. Currently, the recommended method for cooling amputated parts is to place the part, which is wrapped in saline-moistened gauze, in a water-tight plastic bag and immerse the bag in a container of ice water (Fig. 50-2) (Figure Not Available) . A guideline is to use half water and half ice; excessive ice should be avoided. Cooling coils and refrigeration devices have occasionally been used

| TABLE 50-2 -- Axioms for Care of Amputations | |
|---|---|
| **Do's** | **Dont's** |
| Splint and elevate | Apply dry ice or freeze tissue |
| Apply pressure dressing | Place tags on tissue |
| Protect from further trauma or injury | Place sutures in tissue |
| | Sever skin bridges |

| Protect from further contamination | Initiate perfusion of amputated part |
|---|---|
| Provide analgesia | Place tissue in formalin or water |
| Supply tetanus prophylaxis and antibiotic therapy | |
| Obtain radiographs | |

but are generally not available and offer no significant advantages. The tissue containers should be labeled with the patient's name, the amputated part contained within, the time of the original injury, and the time that cooling began.

Treatment for partial amputations with vascular compromise is the same as that just described. Clean the wound with normal saline irrigation. Place a saline-moistened sponge on the open tissue, and wrap the injury in a sterile dressing, incorporating a splint to protect it from further injury. Ice packs or commercial cold packs should be applied over the dressing to cool the devascularized area (Fig. 50-3) (Figure Not Available) .

## COMPLICATIONS

The care of amputated parts should not lead to avoidable complications if the aforementioned principles are followed. Improper management of the parts or stump with subsequent additional injury of the tissue from overzealous hemostasis or cleansing should be avoided. Furthermore, desiccation, maceration, or freezing of tissue from improper storage should not occur. The physician must consider expediting the preoperative work-up of the patient and immediate notification of the replantation team as crucial factors in the patient's care.

Despite optimal initial care, replantation itself may be associated with acute or long-term complications. There is the usual risk of anesthesia and protracted surgery. Postoperative complications include vascular thrombosis, hemorrhage, infection, and reaction to accumulated toxins. It is not unusual for second and third emergency operations to be required to reestablish adequate blood flow. Patients are often placed on anticoagulants, which create an additional risk. Toxins accumulate in ischemic amputated parts, despite cooling. The amount of toxin is directly proportional to the amount of muscle mass and the duration of ischemia. Reports of significant pulmonary failure, electrolyte disturbance, and even death have been reported in replantation

efforts.

Later complications include a significant percentage (60%) of patients with cold intolerance, limited function, anesthesia, pain, paresthesias, malunions, and nonunions. Repeated operative procedures may be required to obtain a functionally useful result. To minimize the morbidity from amputations, proper initial care is essential and may be the most important determining factor in the patient's eventual outcome.

# Chapter 51 - Extensor Tendon Injuries in the Hand and Wrist

**Peter E. Sokolove**

Hand injuries are commonly seen in the emergency department. Extensor tendons are quite superficial, covered only by skin and a thin layer of fascia, and are thus highly susceptible to injury. These injuries may result from lacerations, bites, or burns, but they also may be caused by closed injury with even seemingly superficial lacerations. While some extensor tendon injuries must be managed by a hand surgeon, others may be treated in the emergency department. Extensor tendon injuries are not benign and must be managed with care. The emergency physician must understand the anatomy, principles of treatment, repair technique, and postrepair care of these injuries to ensure the best possible patient outcome.

## FUNCTIONAL ANATOMY

There are 12 extrinsic extensors of the wrist and digits, all of which are innervated by the radial nerve. The muscles that give rise to these tendons originate in the forearm and elbow. The extrinsic extensor tendons reach the hand and digits by passing through a fibro-osseous tendon sheath (retinaculum) located at the dorsal wrist. This synovium-lined sheath provides for smooth gliding of the tendons and prevents bowstringing when the wrist is extended. The dorsal retinaculum contains 6 compartments or subdivisions (Fig. 51-1) . These compartments are numbered from the radial to the ulnar side of the wrist.

The first compartment contains 2 tendons, abductor pollicis longus (APL) and extensor pollicis brevis (EPB). The APL tendon, the most radial of the extensor tendons, inserts on the base of the first metacarpal. It can be palpated just distal to the radial tubercle and also causes radial wrist deviation. The EPB travels with the APL through the first compartment but inserts on the proximal phalanx of the thumb. It can be palpated over the dorsum of the first metacarpal when the thumb is extended against resistance. Both of these tendons can be tested by having the patient spread the fingers apart against resistance.

The second compartment also contains 2 tendons: the extensor carpi radialis brevis (ECRB) and the extensor carpi radialis longus (ECRL). These two tendons arise from the lateral epicondyle of the elbow. The ECRL inserts on the base of the second metacarpal, while the ECRB inserts on the base of the third metacarpal. Both tendons are powerful wrist extensors, and the ECRL also allows some radial wrist deviation. Wrist extension plays an especially important role in the mechanics of the hand, because hand grip strength is maximal only when the wrist is extended.

The third compartment contains only 1 extensor tendon: the extensor pollicis longus (EPL). This tendon crosses over the ECRB and ECRL and travels along the dorsum of the thumb to insert on the distal phalanx. The EPL forms the top of the anatomic "snuff

box," while the bottom is formed by the EPB. The EPL can be visualized when the thumb is extended, and its strength can be tested by having the patient hyperextend at the interphalangeal (IP) joint against resistance. The intrinsic extensor of the thumb can provide some degree of extension at the IP joint. Therefore, if EPL injury is suspected, it is important to compare extension at the IP joint with that of the unaffected thumb.

The fourth and fifth compartments contain the 6 tendons that extend the index through little fingers. Each finger has its own extensor digitorum communis (EDC) tendon. The index and little fingers have an additional independent extensor tendon--the extensor indicis proprius (EIP) for the index finger and the extensor digiti minimi (EDM) for the little finger. The fourth compartment contains the EIP and EDC tendons, while the fifth compartment contains only the EDM tendon. These 6 tendons can be seen over the dorsum of the hand, where they are poorly protected and prone to injury. In this region there are tendinous, ligamentous, and fascial connections between these tendons known as the *juncturae tendini. Because of these interconnections, a patient may be able to extend a digit, albeit weakly, even when there is a complete laceration of its EDC tendon.* In order to avoid missing a tendon injury on the dorsum of the hand, it is important that the examiner test for tendon *strength* and not just for active extension. The course of the extensor tendons along the fingers is more complex, but a basic understanding of this anatomy is essential for the emergency physician to evaluate and treat extensor tendon injuries (Fig. 51-2) (Figure Not Available) . The EIP tendon joins the EDC tendon at the level of the metacarpophalangeal (MCP) joint in the index finger. The EDM tendon parallels the course of the EDC tendon; the 4 EDC tendons eventually insert at the base of the proximal, middle, and distal phalanges. The most proximal insertion of the EDC tendon is at the level of the base of the proximal phalanx. The tendon actually inserts in 2 ways. First, there is a loose dorsal insertion just distal to the MCP joint. In addition, the EDC tendon inserts into the volar plate via the sagittal bands. The sagittal bands are circumferential structures at the level of the metacarpal head that serve to keep the EDC tendon centered over the metacarpal head, as well as to provide a stable connection with the volar plate located on the palmar side of the hand. After its primary insertion at the level of the MCP joint, the EDC tendon then extends dorsally along the digit. The EDC trifurcates over the proximal phalanx (Fig. 51-3) (Figure Not Available) . Its major central slip inserts on the base of the middle phalanx (Fig. 51-4) . The lateral branches of the EDC tendon join with the lateral bands from the interossei and lumbricals to form the co-joined lateral bands. The two co-joined lateral bands then fuse together over the middle phalanx to form the terminal extensor mechanism (TEM), which inserts into the base of the distal phalanx (Fig. 51-5) . The triangular ligament is a connection between the 2 co-joined lateral bands that assists in keeping these structures on the dorsal aspect of the digit.

The sixth dorsal compartment of the wrist contains only 1 tendon: the extensor carpi ulnaris (ECU). This tendon originates at the lateral epicondyle of the elbow and inserts at the base of the fifth metacarpal. The ECU functions as a wrist extensor and ulnar deviator. It can be palpated just distal to the tip of the ulna, and its strength can be tested by forced ulnar deviation of the wrist.

## GENERAL APPROACH TO EXTENSOR TENDON INJURIES

The key to detecting extensor tendon injuries in the emergency department is to perform a careful and thorough history and physical examination. Closed injuries may appear innocuous at first but may result in tendon injuries that often lead to severe deformities or dysfunction if undetected. Closed injuries are also commonly associated with fractures. A hand radiograph is recommended in closed hand injuries when a fracture is suspected or in open-hand injuries where fracture or a foreign body is suspected. It is generally accepted that all open injuries that result from glass should be radiographed. Plain radiographs have a sensitivity of approximately 99% for detecting a 2-mm glass foreign body, as long as it is not obscured by bone. [3]

Open injuries to extensor tendons are quite common, especially on the dorsum of the hand, where they are superficially located. *All dorsal wrist, hand, and digit lacerations should be assumed to have an underlying tendon laceration until proven otherwise.* Digital extension can still occur with partial tendon lacerations of up to 90%. Complete lacerations of an EDC tendon on the dorsum of a hand can also still allow digital extension through the juncturae tendini.

After assessing the strength and neurovascular status of the injured hand, it is imperative that the emergency physician visually inspect the wound thoroughly. Inspection should include an assessment of the degree of wound contamination, as well as a search for foreign bodies and occult tendon lacerations. Since an extensor tendon is a mobile structure, it is imperative that if it is exposed, it is visualized

in its entirety through a full range of motion. *It is especially important to examine the tendon in the position of injury, as frequently the tendon injury does not lie directly under the skin wound.* Examination of the wound must occur under the best possible conditions--with a good light source, a bloodless field, and adequate local anesthesia. If the examining physician suspects, but is unable to locate, a tendon laceration, or if a patient is uncooperative with the examination, the patient should be referred for follow-up in 1 to 2 days for a repeat examination. Interim wound care with skin closure and splint application is advised. Inability to rule out a tendon injury in the emergency department and the mandate for follow-up should be clearly documented on the medical record and disposition instructions.

## PREPARATION FOR REPAIR

Prior to attempting repair of an open extensor tendon injury in the emergency department, it is essential that the treating physician be prepared and have the proper equipment available. Patients should be placed supine on a gurney that ideally has an arm board attached. Bright overhead lighting is important for wound exploration so that the presence of tendon injuries and foreign bodies can be adequately assessed. Instruments should include, at a minimum, a needle holder; 2 skin hooks and retractors; sharp (i.e., "iris") and blunt-nosed scissors; several small hemostats; and one pair of small, single-toothed (i.e., Adson) forceps.

The choice of suture material depends on the location of the tendon injury. For the repair of *complete* tendon injuries on the dorsum of the hand, nonabsorbable, synthetic,

braided sutures are preferred. [2] Polyester sutures, such as Ethibond or Mersilene, are recommended. Nylon sutures are acceptable but are less ideal, as colored nylon may be visible under the skin. Chromic and plain gut should be avoided because they will dissolve before adequate tendon healing has occurred. Silk is not desirable because of its reactivity. Most extensor tendons on the dorsum of the hand will accommodate 4-0 sutures, but 5-0 suture material may be needed for smaller tendons. Small, "plastic repair," tapered needles should be used to avoid tearing the tendon. *Partial* tendon injuries of the digits are best repaired with synthetic, absorbable sutures such as polyglactin (i.e., Vicryl).

It is imperative that the physician use adequate anesthesia so that thorough wound exploration can occur. A field block or regional nerve block (see Chapter 32) can be used on the dorsum of the hand, while local anesthesia or a digital nerve block can be used on the fingers. It is best to use 1% lidocaine *without* epinephrine delivered via a 25- to 27-ga needle. It is important to liberally anesthetize the area around to the wound, since many lacerations *must be extended to afford access to the surgical field*. It is a common error to neglect to extend a laceration and to attempt examination, cleaning, or repair through a small initial skin laceration.

Following the administration of anesthesia, a tourniquet must be placed on the involved limb. It is absolutely essential that adequate control of blood flow be obtained before attempting to repair a tendon laceration. It is very difficult to find the proximal end of a retracted tendon in a bloody field. The patient's arm should be elevated for at least 1 minute prior to tourniquet application to allow blood to drain by gravity. A blood pressure cuff is placed on the mid to upper arm, wrapped in several layers of cast padding, then inflated to 260 to 280 mm Hg. Once inflated, the tubes are clamped tightly using a hemostat. The use of cast padding during inflation helps avoid inadvertent unraveling of the cuff. The use of a hemostat to clamp the blood pressure cuff tubes helps avoid a slow leak in the cuff with resultant deflation. A blood pressure cuff tourniquet is generally well tolerated by patients for approximately 15 to 20 minutes. If tendon repair cannot be accomplished in this amount of time, it is likely that the injury is too complex for repair in the emergency department.

## PATTERNS OF INJURY AND MANAGEMENT

The treatment for extensor tendon injury depends primarily on whether the injury is open or closed, as well as the anatomic location of the injury. The most widely accepted classification system is that developed by Verdan, which divides the hand and wrist into 8 anatomically based zones (Fig. 51-6) (Figure Not Available) . It is quite useful for emergency physicians to become familiar with this classification, because in many instances the zone of injury can help determine whether tendon repair should be attempted in the emergency department.

One must keep in mind that repair of lacerated extensor tendons within 72 hours of injury is still considered primary closure. Therefore, while emergency physicians may repair many extensor tendon injuries immediately, some injuries are best managed with delayed repair. In these cases, initial care in the emergency department should consist of sterile skin prep, copious wound irrigation and inspection for foreign bodies, skin

closure, splint application, and a referral to a hand specialist for further care in 1 to 2 days.

**Zone 7 and 8 Injuries**

Zones 7 and 8 consist of the area over the wrist and dorsal forearm, respectively. Extensor tendon lacerations in these regions can be quite complex and are therefore not repaired in the emergency department. Because of the close proximity of extensor tendons in the distal forearm, lacerations such as stab wounds may appear innocuous but often result in multiple tendon lacerations. At the wrist level, extensor tendons are covered by a retinaculum that is lined with synovium. While this tissue allows smooth gliding of tendons during normal activity, the presence of synovium increases the risk for adhesions following tendon repair. In addition, lacerated tendons in the wrist and distal forearm may retract away from the site of initial injury. This may make tendon retrieval and repair quite difficult and may necessitate incision of the retinaculum and exploration of 1 or more of the compartments.

As a result of the potential complexity of these injuries, all tendon lacerations in zones 7 and 8 require formal surgical exploration and repair. Emergency department management of these patients includes local wound care (see Chapter 36) with primary repair of the skin (see Chapter 37) and placement of a volar splint with 35° of extension at the wrist and 10° to 15° of flexion at the MCP joints (see Chapter 53) . Many hand surgeons recommend prophylactic antibiotics for all open tendon lacerations, while others prescribe them selectively for patients with wounds where contamination is suspected or significant injury to the surrounding tissues is present. There are no prospective data proving or disproving the value of prophylactic antibiotics in either circumstance. These patients should be promptly referred to a hand surgeon so that repair may be undertaken within 1 week of injury.

**Zone 6 Injuries**

Zone 6 consists of the area over the dorsum of the hand. Extensor tendon injuries in this region frequently result from lacerations due to broken glass or another sharp object. Common pitfalls in emergency department management of these injuries are usually related to failure to recognize that the tendon has been injured. It is important to remember that these tendons are superficially located, partial tendon lacerations may occur, and weak extension of a digit is possible in the presence of a complete tendon laceration because of transfer of extensor function through the juncturae tendini. Lacerations of the EIP or EDM tendons are evidenced by an inability to independently extend the index or little fingers, respectively. All of these pitfalls can be avoided if a careful physical examination is performed, including a thorough wound exploration under sterile conditions using a tourniquet, adequate local anesthesia, and good lighting.

Extensor tendon injuries in zone 6 are usually appropriate for repair in the emergency department. Because of the juncturae tendini, extensor tendons in zone 6 are less likely to retract than those in zone 7 or 8; however, the severed tendon may retract when the injury is more proximal. The distal end of a severed tendon is usually easy to find by

passively extending the patient's affected digit to bring the end into view. Retrieval of the proximal portion of a severed tendon is sometimes required and usually can be accomplished in the emergency department. Prior to searching for the proximal end of the tendon, the physician should have a 4-0 nylon suture loaded onto a needle holder. When the proximal end is located, this suture should be placed as a holding suture as far proximal as possible so that the tendon is not lost again. It is often necessary to use a scalpel to extend the wound proximally in a direction parallel to the course of the injured tendon to obtain adequate exposure. One should then begin to search for the tendon by lifting up this overlying skin with a forceps and inspecting the proximal portion of the wound. Sometimes the blood-stained end of a tunnel can be seen; this may contain the proximal end of the tendon. By gently placing a small hemostat or toothed forceps up this tunnel, the tendon stump can often be pulled into view.

Once both ends of the injured tendon have been located, the technique used for repair is dependent on the size and shape of the tendon. While larger, round tendons can accommodate sutures that pass through the core of the tendon, smaller or flat tendons are difficult to repair using this technique. Most of the tendons in zone 6 can be repaired with either a modified Kessler or a modified Bunnell technique, using 4-0 nonabsorbable suture (Fig. 51-7) (Figure Not Available) . Smaller tendons may be repaired using a figure-of-eight or horizontal mattress suture. Small, tapered needles should be used to avoid tearing the tendon. In a cadaver study comparing these multiple suture techniques, it was found that the modified Bunnell technique provided the strongest extensor tendon repair. [5] In addition, this technique produced no gaping between the repaired tendon ends and minimized the postrepair restriction of flexion at the MCP and proximal interphalangeal (PIP) joints. It is important to passively test the degree of flexion at the MCP joint after zone 6 tendon repair to be certain that the tendon has not been excessively shortened. [6]

The approach to partial extensor tendon lacerations is not well defined, largely due to a paucity of literature on this subject; however, a logical approach may be extrapolated from data on flexor tendon injuries. It has been demonstrated that many partial *flexor* tendon lacerations do well without repair, [7] but there is still great disagreement among hand surgeons concerning the need for repair of these injuries. In a recent survey of hand surgeons, 30% of respondents repaired *all* partial flexor tendon lacerations, while 45% of respondents repaired *only* lacerations with >50% cross-sectional area involvement. [8] Except at the wrist level, extensor tendons are not covered with synovium and are less likely than flexor tendons to develop adhesions after repair. This encourages some authors to recommend repair of most partial extensor tendon lacerations. While the ideal approach to these injuries is not known, it is reasonable to consider repair of partial extensor tendon lacerations to be *optional* if <50% of the cross-sectional area is involved.

After repair of a lacerated EDC tendon in zone 6, a plaster splint should be applied so that the wrist is in 45° of extension, the affected MCP joint is in neutral (0° flexion), and the unaffected MCP joints are in 15° flexion. [2] The PIP and distal interphalangeal (DIP) joints should be allowed full range of motion. If there is an isolated EIP or EDM tendon injury, then only the index or little finger must be included in this splint.

**Zone 5 Injuries**

Zone 5 consists of the area over the MCP joint. Open injuries in this region should be considered secondary to a human tooth bite until proven otherwise. This is especially true if the injury occurs over the first or second MCP joint, as this is frequently the location of a clenched-fist ("fight-bite") injury. Emergency department evaluation must begin with a careful and persistent history and physical examination, although patients' reluctance to admit to punching someone in the mouth is notorious. The wound should be inspected through its full range of motion, since the position of the EDC tendon changes with hand position. It is generally recommended that radiographs be obtained in all of these injuries to evaluate for metacarpal head fractures, air in the joint space, or presence of a foreign body, such as a tooth fragment.

If, after a thorough evaluation, it is determined that a human bite to this region has resulted in a superficial skin laceration only, without injury to the underlying tendon or joint, outpatient management is appropriate. The wound should be copiously irrigated and left open. A volar splint is applied with the wrist in 45° of extension, the MCP joints in the neutral position (0° of flexion), and the hand dressed with a bulky dressing. Most authors recommend that 3 to 5 days of prophylactic antibiotics be given to these patients, and patients should be seen in 24 hours for a repeat examination to evaluate for wound infection. If a human bite results in tendon damage, including partial or complete laceration, the patient generally should be admitted for IV antibiotics, and delayed closure should be undertaken by a hand surgeon in 5 to 10 days. Primary closure of even seemingly clean and well-irrigated human bites in this region is not advisable because of the increased risk of wound infection, as well as the potential for septic destruction of the MCP joint if it is violated. If an *open joint* is noted by physical examination or saline arthrogram, patients should also be admitted for IV antibiotics and are generally taken to the operating room for surgical exploration and irrigation.

If a patient suffers a zone 5 tendon injury, and it can be determined with complete certainty that it was caused by a relatively clean, sharp object rather than by a human bite, primary closure is appropriate. Referral of these injuries to a hand surgeon is a common practice, given the complexities of the injury and the sequelae. Careful repair of lacerations to both the EDC tendon and the sagittal bands is necessary to prevent subluxation of the EDC tendon away from the center of the metacarpal head. Initial emergency department management is often limited to skin closure, splinting as described above, and referral to a hand surgeon within 1 to 2 days.

An acute closed injury to zone 5 may result in traumatic subluxation of the EDC tendon as a result of a tear in the extensor hood (Fig. 51-8) (Figure Not Available) . The injury results from a direct blow to the digit that causes forced flexion with or without ulnar deviation; most commonly it involves the long finger, with dislocation of the EDC tendon to the ulnar side. The subluxation becomes more prominent with flexion at the MCP joint. This injury is usually adequately treated with extension splinting for 4 to 6 weeks. Once the hand is splinted in the emergency department, these patients should be referred to a hand surgeon for follow-up within 1 week. Repetitive closed injury to the MCP joint region can produce a condition known as *boxer's knuckle*. This consists of

small tears of the EDC tendon, the sagittal bands, or the joint capsule. These patients tend to present with chronic and recurrent pain and swelling at the MCP joint region and usually have normal radiographs and no tendon subluxation. Elective repair by a hand surgeon generally results in a good outcome.

## Zone 4 Injuries

Zone 4 consists of the area over the dorsal aspect of the proximal phalanx between the MCP and PIP joints. The extensor tendon is a broad, flat structure in this region and is relatively easy to repair. Because the extensor tendon is flat and conforms to the roundness of the proximal phalanx, tendon injuries in this area usually result from a laceration and are almost always incomplete. As a result, extension at the PIP joint is usually not impaired. It is therefore imperative that all of these wounds be explored carefully, remembering that the extensor tendon lies immediately beneath the thin overlying skin. Tendons tend not to retract in this area, so close inspection will usually result in location of the injured tendon.

Central slip lacerations or any laceration that results in an extension lag at the PIP joint are usually repaired by a hand surgeon. The decision regarding whether to repair a partial tendon laceration and whether it should be repaired by the emergency physician in this zone is best discussed with the consulting hand surgeon. In general, because of the duality of the extensor system in this region, lacerations of a single lateral slip can either be repaired with 5-0 nonabsorbable suture or be left unrepaired and splinted. A running suture or simple interrupted sutures with buried knots are appropriate for this area. Postrepair splinting depends on the presence of tension at the repair site. Minor lacerations in zone 4 that do not result in tension on the repair site can be treated with a finger guard for 7 to 10 days and early range of motion. Larger lacerations or those that result in tension at the repair site are usually treated in a splint that extends from the forearm to the digit for 3 to 6 weeks. The splint should be applied so that the wrist is in 30° of extension, the MCP joints at 30° of flexion, and the PIP joint in neutral position. Fingers should be grouped so that either digits 2 and 3 or digits 3 through 5 are immobilized.

It is important to recognize that *complex partial* tendon lacerations (e.g., a laceration of a lateral slip resulting from a saw) in zone 4 may result in damage to the gliding layer located between the tendon and the bone. If the patient is still able to actively extend the digit at the PIP joint, then these *complex partial* tendon lacerations are best managed by debriding the frayed tendon ends and splinting the digit in extension rather than attempting to suture the damaged tendon. The splint should be worn for 10 days, followed by active range of motion.

## Zone 3 Injuries

Zone 3, the area over the PIP joint, is a common site of both closed and open injury. Open injury usually results from laceration with a sharp object. It is imperative that these wounds be carefully explored in the emergency department to rule out penetration of the joint capsule. Patients with wounds that are suspected of penetrating the joint are generally taken to the operating room for surgical exploration, irrigation, and treatment

with IV antibiotics.

Zone 3 tendon lacerations can result in long-term deformity if not carefully repaired, and patients with such injuries are commonly referred to a hand surgeon. Partial lacerations of the central slip or lateral bands are managed variably, and it is advisable to discuss these injuries with the consulting hand surgeon. Lacerations in this area may sometimes result in a complete central slip injury. This may present as an acute boutonniere ("buttonhole") deformity, where the PIP joint rests in 60° of flexion. The presentation may be more subtle, however, and may only be noticeable by weakened extension at the PIP joint or incomplete extension by only a few degrees.

The boutonniere deformity develops when the central slip is ruptured by an open or closed mechanism, leading to unopposed action of the flexor digitorum superficialis tendon (Fig. 51-9) (Figure Not Available) . This results in flexion at the PIP joint, protrusion of the head of the proximal phalanx between the two lateral bands, and disruption of the triangular ligament. When this occurs, the lateral bands are displaced volar to the axis of motion of the PIP joint. The lateral bands then paradoxically become flexors of the PIP joint. In addition, the extensor hood mechanism is pulled more proximally, resulting in increased tension on the terminal extensor mechanism and hyperextension at the DIP joint. Thus, the boutonniere deformity consists of flexion of the PIP joint with hyperextension at the DIP joint.

Open central slip injuries are usually managed operatively, and complex injuries may require direct attachment of the tendon to bone or tendon reconstruction. If the consulting hand surgeon chooses not to repair the tendon injury immediately, the skin should be closed and a plaster splint applied in the same fashion as described for zone 4 injuries. Thermoplastic splints allow splinting of the hand without involvement of the wrist but are generally not available in the emergency department setting. These patients should be promptly referred to a hand surgeon so that repair may be undertaken within 1 week of injury.

Patients with closed injuries to zone 3 present commonly to the emergency department. They may complain of a direct blow to the dorsal PIP joint, or they may complain of a "jammed" finger. This injury occurs when an object such as a ball delivers a sudden axial loading force with forced flexion of the PIP joint while it is extended. These patients commonly complain of a painful, swollen PIP joint, which often makes the examination difficult. Some of these injuries probably represent PIP joint dislocations that were spontaneously or manually reduced prior to the patient's emergency department presentation. [9] The tendon injury that is important to recognize in this setting is an occult isolated central slip rupture. Patients may have decreased extension at the PIP joint, but frequently extension is normal. With forced extension against resistance, patients usually have pain and may have decreased strength. With acute central slip rupture, PIP joint extension may be particularly weak when the MCP joint is flexed (Elson test; Fig. 51-10) (Figure Not Available) . [10]

The boutonniere deformity usually does not develop in patients with closed zone 3 injuries until 10 to 14 days after injury. The only way to prevent this deformity is to have a high index of suspicion for its presence and treat these patients conservatively. It is

advisable that all patients with a swollen, tender PIP joint and pain with flexion or extension be splinted and referred for follow-up within 1 week. A dorsal splint should be applied overlying the PIP joint, keeping it in full extension. This can be accomplished using an aluminum foam-backed splint or a Bunnell ("safety pin") splint, although the latter is generally not available in the emergency department. [11] The MCP and DIP joints should be left free to have full, active range of motion (Fig. 51-11) .

## Zone 1 and 2 Injuries

Zones 1 and 2 consist of the area over the DIP joint and the middle phalanx, respectively. In zone 2 the cojoined lateral bands come together to form the terminal extensor mechanism (TEM) and are held together, in part, by the triangular ligament. The TEM inserts on the base of the distal phalanx and allows extension at the DIP joint. Complete disruption of the TEM results in inability to extend at the DIP joint. Because of the unopposed action of the flexor digitorum profundus (FDP) tendon, the DIP joint rests in the flexed position. This is known as a mallet deformity of the finger (Fig. 51-12) (Figure Not Available) .

Tendon *lacerations* in zones 1 or 2 that result in a partial or complete mallet deformity should generally be repaired by a hand surgeon. This procedure usually consists of repair of the lacerated tendon as well as placement of a K-wire through the distal phalanx into the middle phalanx to stabilize the joint. Sometimes only an external splint is used after tendon repair. Occult partial tendon lacerations are important to recognize to prevent development of a mallet deformity. If there is a partial tendon laceration in zones 1 or 2 that does not result in any extension lag, the approach to repair is variable, and it is advisable to discuss the repair with the consulting hand surgeon. In general, partial tendon lacerations of <50% of the tendon area that do not result in an extension lag may be splinted in extension for 7 to 10 days with or without repair of the tendon itself. [11] Partial tendon lacerations of >50% that do not result in an extension lag may be repaired by a hand surgeon or an emergency physician who is experienced in the repair of these injuries. In either case, it is advisable to discuss whether tendon repair will occur in the emergency department or the operating room with the consultant hand surgeon.

If the zone 1 or 2 tendon laceration is repaired in the emergency department, it should be approximated using a figure-of-eight or horizontal mattress, 5-0 nonabsorbable suture. It is important that the tendon ends be approximated but not pulled too tightly; otherwise joint stiffness and limitation of flexion will occur. After repair of a partial tendon laceration, the DIP joint should be splinted in extension for 6 weeks, followed by 2 to 4 weeks of night splinting and active range-of-motion exercises. Patients should be warned after tendon repair that there is likely to be some residual loss of flexion at the DIP joint, even in the best case.

*Closed* injuries in zones 1 and 2 may result in a partial or complete mallet deformity depending on the injury pattern. These injuries are usually caused by an axial loading force with forced flexion of the DIP joint while it is being held in extension. A common emergency department presentation of this injury is a patient who complains of pain and

swelling at the DIP joint after a ball strikes his or her fingertip.

Closed tendon injuries in this region can generally be classified into three types. In the first type of injury, there is a closed rupture of the TEM. The second type of injury is an avulsion fracture of the dorsal lip of the distal phalanx. This fracture is intra-articular, but there is no volar displacement of the remaining portion of the distal phalanx. Both type 1 and type 2 injuries can be treated by splinting in full extension for 6 weeks. The splint should hold the DIP joint in extension while allowing free range of motion of the PIP joint (Fig. 51-13) . The splint can be constructed from an aluminum, foam-backed splint or from a prefabricated Stack splint.

The third type of closed injury is an intra-articular avulsion fracture of the dorsal lip of the distal phalanx *with* volar displacement of the remaining portion of the distal phalanx (Fig. 51-14) . Normally the DIP collateral ligaments hold the distal phalanx in place; however, if there is a large enough fracture fragment (usually >50% of the articular surface), then the remaining distal phalanx fragment displaces in the volar direction secondary to unopposed action of the FDP tendon. When volar displacement of the distal phalanx occurs, this injury is usually treated operatively by placement of a K-wire and open reduction and internal fixation of the fracture. It is important to remember that it is the presence of volar subluxation, not the size of the avulsion fracture, that determines the need for operative management.

Any injury, whether open or closed, that results in a complete disruption of the TEM may result in a swan-neck deformity (Fig. 51-15) (Figure Not Available) . This deformity consists of flexion at the DIP (a mallet finger), as well as hyperextension at the PIP joint. This results from a dorsal and proximal displacement of the lateral bands causing increased extension forces on the middle phalanx. This complication often can be avoided if disruption of the TEM is diagnosed and treated correctly in the emergency department.

## COMPLICATIONS

All extensor tendon repairs are subject to the usual complications of wound infection and skin breakdown secondary to prolonged splinting. Tendon rupture is a rare complication following tendon repair and may result from inadequate suture technique or premature motion against resistance. It is important when extensor tendons are repaired for at least 5 throws to be used and a square knot to be tied. All extensor tendon repairs require some period of complete immobilization during tendon healing, and it is important for the emergency physician to stress the necessity for patient compliance.

Extensor tendon injuries in zone 7 tend to have the worst prognosis. Because of the presence of a synovial lining, postrepair adhesions may occur. [6] These adhesions may lead to decreased excursion of the extensor tendons with resultant decreased mobility at the wrist. There may also be limitation of finger flexion when the wrist is flexed, as well as finger extension when the wrist is extended. Because of the lack of synovium,

the relatively simple anatomy, and the usual lack of associated injuries, zone 6 tendon injuries tend to have fewer complications than other areas of the hand. The tendons in zone 6, however, do have a tendency to shorten if the tendon ends are approximated too tightly. This may result in a restriction of PIP and MCP joint flexion that is much more pronounced than any loss of extension. [6]

Zone 5 injuries are particularly prone to infection, as injuries in this region commonly occur from a human bite. In addition, if the extensor hood covering the MCP joint is not carefully repaired, subluxation of the EDC tendon may occur. [4] If complex partial tendon lacerations in zone 4 are managed too aggressively, then tendon shortening and stiffness may result. As discussed previously, these injuries are often best managed with splinting alone. A common complication of zone 3 extensor tendon injuries is development of a boutonniere deformity. This usually results from failure to diagnose or adequately immobilize a central slip injury. Similarly, undiagnosed or improperly treated extensor tendon injuries in zones 1 and 2 may lead to either a swan-neck or chronic mallet deformity of the digit. DIP joint splinting itself may result in skin ulceration or tape allergy, usually presenting in the second week of treatment. [12] Skin breakdown may be encouraged if the DIP joint is splinted in hyperextension, because of decreased skin perfusion.

## POSTREPAIR CARE AND REHABILITATION

Proper care after diagnosis and repair of an extensor tendon injury is extremely important for optimal patient outcome. Even the best initial tendon repair can have a poor result if subsequently treated improperly. The rehabilitation of tendon injuries has evolved since 1980 to include dynamic splinting and active range-of-motion exercises to obtain maximal motion of the affected digit.

Zone 1 and 2 injuries are usually treated with static splinting as described previously. After 6 weeks, active range-of-motion exercises should begin. Night splinting is recommended for an additional 4 weeks and during any athletic activities. [9] It is advisable to give the patient a number of extra splints so that the patient (or family) can change the splint frequently to avoid pressure injury. During splint changes, it is important that the DIP joint be held in full extension either by using the other hand or by placing the finger against a table. Closed injuries of the central slip (zone 3) are often treated with a boutonniere splint for 4 weeks, followed by 2 weeks of gradual flexion exercises and night splinting. During the initial 4 weeks, the patient should be instructed to passively flex the DIP joint every hour to maintain gliding and proper position of the lateral bands.

Lacerations in zones 3 and 4 have traditionally been treated with static splinting from the forearm to digits. An alternative approach is to splint only the DIP and PIP joints in extension, and begin a "short-arc-motion" protocol within 1 to 2 days of repair. [13] This consists of active motion at the PIP joint progressing from 0° to 30° the first 2 weeks to 0° to 50° the fourth week. When compared to static splinting, this protocol may lead to better PIP and DIP joint flexion, without resulting in tendon rupture or boutonniere deformity.

Early motion following extensor tendon repair has been found to be most useful in zones 5 though 7. A dynamic extension splint is commonly used in which the wrist is extended 45°, and all finger joints rest in the neutral position (Fig. 51-16) (Figure Not Available) . A volar block allows 30° to 40° of MCP joint flexion, while a dynamic traction mechanism passively extends the digits. Dynamic splinting is started 1 to 3 days following repair. Active motion is added at 3 to 4 weeks, and resistance is added at 7 weeks. A short-arc-motion protocol with controlled active motion at the MCP joint has also been shown to be safe and effective when started 24 to 48 hours following repair. [13] Both dynamic splinting and early active motion are most beneficial when managed closely by a skilled hand therapist. Patients must be reliable and motivated to take advantage of these techniques. It is best to refer patients to a hand surgeon or hand therapist as soon as possible following repair so that rehabilitation can begin in a timely manner.

## EXTENSOR TENDON INJURIES OF

THE FOOT

The extensor tendons of the foot are less commonly injured than the extensor tendons of the hand and wrist. The most important extensors of the foot and ankle that may be presented with injuries in the emergency department are the tibialis anterior, extensor hallucis longus, and extensor digitorum longus tendons.

The tibialis anterior muscle originates on the shaft of the tibia and interosseous membrane and inserts on the medial cuneiform and the base of the first metatarsal. The tibialis anterior extends the foot at the ankle joint and inverts the foot at the subtalar and transverse tarsal joints. [14] Spontaneous rupture of the tibialis anterior tendon may be seen in both elderly and young patients who have been injured during athletic activity. Injury to this tendon commonly results from forceful attempted dorsiflexion of the ankle while it is held fixed in the plantar-flexed position. [15] Patients generally present with decreased strength of foot dorsiflexion, because toe extensors are used to accomplish this motion. Ruptures or lacerations of the tibialis anterior tendon should be referred promptly to an orthopedic surgeon for consideration of formal operative repair.

The extensor digitorum longus (EDL) and extensor hallucis longus (EHL) tendons both originate from the shaft of the fibula and interosseous membrane. The EHL tendon inserts into the base of the distal phalanx of the great toe, while the EDL tendon divides into four branches that insert on toes 2 through 5 (Fig. 51-17) (Figure Not Available) . Both the EHL and EDL tendons primarily result in extension of the toes and dorsiflexion at the ankle. The extensor digitorum brevis (EDB) and extensor hallucis brevis (EHB) muscles originate from the upper part of the calcaneus. The EHB tendon joins the lateral aspect of the EHL tendon prior to inserting on the great toe. The EDB muscle gives rise to three tendons that join the lateral side of the EDL tendons going to toes 2 through 4.

Injury to the EHL and EDL tendons may result from a sharp object lacerating the dorsum of the foot. Patients may present with weakness of, or an inability to extend, the

involved toe. Whether one should repair EHL or EDL tendon lacerations is controversial. However, many authors favor repair; failure to repair EDL tendons may result in a claw deformity of the adjacent toes. [16] Lacerations of EHL and EDL at the level of the ankle are usually repaired, while lacerations on the dorsum of the foot and the toe are managed variably. If there is any flexion deformity of the involved toe, one should probably repair the lacerated tendon. Repair is also favored when both tendon ends are easily visualized in the wound. Since management of theses injuries is controversial, it is advisable to discuss the care of these patients with the consulting orthopedic surgeon. Extensor tendon repair of the foot is usually not performed in the emergency department setting. Superficial cutaneous nerves are easily injured on the dorsum of the foot during wound exploration, which can lead to the formation of a chronic, painful neuroma. If the injury is repaired in the emergency department, the technique for repair is similar to that used for the dorsum of the hand (zone 6). A posterior splint that includes the toes should be applied following tendon repair. The ankle should be splinted at 90° with the toes in the neutral position.

# Chapter 52 - Management of Common Dislocations

**Robert McNamara**

Joint dislocations are frequently encountered in patients presenting to the emergency department. They can range from a simple finger injury to limb- or life-threatening consequences of high-energy trauma. Although the dislocated joint is most often clinically obvious, the presentation may be obscure or masked by other injuries. Emergency physicians must be capable of detecting and managing these injuries; appropriate timely referral to a consultant is generally required for complex dislocation injuries.

This chapter addresses the diagnosis and management of joint dislocations. Keys to the clinical assessment and radiographic evaluation of these injuries are discussed along with methods of reduction. The emphasis of the chapter is on simple dislocations that should be diagnosed and generally managed in the emergency department (ED). Fracture-dislocations that commonly require operative intervention and emergency orthopedic consultation are not discussed.

## PREPARATION OF THE PATIENT

Although many authors claim their reduction method is well tolerated without premedication, they generally have not quantitatively measured the discomfort of their patients. However, *verbal* techniques for alleviating anxiety and discomfort are not to be discounted as they can be of great assistance during joint reduction. In field settings, simple hypnosis techniques have been successfully used for major joint dislocations. [1] In the ED, verbal reassurance and distracting conversation are useful adjuncts.

There are no set guidelines for the use of pharmacologic adjuncts in the management of dislocations. Each patient and presentation is unique and the treating physician must use judgment as to whether premedication is required, which agent to use, and what dose to give. The calm cooperative patient often tolerates gentle reduction attempts of a major joint such as the shoulder, but even the most stoic of patients may be quite uncomfortable with the manipulations necessary for reduction of a dislocated finger. Attempting any reduction technique in an extremely anxious patient without premedication will generally frustrate the operator and further upset the patient, and it may hinder a successful outcome.

In most circumstances, analgesia and/or sedation of some sort will be used; generally the intravenous (IV) route for drug administration is the method of choice, as it allows for rapid relief of patient discomfort and facilitates repetitive dosing for the desired effect. As opioids and benzodiazepines are the most commonly used drugs in this setting, venous access also allows for rapid reversal with naloxone or flumazenil, respectively. Chapters 32 to 34 include a detailed discussion of anesthesia and analgesia for ED procedures.

Benzodiazepines are commonly used for muscle relaxation; the relatively short-acting midazolam is a popular agent. A combination of a benzodiazepine and opioid is

frequently used, but this combination increases the need for careful monitoring of cardiorespiratory status. Intramuscular ketamine can also be considered, generally in the pediatric population, to minimize emergence reactions. Respiratory depression in the setting of procedural sedation and/or analgesia generally occurs after completion of the procedure, when the patient's discomfort lessens. For this reason, close postreduction monitoring until the patient has regained alertness is essential.

Intra-articular lidocaine has been used for anesthesia prior to shoulder reduction. [2] With this approach, 20 mL of 1% lidocaine is injected intra-articularly 15 minutes prior to a reduction attempt. After local povidone-iodine preparation, a 3.8 cm, 22 ga-needle is placed 2 cm laterally and directly beneath the acromion. The needle passes from the lateral aspect of the shoulder over the humeral head in a slightly caudad direction to enter the shoulder joint. After aspirating any intra-articular hematoma, the lidocaine solution is slowly injected over 30 seconds. A previously unopened vial of lidocaine should be used for the injection and the operator should wait a few minutes postinjection for the local anesthetic to take effect. Such an approach can reduce postreduction time in the ED. [2] Distal extremity dislocations, particularly those of the digits, are quite amenable to nerve blocks for anesthesia. These may include simple digital blocks or more proximal blocks, such as at the level of the wrist (see Chapter 32) .

## GENERAL PRINCIPLES

The clinical assessment of the patient with a dislocation must include a search for other injuries, especially if the mechanism was of high energy. This is generally most important for hip, knee, and posterior sternoclavicular dislocations. For all dislocations, a detailed extremity neurovascular

examination should be conducted and appropriately documented prior to focusing attention on the injured joint.

Although most dislocations are clinically obvious, the history and mechanism of injury can be quite helpful in certain circumstances. For example, a painful shoulder joint in a seizure patient should prompt assessment for a posterior shoulder dislocation, whereas a history of the knee striking the dashboard will clue one to the potential for a hip dislocation. Some dislocations will have been reduced prior to physician assessment. A careful history will uncover these injuries and prompt the necessary assessment of the ligamentous integrity of the joint and guide proper immobilization and follow-up care. Dislocations that commonly present in a reduced state include finger dislocations, knee dislocations, patellar dislocations, and radial head subluxations.

Although the chance of a gentle reduction attempt causing a fracture is extremely low, prereduction radiographs of dislocated joints are generally recommended. Reasons for this include the difficulty in distinguishing a fracture-dislocation by clinical examination and the potential for medicolegal problems if the fracture is not identified prior to reduction attempts. More important, certain associated fractures predict a poor outcome from closed reduction and make orthopedic consultation a consideration prior to such attempts. The obvious exceptions to this rule include suspected radial head subluxation in young children, clinical circumstances in which radiographs are not readily available

(e.g., in the wilderness), and clinical conditions (i.e., vascular compromise or threatened skin penetration) that dictate the need for prompt reduction.

Postreduction radiographs are traditionally obtained, although the need for this in a clinically obvious successful shoulder joint relocation has been questioned. [3] Although postreduction films are often not clinically useful and may not be cost-effective, they are generally obtained for medicolegal purposes. Patients administered sedatives and opioids may not remember the actual successful reduction or the immediate postreduction period. A reinjury after release from the ED without the corroboration of a successful reduction can raise questions about the adequacy of the initial procedure. Occasionally a fracture is detected on postreduction radiographs that was not obvious on the initial films, or a previously noted minor fracture may be found to reside in an intra-articular location.

The proper terminology for dislocations describes the relationship of the distal (or displaced) segment relative to the proximal bone or the normal anatomic structure. The terms *anterior* and *posterior* are used in most dislocations. Therefore, if the head of the humerus lies anterior to the glenoid fossa, the injury is an *anterior shoulder dislocation*. Similarly, if the olecranon lies behind the distal end of the humerus, the injury is a posterior elbow dislocation. In the hand, wrist, and foot, one uses the terms *dorsal* and *volar*. *Palmar* and *plantar* are sometimes used in place of volar to describe the position of the dislocated part. Dislocations can be open or closed and may have associated fractures requiring separate description.

It is generally accepted that the sooner a dislocation is reduced the better. This alleviates the patient's discomfort and corrects the distortion of surrounding soft tissue structures. In some studies the success rate of relocation (reduction) is higher when attempted closer to the time of injury. [4] However, there is no reason to forego an attempt at a closed reduction due to time factors in the vast majority of dislocations. Chronic dislocations of several days, weeks, or more are often difficult to reduce in a closed manner, but such presentations are infrequent.

General points about the reduction itself include the need for patience on the operator's part and the avoidance of excessive or abrupt applications of force. Gentle and gradual application of the various reduction techniques lessen the risk of complications. The operator must clearly understand the technique to be applied, and one should not hesitate to review a description of the procedure on a regular basis. Review of the technique is most important for uncommon dislocations, but it is a good habit even for the more common dislocations performed by seasoned clinicians.

A certain percentage of all types of dislocations are not amenable to closed reduction. Inability to complete a closed reduction is generally a result of the interposition of soft tissue structures or fracture fragments and not necessarily due to improper technique. If one has achieved sedation/analgesia adequate to permit relaxation of the patient's muscle tone, reduction should be relatively straightforward. When reduction under adequate sedation/analgesia is unsuccessful, multiple attempts at closed reduction are inappropriate. Generally, orthopedic consultation should be considered after 2 failed

attempts.

Once an attempt at reduction is completed, the operator should recheck the neurovascular status. For the elbow, hand, and forefoot joints, passive range of motion is performed to assess the stability of the reduction and to ensure a smoothly gliding joint that is free of intra-articular obstruction. In addition to close monitoring of the medicated patient, proper aftercare involves adequate immobilization of the injured joint for comfort and to prevent repeat dislocation. Recommendations for follow-up care are dependent on the injury and its severity.

This chapter covers dislocations of the various joints with the exception of the wrist dislocations, which are complex and require orthopedic consultation. Assessment and management principles, including reduction methods, are presented and aftercare is discussed.

# SHOULDER DISLOCATIONS

The human shoulder joint is remarkable for its degree of possible motion. The anatomic features that allow for this mobility, however, contribute to its instability. The glenohumeral joint has the greatest range of motion of any joint in the body, largely due to the loose joint capsule and the shallow nature of the glenoid fossa. [5] Posterior dislocation is uncommon, largely due to the anatomic support of the scapula and the thick muscular support in this area. The anterior support is less pronounced, with the inferior glenohumeral ligament serving as the primary restraint to anterior dislocation. [6] The depth of the glenoid fossa is somewhat increased by the fibrocartilaginous glenoid labrum, which forms the rim of this structure.

Most shoulder dislocations are anterior (i.e., the humeral head becomes situated in front of the glenoid fossa). Posterior dislocations are the next most common, but they generally account for <2% of shoulder dislocations. [7] Uncommon variations include inferior (luxatio erecta), superior, and intrathoracic dislocations. Dislocations of all types, including the shoulder, are less common in children because of the relative weakness of the epiphyseal plate as opposed to the ligamentous support of the joint.

**Anterior Shoulder Dislocations**

Anterior dislocations of the shoulder are the most common major joint dislocation encountered in the ED. The usual mechanism of injury is indirect, with a combination of abduction, extension, and external rotation. [5] [6] Only rarely is the mechanism a direct blow to the posterior aspect of the shoulder. Occasionally, especially with recurrent dislocations, the mechanism is surprisingly minor, such as mere external rotation of the shoulder while rolling over in bed or raising the arm overhead. [8] The occurrence of a first dislocation at a younger age is associated with a high risk of recurrence. Rowe found a recurrence rate of 83% with a first dislocation before age 20 years vs 16% in patients with a first dislocation after age 40 years. [8]

The 3 types of anterior dislocations are subcoracoid (accounting for >75% of anterior

dislocations), subglenoid, and the uncommon subclavicular. [8] These are classified according to the location where the humeral head comes to rest (Fig. 52-1) (Figure Not Available) .

**Clinical Assessment**

The presentation of anterior shoulder dislocation is usually obvious (Fig. 52-2) . The patient supports the injured extremity and leans toward the injured side. Visual inspection reveals loss of the rounded appearance of the shoulder due to the absence of the humeral head beneath the deltoid region. The acromion is prominent and an abrupt drop-off below the acromion can be seen or palpated. An anterior fullness in the subclavicular region is visible in thinner individuals and is easily palpable in most others. Comparison to the uninjured side is a useful aid for both visual examination and palpation. Any attempt at internal rotation is quite painful and is resisted by the patient. The inability to place the palm from the injured extremity on the uninjured shoulder is consistent with a shoulder dislocation; postreduction, this maneuver should be possible.

A careful assessment of the neurovascular status of the affected extremity is essential. Injury to the axillary artery is rare, usually occurring in the elderly, [5] and can be quickly assessed by palpation of the radial pulse. It is important to assess the status of the axillary nerve, as this is the most common nerve lesion found in anterior dislocations. [9] The sensory component of the axillary nerve is assessed by testing for sensation over the lateral aspect of the upper arm (Fig. 52-3) (Figure Not Available) . The motor component of the axillary nerve would be tested by assessing the strength of the deltoid muscle, a difficult undertaking in the patient with a dislocated shoulder. The neurologic examination should include a complete assessment of all major nerves to the arm, as other nerve injuries such as to the ulnar and radial nerve may occur. [8] [9] The presence of a neurologic deficit does not preclude closed reduction. Nerve injuries in this setting have a good prognosis, but the patient should be informed of the findings and the need for follow-up.

**Radiologic Examination**

Associated fractures are detected in up to 24% of anterior shoulder dislocations, with fractures of the greater tuberosity being the most common. [8] The presence of a fracture of the greater tuberosity does not change the initial management of anterior shoulder dislocations, and these fractures usually heal well after closed reduction in the routine fashion. [5] Fractures of the humeral neck, however, frequently are displaced with attempts at closed reduction, the result of which is often avascular necrosis of the humeral head. [10] The fact that humeral neck fractures are a known complication of shoulder relocation [4] suggests the value of prereduction radiographs in anterior shoulder dislocations. However, it can be argued that a *clinically obvious recurrent dislocation without a high-energy injury mechanism* (a diagnosis that is usually offered by the patient) can be reduced without prior radiographs, as fracture is quite unlikely in this situation.

Anterior dislocations are not subtle on the routine anteroposterior (AP) radiograph, and this view detects the most important fracture to identify, that of the humeral neck. An

adequate AP view, when combined with the typical clinical examination, allows for successful management of most anterior shoulder dislocations. The true AP view of the shoulder is taken at a right angle to the scapula, requiring rotation of the patient to 30° to 40°, as shown in Figure 52-4 (Figure Not Available) .

The typical lateral views obtained include the scapular Y view (Fig. 52-5) (Figure Not Available) , the transthoracic view, or the axillary view. These views rarely add to the AP in the obvious anterior dislocation, but they are of value in *posterior* dislocations. The usefulness of additional views in anterior shoulder dislocations is primarily to detect fractures, and the previously mentioned lateral views (especially the transthoracic view) are quite limited in this respect. [11] The apical oblique view has been found to be more valuable in acute shoulder trauma than the oblique scapular projection. [11] This view is obtained by angling the beam 45° caudad with the patient in a 45° oblique position (Fig. 52-6 (Figure Not Available) *A* and *B*).

Postreduction radiographs are generally obtained to document the success of the reduction, and they occasionally reveal a fracture not detected on the prereduction radiographs. Harvey and coworkers have questioned the need for routine postreduction radiographs in clinically obvious successful reductions, as they rarely add new information. [3]

**Reduction Techniques**

Hippocrates (450 b.c.) is generally credited with the first detailed description of reduction techniques, and it is believed that a drawing on the Edwin Smith Papyrus (3000 to 2500 b.c.) is the earliest depiction of such a method. [5] The Hippocratic technique involves placement of the operator's foot in the axilla to effect countertraction. This technique is problematic and is not recommended by recent authors.  Likewise the Kocher method, which involves somewhat forceful leverage of the humerus, has an increased rate of complications and is generally discouraged in favor of other techniques. [5] [6]

This section discusses several methods of reduction that are well studied, proven to be safe, and easy to master. Although all techniques are generally acceptable, many authors state that their technique is quite painless, but only one study has quantified the actual pain reported by patients. [13] McNamara found that scapular manipulation was generally well tolerated; 62% of patients not receiving premedication reported no or only mild pain during the reduction. Additionally, pain ratings were not lessened in the premedicated group. [13] Unfortunately, in other reports of "painless" techniques, objective data are lacking. As noted previously, intra-articular lidocaine also may be used to reduce the pain of reduction (Fig. 52-7) . Regardless of the reduction technique used, gradual, gentle application of the technique is essential.

Operator judgment is an important part of the decision as to whether reduction should be attempted without premedication. The advantages of such an approach include the avoidance of potential complications from drug therapy and a more rapid patient disposition. Certainly the patient who is markedly intoxicated may require little if any supplemental sedative therapy. All patients who are more than mildly anxious or who are reluctant to cooperate with an attempt at reduction without medication should

receive premedication. Generally, only one attempt is made and, if unsuccessful, reduction is attempted with the use of medication. When in doubt, it is best to use pharmacologic adjuncts.

**Stimson maneuver.**

The Stimson maneuver (Fig. 52-8) (Figure Not Available) is a classic technique that offers the advantage of not requiring an assistant. The patient is placed prone on an elevated stretcher and about 2.5 to 5.0 kg (5 to 10 lb) of weight is suspended from the wrist. [5] [6] The weights can be strapped to the wrist, or a commercially available Velcro wrist splint can be placed and the weights hung from this with a hook. [14] The slow steady traction of this method often permits reduction, but it may take 15 to 20 minutes. Reduction may be facilitated by gentle external rotation of the extended arm.

Variations of this method include the recommendation for flexion of the elbow to further relax the biceps tendon and the application of manual traction instead of weights. Rollinson allowed the arm to hang under its own weight after a supraclavicular block and reported a 91% success rate with usually no more than a gentle pull on the arm after 20 minutes in this position. [17] Each of these variations of the Stimson method can be used in combination with the scapular manipulation technique described below. Indeed, a success rate of 96% has been reported using the combined prone position, hanging weights, IV drug therapy, and scapular manipulation. [14]

Disadvantages of the Stimson method include the time required and the danger of the patient's slipping off the elevated bed. A "seatbelt" strap or bedsheet may be placed around the patient and stretcher to avoid patient movement off the stretcher. Additionally, a bed that elevates to a suitable height for the patient's arm length, a convenient method to hang the weights, the weights themselves, and adequate staff to monitor the patient are often difficult to locate and organize in a busy ED.

**Scapular manipulation technique.**

This method is popular due to its ease of performance, reported safety, and acceptability to patients. No complications from this technique have been reported in the literature. Shoulder reduction using this method focuses on repositioning the glenoid fossa rather than the humeral head, and it and requires less force than other methods. [14] The success rate is high, generally >90% in experienced hands.

The initial maneuver for scapular manipulation is traction on the arm as it is held in 90° of forward flexion. This may be performed with the patient prone and the arm hanging down as described in the Stimson method, with or without flexion of the elbow to 90° (Fig. 52-9 *A*). Alternatively, this traction may be applied by the operator placing an outstretched arm over the seated patient's mid-clavicle while pulling the injured extremity with the other arm (Fig. 52-9 *B*). Regardless of the means of arm traction, slight external rotation of the humerus may facilitate reduction by releasing the superior glenohumeral ligament and presenting a favorable profile of the humeral head to the

glenoid fossa. [19]

The prone patient position is recommended for those not familiar with the technique, as it facilitates identification of the scapula for the second part of the method. Nonetheless, the technique can be performed with the patient supine, given that the patient's shoulder is flexed to 90° and the scapula is exposed during gentle upward traction on the humerus. [20] Although seated scapular manipulation offers the advantage of not requiring the patient to go through the awkward and potentially uncomfortable assumption of the prone position, it is a technically more difficult variation of scapular manipulation. When placing the patient in the prone position it is important to place the injured shoulder over the edge of the bed to allow the arm to hang in a perpendicular manner for the application of traction. [18]

After application of traction, the scapula is then manipulated to complete the reduction. Anderson and coworkers recommend manipulation of the scapula after the patient's arm is relaxed; [18] however, success is possible with no delay in the performance of this second step. [13] The manipulation of the scapula is carried out by stabilizing the superior aspect of the scapula with one hand and pushing the inferior tip of the scapula medially toward the spine (see Fig. 52-9 *A*). The thumb of the hand stabilizing the superior aspect of the scapula can be placed along the lateral border of the scapula and used to assist the pressure applied by the thumb of the other hand. The tip is pushed as far as possible in the medial direction, and a small degree of dorsal displacement of the scapular tip is also recommended. [18]

When the patient is properly positioned, with the affected arm hanging in a perpendicular fashion, the lateral border of the scapula may be difficult to find in larger subjects. This border is generally located quite lateral with the patient in this position, and it must be properly located prior to any reduction attempt. The reduction itself is occasionally so subtle that it may be missed by both the patient and the operator. A minor shift of the arm may be the only clue to the successful reduction. Careful palpation of the subclavicular area prior to repositioning the patient may be used to determine the success of the reduction.

**External rotation method.**

This method offers the advantage of requiring only one performer and no special equipment. The technique requires no strength or endurance on the part of the operator. Additionally, it has been reported to be well tolerated by patients. The actual pain experienced by patients with this technique has not been quantified, but Plummer and Clinton state it can be performed with "little, if any sedation." [12]

In this technique the basic maneuver is *slow*, *gentle* external rotation of the fully adducted arm. In 1957, Parvin described a self-reduction external rotation technique that he attributed to Boehler. [21] The patient sits on a swivel-top chair and grasps a fixed post at waist height and slowly turns the body to enact external rotation. Parvin reported that the reduction usually takes place at 70° to 110° of external rotation. [21]

More recently, this method has been described with the patient supine and the affected

arm adducted tightly to the side of the patient (Fig. 52-10) (Figure Not Available) . The elbow is flexed to 90° and held in the adducted position with the operator's hand closest to the patient. The other hand holds the patient's wrist and guides the arm into slow and gentle external rotation. The procedure may require several minutes, because each time the patient experiences pain, the procedure is momentarily halted. Although the report of Mirick and colleagues mentioned using the forearm as "a lever," [22] a later description clearly recommends allowing the forearm to "fall" under its own weight. [12] No additional force should be applied to the forearm and no traction is exerted on the arm.

The end point of the reduction may be difficult to identify, as reduction is frequently very subtle. It is therefore recommended to continue the external rotation until the forearm is near the coronal plane (lying on the bed, perpendicular to the body), a process that usually takes 5 to 10 minutes. [12] Reduction may occasionally be noted when the arm is rotated back internally. [23] The success rate of this technique in 3 series performed by emergency physicians was around 80%. One series reported that 36% of reductions could be performed without analgesia.

**Milch technique.**

There are many reports in the literature on the use of this method with praise for its gentle nature, high success rate, lack of complications, and tolerance by patients. [19] [25] [26] It can be described as "reaching up to pull an apple from a tree." The basic steps of this technique are abduction, external rotation, and gentle traction of the affected arm. Finally, if needed, the humeral head is pushed into the glenoid fossa with the thumb or fingers (Fig. 52-11) .

Milch, in describing this technique, wrote that the fully abducted arm was in a natural position in which there was little tension on the muscles of the shoulder girdle. [27] He postulated that this was related to our ancestral "arboreal brachiation" (swinging from trees). [27] The primary step in this technique is to have the affected arm abducted to an overhead position. Russell and coworkers had their patients raise the arm and put the hand behind the head as a first step. [19] Although this seems odd, patients can usually do this quite readily with little assistance and be quite comfortable in this position. Alternatively, the operator may abduct the arm by grasping the patient's arm at the elbow or the wrist. Lacey and Crawford found that the prone position, with the patient's shoulder close to the end of the bed, facilitated this step. [25]

Once the arm is fully abducted, gentle longitudinal traction is applied with slight external rotation. If reduction does not occur quickly, the humeral head can be pushed upward into the glenoid fossa using the thumb or fingers of the other hand. Beattie and associates reported a success rate of 70% with the Milch technique, [4] but others report success rates of 90%. [19] [26]

**Traction-countertraction.**

This method is commonly used in the ED, largely out of tradition, as it has a high rate of success and many emergency physicians are unfamiliar with other methods. Familiarity

is an advantage of this technique, but it requires more than one operator, some degree of force, and, occasionally, endurance. This technique is usually quite uncomfortable for the patient, and premedication is recommended prior to any attempt.

With the patient supine, a sheet or strap is wrapped around the upper chest and under the axilla of the affected shoulder (Fig. 52-12) (Figure Not Available) . An assistant holds this sheet so as to apply the countertraction. The operator's foot should *not* be used in the axilla to provide countertraction. Traction may then be applied to the extended arm, but this generally results in operator fatigue, especially if the operator relies on biceps strength to provide continuous traction. Preferably, the elbow of the affected side is flexed to 90° and a sheet or strap is wrapped around the proximal forearm and then around the operator's back. The bed should be elevated to a point at which the sheet can sit at the level of the operator's ischial tuberosities. This allows the operator to comfortably lean back and use the body weight to supply the force of traction, eliminating the possibility of operator fatigue. The portion of the sheet that is positioned on the patient's forearm has a tendency to ride up; flexion of the elbow beyond 90° will minimize this problem. Alternatively, the operator merely leans backward with the arms fully extended, using the continuous weight of the body rather than the strength of the biceps to provide constant traction.

Once traction is applied, the operator must be patient, as the procedure may take a number of minutes to be successful. Inadequate premedication is noted by the patient who resists the procedure or is notably uncomfortable during the reduction attempt. The operator should not hesitate to order supplementary medications. Gentle limited external rotation is sometimes useful to speed reduction. [5] Applying traction to an arm that is slightly abducted from the patient's body is often successful, but some operators prefer to slowly bring the arm medial to the patient's midline while maintaining traction, or have an assistant apply a gentle lateral force to the midhumerus to direct the humeral head laterally. Successful reduction is usually presaged by slight lengthening of the arm as relaxation occurs, and a noticeable "clunk" may occur at the point of reduction. A brief fasciculation wave in the deltoid may also be seen at the time of reduction.

**Other methods.**

Poulsen reported a method termed the *Eskimo technique*, which may be performed in field settings. [28] In this technique, the patient lies on the unaffected side and is lifted a short distance off the ground by grasping the abducted arm of the injured side. The patient's body weight acts to effect the reduction. Poulsen's success rate was 74% in a series of 23 patients, all of whom were premedicated. [28] Poulsen also postulated that this technique could place undue stress on the brachial plexus or axillary vessels. [28] Use of this technique, when other options are available, should probably be reserved until a larger experience is reported.

Noordeen and associates reported a simple method in which the patient sits sideways in a chair, with the affected arm draped over the backrest. [29] The operator holds the arm with the wrist supinated, and the patient is instructed to stand up. The success rate was 72% in 32 patients treated in this manner. [29] A variation of the chair technique, which was successful in 97% of 188 anterior shoulder dislocations, involves operator-applied

traction to the patient's flexed elbow by means of a cloth loop (e.g., stockinette). [30] While standing beside the patient, the operator supports the involved elbow, holding the cloth loop in 90° of flexion, while stepping down on the cloth loop. The patient sits in the chair, and an assistant may help support the patient by applying countertraction under the involved arm.

Waldron described a technique, without detailing the success rate, that is essentially a reverse of the Stimson method. [31] The patient is placed supine, the affected arm is forward flexed to 90°, and upward traction is applied at the distal humerus with the support of the epicondyles. The elbow is allowed to passively flex, and gentle internal and external rotation is applied through an arc of 20° total. [31]

**Postreduction Care**

After an attempt at reduction, the neurovascular status of the affected extremity should be rechecked and the results documented on the patient record. Indirect evidence that the reduction has been successful includes an immediate reduction in pain, restoration of the round shoulder contour, and increased passive mobility of the shoulder. No harm is done by putting the joint through a limited range of motion. If the patient can tolerate placement of the palm from the injured arm on the opposite shoulder, it is quite likely that the shoulder reduction was successful (Fig. 52-13) .

Postreduction radiographs are recommended, with a careful search for new fractures. Although most greater tuberosity fractures do not alter patient management, patients with greater tuberosity fractures displaced >1 cm after closed reduction should receive prompt orthopedic consultation, as they may require operative repair. [5]

It is important to prevent further external rotation or abduction of the reduced shoulder; adequate immobilization can be obtained by a commercially available shoulder immobilizer. Orthopedic follow-up is recommended for all anterior shoulder dislocations. The incidence of rotator cuff injury is as high as 38% [32] and may complicate restoration of normal function. Younger patients will generally be immobilized for at least 3 weeks and can be instructed to follow up within a week or so of the event. The older the patient, the shorter the time of immobilization. [5] Those older than 60 years should have early follow-up to allow for early mobilization and avoidance of shoulder joint stiffness.

It is appropriate to prescribe oral analgesics at the time of disposition and to instruct the patient to return for any worsening of the clinical condition. Periodically one may encounter a return visit from a successfully treated patient who is in severe pain from a hemarthrosis. Trimmings reported excellent relief of pain by aspiration of the hemarthrosis 24 to 48 hours after shoulder reduction in a series of patients older than 60 years. [33] This can be accomplished using the technique of arthrocentesis described in Chapter 57 . Intra-articular instillation of 10 to 20 mL of 1% lidocaine as has been recommended for shoulder reduction [2] may be helpful for further pain relief.

## Posterior Shoulder Dislocations

Posterior shoulder dislocations account for <5% of all shoulder dislocations. [7] Although uncommon, posterior dislocations are easily overlooked and the emergency physician must be knowledgeable about these injuries to avoid a misdiagnosis. Delays in diagnosis for weeks to months have been reported with posterior dislocations. The mechanism of injury is almost always indirect, with a combination of internal rotation, adduction, and flexion. [5] The precipitating events include seizure, electrical shock, and falls. The patient may also present at a point well past the original event. [35]

**Clinical Assessment**

While they are clinically less obvious than anterior dislocations, posterior shoulder dislocations do present in a typical, recognizable manner. Mistakes are made when the operator is overly reliant on the AP radiographs, which are potentially misleading. [5] The injury may be misdiagnosed as a soft tissue contusion or acromioclavicular strain. The principal signs of posterior dislocation are an arm that is somewhat fixed in adduction and internal rotation. Abduction and external rotation are limited, and attempts to perform these movements can elicit pain. [5] [7] Inspection and palpation reveal a loss of the normal anterior contour of the shoulder and a prominent coracoid and acromion. The shoulder is flattened anteriorly and rounded posteriorly, where the humeral head may be palpable.

Comparison to the opposite shoulder should be undertaken with the understanding that this injury may occasionally occur bilaterally. Neurovascular assessment is performed in the standard manner, although such complications are unusual with posterior dislocations.

**Radiologic Examination**

The key point regarding radiographs for posterior shoulder dislocations is the subtle nature of this dislocation on a single AP radiograph (Fig. 52-14 (Figure Not Available) *A* and *B*) and the diagnostic value of the scapular Y view (see Fig. 52-5 (Figure Not Available) *C*) or the axillary view (Fig. 52-14 (Figure Not Available) *C*). The diagnosis of posterior shoulder dislocation using the axillary view is quite easy, whereas the routine AP and lateral views are difficult to interpret in around half of cases. [35] The axillary view is generally available in the radiology department and can be obtained with as little as 20° to 30° of abduction, with the plate placed on the shoulder. [35] In addition to easy visualization of the posteriorly situated humeral head, the axillary view often reveals an impression fracture of the humeral head (Fig. 52-14 (Figure Not Available) *D*).

Whereas the axillary view is diagnostic, clues to posterior dislocation exist on the AP film. The internally rotated humeral head appears symmetrical on the AP film in the shape of a light bulb as opposed to the normal club-shaped appearance created by the greater tuberosity. [36] With posterior dislocation, the space between the articular surface of the humeral head and the anterior glenoid rim is widened, and there is a decrease in the half-moon-shaped overlap of the head and the fossa (Figs. 52-15 (Figure Not Available) and 52-16) (Figure Not Available) . [34] [36] There may also be a compression fracture of the medial aspect of the humeral head, indicated by a dense line. This is

known as the "trough" sign. [36] A fracture of the lesser tuberosity (see Fig. 52-16) (Figure Not Available) should always prompt a search for the presence of a posterior shoulder dislocation. [34]

### Reduction Technique

An acute posterior dislocation may be reduced by traction on the internally rotated and adducted arm combined with posterior pressure on the humeral head. [5] [35] Premedication is generally indicated and countertraction may be applied with a sheet looped in the affected axilla much as described for anterior dislocations. Neer and Rockwood recommend applying lateral traction on the upper humerus if reduction is delayed. [5] Hawkins and coworkers suggest that posterior dislocations with an impression defect of the humeral head that is >20% of the articular surface require open reduction. [35] Posterior dislocations that have been diagnosed late are difficult to reduce in a closed manner, but an attempt with adequate premedication is generally indicated. [35]

### Postreduction Care

As with anterior dislocations, a repeat neurovascular examination and radiographs are obtained after reduction attempts. As before, the patient's ability to place the palm of the injured arm on the opposite shoulder is suggestive of a successful reduction. Given the rarity of these injuries, orthopedic consultation is often sought early in the care of these patients. Certainly in a training environment, involvement of an orthopedic resident benefits his or her education and should be considered early on. If a successful reduction is unstable, immobilization with application of external rotation, often via a spica cast, will be needed.

## Unusual Shoulder Dislocations

Inferior dislocations of the shoulder, known as *luxatio erecta*, are quite rare, but also quite obvious. The patient presents with the arm locked in marked abduction with the flexed forearm lying on or behind the head. [5] Occasionally, the humerus may have less abduction, thus potentially obscuring the diagnosis. [37] The humeral head can be palpated along the lateral chest wall. Neurovascular compression may be present, but this is usually reversed once reduction is accomplished. [5] Overhead traction (generally with the arm in full abduction) is applied in the longitudinal direction of the arm and cephalad pressure can be exerted over the humeral head much as in the Milch technique. [5] [37] Countertraction toward the patient's feet can be applied using a sheet placed over the injured shoulder. After reduction, the abducted arm is brought into adduction against the body and the forearm supinated. [38]

Scapular dislocation or "locked scapula" is a rare condition that presents with an obvious protrusion of the lateral border of the scapula and significant swelling of the medial border due to tearing of the musculature. [39] Reduction is accomplished by traction on the abducted arm and medial pressure on the scapula.

# ACROMIOCLAVICULAR SUBLUXATION AND DISLOCATIONS

The acromioclavicular (AC) joint is a true diarthrodial joint with a synovial cavity surrounded by a relatively lax capsule and the weak acromioclavicular ligament. This structure allows for the necessary gliding motion for shoulder movement. [40] The major stability of the AC joint comes from the coracoclavicular ligament, which has posterior (conoid) and anterior (trapezoid) components. The mechanism of injury is generally from a direct force such as a fall on the point of the shoulder. [5] There are 4 grades of injury to the AC joint; they are classified by degree or type (I through IV) (Fig. 52-17)

## First degree (type I).

This injury consists of a minor tear in the AC ligament. The coracoclavicular ligament is intact. The clinical findings are limited to tenderness in the area of the AC joint. Radiographs show little if any change in the position of the clavicle in relation to the acromion. [40] The management of this condition consists of a sling for comfort, ice, and mild analgesics. The sling can usually be discontinued after 7 to 10 days. [5] Orthopedic referral is generally not necessary unless return to normal function is delayed beyond 2 weeks.

## Second degree (type II).

In addition to a complete tear of the AC ligament, the coracoclavicular ligament in this injury is stretched or incompletely torn. [40] The patient generally supports the injured arm and has slight swelling and definite tenderness over the AC joint. Radiographs demonstrate a definite change in the relationship of the distal clavicle to the acromion. The inferior edge of the clavicle should not be higher than the top of the acromion in this injury. [40] Others use the radiographic criteria that the coracoclavicular distance is within 5 mm of the uninjured side. [5] This injury is usually treated in a closed fashion with a sling. [5] Orthopedic referral is recommended, and some will use a sling-strap device that elevates the arm and depresses the clavicle for these injuries. [41]

## Third degree (type III).

In this injury, the distal end of the clavicle is essentially free floating, as both the AC and coracoclavicular ligament are completely disrupted. [40] The arm is supported by an uncomfortable patient and the distal clavicle is usually seen to be riding high above the acromion. The diagnosis is generally obvious, and radiographs are mainly used to rule out an associated fracture. Radiographic criteria for this degree of injury include an inferior border of the clavicle above the acromion [40] or a difference of 5 mm in the coracoclavicular distance as compared with the normal side. [5] These injuries require orthopedic referral, and a fair bit of controversy exists regarding the subsequent management of these injuries. [40] Larsen and colleagues conducted the only prospective, randomized trial of conservative vs operative management for significant AC separations and concluded that conservative management was generally better, with possible exceptions made for patients with significant cosmetic deformity and for

those who frequently keep the arm at 90° of abduction. [42]

**Fourth degree (type IV).**

In this injury, the distal clavicle is free floating and posteriorly displaced into the mass of the trapezius muscle. Orthopedic referral is required.

### Radiographic Examination

The diagnosis is usually made clinically, with pain and local tenderness at the AC joint in the absence of other findings. Radiographs are generally indicated to rule out associated fractures and to aid in assessing the degree of injury. In patients with minor clinical findings clearly suggestive of a first-degree injury, radiographs may not be necessary. A single radiograph of the injured shoulder usually suffices, but some clinicians prefer to obtain comparison views of the opposite shoulder. The value of comparison views remains uncertain. It has been traditionally recommended that "weighted" films be obtained in suspected type I or II injuries. [5] Weighted films are superfluous in clinically obvious type III injuries. Weighted films after "unweighted" routine radiographs are obtained by strapping about 4.5 to 7.0 kg (10 to 15 lb) of weight to the patient's wrists and repeating the radiographs. It is important that the patient keep the shoulders as relaxed as possible during this study. As expected, this study may cause significant discomfort for the patient.

Bossart and others examined the routine use of "weighted" studies of the AC joints and recommended abandoning their use in the ED. [43] In a prospective study of 70 type I or II injuries, the use of weights was associated with less evident separation in 7 cases. Only 3 injuries were re-categorized as type III after the performance of weighted films. [43] This yield is not necessarily inconsequential if subsequent management would entail a change in therapy for these 3 patients. However, Bossart and colleagues noted that surgery is often not recommended with type III injuries. [43] It is probably best to discuss the merit of weighted films with the consultant orthopedists and to obtain the study only in the selected cases in which open treatment would be dictated by a type III injury.

## STERNOCLAVICULAR DISLOCATIONS

Despite the fact that the sternoclavicular joint is the least stable joint in the body, sternoclavicular dislocations are rare. [5] The primary supports of this joint are the sternoclavicular and costoclavicular ligaments. [41] Anterior dislocations are much more common and are usually the result of an indirect mechanism involving a blow to the anterior shoulder. [41] Posterior dislocations also usually result from a blow to the shoulder but can also be the result of a direct superior sternal or medial clavicular blow. [5] Posterior sternoclavicular dislocation is potentially life-threatening, as injury to the great vessels or compression of the airway may occur. [5] [44] Any suggestion of these complications should prompt immediate surgical consultation.

The presentation of these injuries is usually straightforward, with pain, swelling, tenderness, and deformity of the joint. Plain radiographs of this joint are difficult to

interpret and generally include an apical lordotic-type view with the radiographic tube angled at 45° cephalad. Confirmation of the diagnosis is best made using a thoracic computed tomographic (CT) scan. [45] Children may have epiphyseal disruption with retrosternal displacement of the medial clavicle. [46]

Closed reduction of both types of sternoclavicular dislocation involves placing a rolled blanket or a sandbag between the scapula and applying traction to the 90° abducted arm in line with the clavicle. [5] The clavicle can also be pushed (anterior) or lifted (posterior) back into position. [5] Posterior dislocations may be difficult to reduce and to maintain reduced in a closed manner [44] and some recommend general anesthesia for all of these. [5] Given the rarity of this injury and the potential for major underlying complications, early consultation is recommended in suspected posterior sternoclavicular dislocations

# ELBOW DISLOCATIONS

The elbow is second only to the shoulder as a site for major joint dislocations in adults, and it is the most commonly dislocated joint in children. Anatomically, the principle articulation of the humerus and ulna is a stable hinge joint with the intercondylar groove of the distal humerus nestled in the olecranon fossa. Due to the stability of the elbow, any dislocation is expected to be accompanied by significant soft tissue damage, and associated fractures are common. Elbow dislocations are often simply divided into posterior and anterior dislocations (Fig. 52-18) (Figure Not Available) . However, there are actually several different types of elbow dislocations, including the two just mentioned as well as medial, lateral, divergent, and isolated dislocations of the radius. [47] In the rare divergent dislocations, the radius and ulna are dislocated in opposite directions, either anterior and posterior or medial and lateral. [47]

The most feared complication of elbow dislocation is injury to the brachial artery. This complication can occur with any type of elbow dislocation and is a frequent occurrence in open dislocations. [48] Vascular compromise can be delayed in onset either from unsuspected arterial injury or from progressive soft tissue swelling. The circulatory status of the arm must be carefully monitored even after successful reduction. [47] Although not an absolute, patients with these injuries are frequently admitted to the hospital for observation, and orthopedic consultation should be sought prior to disposition. [47]

Injury to the median and ulnar nerves may be the result of stretch, severance, or entrapment. It is difficult to clinically distinguish these etiologies, and management of nerve problems is frequently expectant. [47] It is imperative to conduct a careful neurologic examination before and after reduction, as any increase in findings would indicate entrapment and the need for surgical intervention. [47] Myositis ossificans is also a potential complication of this injury, which underscores the advisability of orthopedic consultation early in the course of care.

**Posterior Dislocations**

Posterior dislocations make up the vast majority of elbow dislocations [49] and most often are actually posterolateral dislocations. [47] The usual mechanism is a fall on the outstretched hand, with the arm in extension. The clinical examination is usually diagnostic unless severe soft tissue swelling is present. The patient presents with a shortened forearm that is held in flexion, and the olecranon is prominent posteriorly. The normally tight triangular relationship of the olecranon and the epicondyles of the distal humerus is disturbed in a posterior dislocation. A defect may also be palpated above the prominence of the olecranon. [47]

**Radiologic Examination**

Two radiographic views, preferably an AP and a true lateral view, are obtained. The diagnosis is obvious with proper radiographs. A careful search for fractures is undertaken, as they occur commonly in this injury. In one series of 60 elbow dislocations, 58 of which were posterior, 39% of patients had an associated fracture. [49] In children younger than 14 years, the fracture is usually a medial epicondyle separation, as the epiphyseal plate gives way before the medial collateral ligament of the elbow. [49] Postreduction radiographs are also necessary.

**Reduction Techniques and Postreduction Care**

Although, as with shoulder reduction, many authors claim that their method of reduction is virtually painless, this has not been objectively documented. In general, patients with posterior elbow dislocations are quite uncomfortable, and it is beneficial to administer IV analgesics early in the course of care, preferably prior to positioning for radiographs.

In addition to, or in lieu of, parenteral analgesia, some clinicians inject the elbow joint with a local anesthetic (e.g., 3 to 5 mL of 2% plain lidocaine) prior to attempting reduction. This should be done using strict sterile technique, with a previously unopened anesthetic vial. It may require 10 to 15 minutes for the anesthesia to take effect. The technique is similar to the technique used for aspiration of the elbow joint (see Chapter 57) . Prior to injection, the joint should be aspirated to remove blood.

**Traditional traction method.**

The traditional method  of reducing a posterior dislocation is to place the patient in the supine position and have an assistant stabilize the humerus with both hands (Fig. 52-19) (Figure Not Available) . The operator then grasps the wrist and applies slow and steady in-line traction. The elbow is slightly flexed to keep the triceps mechanism loose, and the wrist is held supinated as traction is applied. Reduction is usually signified by a "clunk" that is heard or felt. If this method is not successful after a reasonable period of traction (10 minutes), the forearm may be gently flexed to try and effect reduction. Alternatively, downward pressure on the proximal volar surface of the forearm may help free up the coronoid process.

Several authors have described variations of a prone method of reduction that is reportedly well tolerated by patients. [21] [50] [51] The patient is positioned with the arm hanging over the padded back of a chair or over the edge of the bed. Minford and Beattie simply applied pressure to the prominent posterior aspect of the olecranon in this position for successful reduction. [51] Lavine applied traction with the elbow flexed over the edge of a chair by pulling down on the hand while using the thumb to guide the olecranon into place (Fig. 52-20 *A*). [50] Parvin positioned the patient as for the Stimson method of shoulder relocation and applied gentle downward traction to the wrist. [21] When the olecranon was felt to ride distally on the humerus, the reduction was completed by lifting the humerus.

**Recommended initial approach.**

A prone technique is advantageous, as patients tolerate this position quite well. The elbow is allowed to hang flexed over the edge of the bed, and an assistant is positioned with the back toward the patient such that the humerus can be encircled with both hands and pressure applied with the thumbs to the posterior aspect of the olecranon. This pressure on the olecranon is intended to lift it up and away from the humerus. The operator applies longitudinal traction to the arm with the elbow in slight flexion. If traction is not succeeding, an attempt may be made to flex the elbow or the assistant can be instructed to lift the humerus (Fig. 52-20 *B*). Reduction is generally noted by a definite "clunk."

**Postreduction care.**

Once reduction is achieved, the elbow should immediately be put through a gentle range of motion to ensure that the reduction is stable and that there is no mechanical block to movement. [47] An inability to move the elbow through a smooth range of motion after reduction is often caused by a trapped medial epicondyle fracture that requires operative intervention. [47] The elbow is generally immobilized in at least 90° of flexion with a long-arm posterior splint. A complete recheck of the neurovascular status is performed along with postreduction radiographs.

Any delayed vascular compromise in the reduced elbow is first addressed by loosening the splint and decreasing the degree of flexion. This usually restores the pulse. [47] The risk of vascular compromise is a reason to consider in-hospital observation. Some operators observe the patient in the ED for 2 to 3 hours postreduction, evaluating for delayed neurovascular compromise prior to consideration of release to home.

## Anterior Dislocations

Anterior dislocations of the elbow are quite rare; they usually result from a direct posterior blow to the olecranon with the elbow flexed. [47] On physical examination, the arm is extended and there is anterior tenting of the proximal forearm with prominence of

the epicondyles posteriorly and an absent olecranon. [47] These injuries are the result of a great deal of force; they are frequently open and accompanied by significant neurovascular injury. An avulsion of the triceps mechanism may also occur.

Reduction of an anterior dislocation of the elbow involves in-line traction and backward pressure on the proximal forearm (Fig. 52-21) (Figure Not Available) . An assistant provides countertraction by grasping the humerus with 2 hands. Given the infrequent nature of anterior dislocations and the high probability of a severe associated injury, the emergency physician should consider early orthopedic consultation in such dislocations.

## Radial Head Subluxation (Nursemaid's Elbow)

Radial head subluxation is a common pediatric presentation generally occurring between the ages of 1 and 3 years. The mean age of presentation is just over 2 years, but this entity has been reported in infants younger than 6 months [52] [53] and in older children up to the preteen years. [54] There is a slight predilection for this injury to occur in girls and in the left arm. [53] [54] The classic mechanism of injury is longitudinal traction on the arm with the wrist in pronation, as occurs when the child is lifted up by the wrist. [53] There is no support for the common assumption that a relatively small head of the radius as compared to the neck of the radius predisposes the young to this injury. [55] The pathologic lesion is generally a tear in the attachment of the annular ligament to the periosteum of the radial neck, with the detached portion becoming trapped between the head of the radius and the capitellum. [55]

### Clinical Assessment

The history offered by the caretaker may not be that of the classic pulling type mechanism. Schunk, in a series of 83 patients, reported that only 51% described such a mechanism, whereas 22% reported a fall. [53] In patients younger than 6 months, the mechanism in the majority is simply rolling over in bed. [52] It is important to remember this and not to proceed with a child abuse investigation unless other suggestive features are present. The typical patient with a nursemaid's elbow presents in no distress with the arm held slightly flexed and pronated at the side (Fig. 52-22) (Figure Not Available) . This has been termed the *nursemaid's position*. [56] The exact area of pain is often difficult to locate. The child will refuse to use the arm, and this may be the chief complaint. [54] The older child will usually point to the dorsal aspect of the distal forearm when asked where it hurts. This may mislead one to suspect a buckle fracture of the distal radius.

Although tenderness about the elbow has been reported occasionally, [53] there is often little tenderness or swelling of the elbow region. [53] [54] In the cooperative child, the arm and shoulder are carefully palpated to discern any tenderness. Areas of focus on palpation should include the clavicle and the distal radius, as these are common sites of pediatric fractures. When patient anxiety interferes with a reliable assessment of tenderness in a child whose arm is in the classic nursemaid's position, the examiner can stand at a distance and have the parent or caretaker palpate the extremity to ascertain tenderness. This may also be done in the cooperative patient to reassure the doubtful parent regarding the absence of a fracture. If no tenderness is noted by palpation, it is

appropriate to attempt a reduction without prior radiographs. Although resistance to or pain with supination is a frequent finding in such patients, [53] one need not test for this finding until the time of reduction.

**Radiographic Examination**

Radiographs are generally not needed in a child presenting with an arm in the nursemaid's position that is nontender (or minimally tender in the radial head area) on palpation, regardless of the history. Radiographs are generally normal when obtained and, if obtained, the positioning of the child's arm by the x-ray technician for these studies often effects reduction. [53] Frumkin described 3 cases of nursemaid's elbow in which a line drawn through the longitudinal axis of the radius did not normally bisect the capitellum on prereduction radiographs, but did so after reduction. [55] Others recommend performing radiographs if the child is not moving the arm normally 15 minutes after reduction. [56] However, this time frame may be too short, as reuse can be delayed for >30 minutes. Quan and Marcuse recommend an approach in which no radiographs are obtained on the first visit, including in those children released from the ED prior to full use of the arm. At the time of a 24-hour follow-up visit, Quan and Marcuse perform radiographs only if repeat attempts at manipulation are not successful. [54]

While this condition does not generally require x-rays, they can be valuable in atypical presentations if external signs of trauma are present (e.g., swelling, abrasions, ecchymoses), or if the child does not use the arm normally after the subluxation is considered reduced. Other less common conditions that can present with similar findings are fractures, joint infections, tumors, or osteomyelitis.

**Reduction Technique**

Reduction of a nursemaid's elbow (Fig. 52-23) (Figure Not Available) is generally performed without premedication. It is important to explain to the caretaker that the reduction will likely cause the child discomfort, but that this is transient and a clue to the diagnosis. The child is positioned seated on the lap of an assistant who stabilizes the arm by holding the humerus adducted to the side. The operator then grasps the elbow with one hand placing the thumb over the region of the radial head. Although it has been stated that the thumb can apply pressure to the radial head, this positioning is mainly useful for palpation of the reduction "click." The other hand grasps the wrist and is then used to supinate the extended forearm in a steady, deliberate manner. Slight traction prior to supination is generally recommended, but it is unclear whether this actually adds to the supination maneuver. Once supinated, the arm can be flexed or extended; however, flexion is the most common maneuver and may actually be somewhat more successful than extension. [53] An audible or palpable click signifies successful reduction, but it is not always noted. Once the reduction has been performed, the child usually cries for a few minutes. Generally the operator should leave the room and then return in 10 to 15 minutes to do a repeat examination.

If a click is detected, the child will generally regain use of the arm quickly (almost always by 30 minutes). [54] Therefore, if a definite click is detected, it is reasonable to observe

the child for up to 30 minutes prior to further intervention. If there is still no use at 30 minutes, the operator may try to determine if supination is still painful, which would suggest the need for a repeat attempt. In those in whom a click is not detected, the majority will not use the arm by 30 minutes. [54] If a click was not detected, it is suggested to repeat the attempt after 10 to 15 minutes of nonuse. Two or more attempts are required to produce the click in up to 30% of patients. [54]

If the child has not regained the use of the arm after a few attempts and a reasonable period of time, radiographs can be obtained. [56] X-ray films also may help relieve parental anxiety. Alternatively, instructions should be given for 24-hour follow-up if normal function is not restored, with consideration for radiographs at the time of follow-up. [54] In 2 series of patients with nursemaid's elbow, [53] [54] of 10 patients released without normal arm use, 6 had spontaneous restoration of function, and the other 4 required remanipulation, which successfully restored function. The use of a posterior splint to protect the elbow of the child who refuses to use the arm after a presumed reduction is of uncertain value. Some form of immobilization (e.g., splint, sling, or both) may be valuable in the child with significant residual discomfort following a prolonged period of subluxation or in whom recurrent subluxations have occurred. On occasion, a successful reduction painfully resubluxates with movement; in this case, immobilization and referral may be necessary. [55] If reduction has been achieved clinically and maintained in the ED, analgesics or a follow-up visit are unnecessary. Because other pathology can *rarely* mimic this condition (e.g., occult fractures, osteomyelitis, joint infection, and tumors), full, unrestricted, and painless use of the arm must be documented at either the initial ED visit or at a follow-up visit within 24 hours.

## HAND INJURIES

The hand is an extremely common site of injury due to the demands placed on it and the exposed nature of this region. Proper motion and function of the hand are intimately related to normal anatomic alignment. [57] The emergency physician must therefore be skilled in the diagnosis and management of dislocations about the hand. An improperly managed hand injury can result in significant disability that the patient is reminded of on a daily basis.

Anatomically, the joints of the digits are quite similar and consist of a hinge joint with a tongue-in-groove type arrangement. [57] The soft tissue support includes 2 collateral ligaments that are attached to a volar plate (Fig. 52-24) (Figure Not Available) . The volar plate is dense fibrous connective tissue that is thickened at its distal attachment and thinner at its proximal attachment, to allow for folding with joint flexion.  Dorsal dislocation of a digit requires failure of the volar plate, whereas lateral dislocation disrupts a collateral ligament and induces at least a partial tear in the volar plate (see Fig. 52-24) (Figure Not Available) .

Radiographic examination of all hand injuries is relatively straightforward, including at least 2 views of the injured area. The most important radiographic error is failing to get a true lateral view of the injured joint. [58] This may lead to missing a fracture or a loose body in the joint.

Anesthesia is generally required for the proper management of dislocations about the hand. This is most often accomplished by finger or wrist block, although a more proximal regional or Bier block may be used on occasion (Chapters 32 and 34) . Getting a secure grip on the digits may be difficult and may complicate the reduction. Wearing rubber gloves or wrapping gauze around the fingers may be useful.

## Thumb Dislocations

The opposable thumb is an essential structure for countless activities. Despite its strong ligamentous and capsular support, the exposed positioning of the thumb makes it a frequent site of dislocations and subluxations. The metacarpophalangeal (MCP) joint is similar to those of the fingers but has a stronger volar plate and collateral ligaments.

### Interphalangeal (IP) Joint Dislocation of the Thumb

The single interphalangeal (IP) joint of the thumb has strong cutaneous-periosteal attachments, and dislocations of this type are therefore frequently open. [58] Dislocations are generally dorsal and can be reduced in a manner similar to IP dislocations of the finger (Fig. 52-25) (Figure Not Available) . The mechanism of injury is recreated by longitudinal traction and hyperextension to distract the phalanges. Reduction is accomplished by flexing the finger with continued traction and by applying direct pressure to the base of the distal phalanx. [58]

Following reduction, the range of motion is tested and the stability of the reduction is ascertained. An adequate reduction that is documented on postreduction films is then splinted in slight flexion for 3 weeks. [58] Orthopedic referral is advisable.

### Metacarpophalangeal Joint Injury of the Thumb

#### Dorsal dislocation.

The MCP joint of the thumb can be dorsally dislocated by a hyperextension injury. The proximal phalanx will come to rest in a position dorsal to the first metacarpal (Fig. 52-26) (Figure Not Available) . There are two basic types of MCP dislocations (this applies to the fingers also): simple and complex. The simple type is amenable to closed reduction, whereas the complex requires operative reduction due to interposed soft tissue. Additionally, a simple MCP dislocation can be converted into a complex one during reduction. [57] In a complex MCP dislocation, the volar plate becomes entrapped dorsal to the metacarpal head (Fig. 52-27) (Figure Not Available) with the flexor tendons and lumbricals acting to complete the trapping of the metacarpal head. [58]

Clinical features that suggest a complex MCP dislocation include a proximal phalanx that is less acutely angulated than with a simple dislocation (i.e., <60°). [58] There may also be dimpling on the palmar skin due to pressure from the entrapped metacarpal head. This will be seen over the thenar eminence of complex thumb MCP dislocations. [57] On radiographic studies of simple dislocations, the joint surfaces are in close contact,

whereas they are separated in complex dislocations. The presence of a sesamoid bone in the joint space is diagnostic of a complex MCP dislocation (Fig. 52-28) (Figure Not Available) . [57]

Reduction of a simple MCP dislocation (Fig. 52-29) (Figure Not Available) involves hyperextension of the joint as far as possible with the wrist in flexion to relax the tendons. Once maximal hyperextension is achieved, the base of the proximal phalanx is pushed distally while the joint is brought back into flexion. [57] Applying simple traction alone as an initial maneuver risks trapping the volar plate and creating a complex dislocation. [57] After reduction, the stability of the reduction is tested by placing the joint through a range of motion, and the integrity of the collateral ligaments can be assessed with the MCP joint in flexion (see below). Simple MCP dislocation injuries generally require casting for 3 weeks with the joints in moderate flexion. [58]

**Ulnar collateral ligament rupture.**

Also known as *gamekeeper's* or *skier's thumb,* this injury results from a laterally directed force at the thumb MCP joint. A rupture of the ulnar collateral ligament of the MCP joint occurs (Fig. 52-30) (Figure Not Available) . The usual mechanisms include falling with a ski pole in hand or having the thumb alone draped over the steering wheel in an auto crash. These injuries most often present in the reduced state with just the complaint of pain in the area. It is essential to recognize this injury to prevent further disability in the patient, as this ligament is important for the grasping function of the thumb.

The diagnosis is generally made through stress testing of the MCP joint (Fig. 52-31) . Radiographs occasionally demonstrate an avulsion-type fracture. The exact positioning of the thumb for stress testing is debatable, but the metacarpal should be stabilized with the thumb and index finger of one hand and stress applied with the other hand. Louis and coworkers recommend testing this joint in full flexion, as virtually no lateral movement of the MCP joint should be noted in this position. [59] Instability in full flexion of >35° was found to be indicative of complete rupture. [59] Others suggest testing the MCP joint in 20° to 30° of flexion to lessen the stabilizing effects of the volar plate; the results should be compared with stability on the other side. [58]

Partial injures to the ulnar collateral ligament are generally casted for 3 weeks; complete rupture usually requires operative repair. [59] An associated non-displaced fracture may be treated closed; displacement is an indication for operative repair. [59]

**Carpometacarpal Dislocations of the Thumb**

Carpometacarpal dislocations of the thumb are uncommon; when present, they often occur with an associated fracture. Closed reduction is generally unstable, usually necessitating operative stabilization through the percutaneous placement of Kirschner wires. [58]

## Finger Dislocations

The basic anatomic structure of the fingers is similar to that of the thumb with the exception that there is more lateral support of the MCP joints, making collateral ligament injury here much less common than in the thumb. The treatment principles are also similar. It is advisable to order radiographs for a specific finger (not just "hand" films). Complete views of the finger will include an AP view, a true lateral view, and an oblique view. The true lateral view is extremely important for detection of subtle dislocations or small avulsion fractures on the volar surface (Fig. 52-32 (Figure Not Available) *A* and *B*).

**Proximal Interphalangeal Dislocations**

The proximal interphalangeal (PIP) joint is extremely important, and any loss of motion in this joint severely restricts normal function. [57] This joint is also prone to stiffness, so careful treatment of injuries to this area is essential. Injuries to the PIP joint are generally slow to heal and often result in an increase in joint size due to scar tissue formation. [57] Because of this propensity for a less-than-perfect outcome, it is advisable to refer PIP injuries after emergency care.

Proper examination of the PIP joint (Fig. 52-33) (Figure Not Available) includes ulnar and radial stress to test the integrity of the collateral ligaments and hyperextension to determine the integrity of the volar plate. The inability to actively extend the flexed PIP joint against resistance suggests a central slip rupture, which may progress to a boutonniere deformity. [57] Such an examination should be carried out after any successful joint reduction. This examination should also be conducted in the painful PIP joint that is radiographically normal to detect soft tissue injury in a spontaneously or field-reduced dislocation. This is extremely important in athletes, as coaches often reduce these injuries. [57]

**Dorsal PIP dislocations.**

These are one of the most common types of dislocations encountered in the ED. The mechanism is usually a blow to the end of the finger, such as from a thrown ball, which creates an axial load and hyperextends the finger. [58] The middle phalanx comes to rest dorsal to the proximal phalanx (Fig. 52-34) . There is always an associated disruption of the volar plate. [57] [58] The deformity is obvious on clinical examination, and radiographs clearly demonstrate the injury. An associated fracture of the volar lip may be detected. If this fracture affects >33% of the joint surface, a closed reduction will be unstable and operative repair will be necessary, as the collateral ligament is attached to the bony fragment. [58]

A dorsal PIP dislocation can be reduced after a finger block. The usual method (see Fig. 52-25 (Figure Not Available) *E-H*) is to exaggerate the injury by slight traction and hyperextension, thereby distracting the injury. One can then apply pressure to the base of the middle phalanx as the finger is brought into flexion. These injuries usually reduce fairly easily, and failure of routine attempts should raise the suspicion of interposed soft tissue, for which an orthopedic consultation should be sought.

After reduction is completed, the joint should be placed through a range of motion to ensure stability of the reduction. If stable, the joint is generally splinted in flexion of 20° and 30° for 3 weeks. [57] [58] As PIP injuries can be slow to heal and are complicated by stiffness, it is advisable to refer patients with these injuries for orthopedic follow-up.

**Volar PIP dislocations.**

These are uncommon injuries and virtually always are accompanied by an injury to the central slip of the extensor tendons, which can lead to a boutonniere deformity if improperly immobilized (see Chapter 51) . [57] It is generally best to seek early orthopedic consultation for these injuries, as many require operative repair. If closed reduction is accomplished by the emergency physician, splinting in extension should be undertaken for 3 weeks, with early orthopedic follow-up. [57]

**Lateral PIP dislocations.**

These injuries are fairly common and are often reduced in the field. The patient will often dramatically describe how the finger was pointing in an unnatural manner. The injury can be detected by ulnar and radial stress of the PIP joint. If still dislocated, reduction is usually easily accomplished by recreating the injury and applying longitudinal traction to the finger. Partial tears of the collateral ligaments can be treated by buddy taping the finger for 3 weeks. [57] The management of complete tears is controversial, with some using operative therapy for all such injuries. [57] Referral is suggested for all but the mildest of PIP collateral ligament injuries.

**Distal Interphalangeal Dislocations**

As in the thumb, the distal phalanx is attached firmly to skin and subcutaneous tissue by osteocutaneous fibers. For this reason, dislocations of the distal interphalangeal (DIP) joint are frequently open. [57] A DIP dislocation is usually dorsal, and the mechanism is a blow to the end of the finger. Despite the dislocation, the DIP joint may retain some range of motion, so it is important not to overlook these injuries. [60] Lateral radiographs are diagnostic.

Management of the dorsal DIP dislocation involves reduction in a fashion similar to that described for other IP joint injuries. The injury is distracted by traction and hyperextension and relocated by pressure on the base of the distal phalanx during flexion. [57] Subsequent management involves application of a dorsal splint with 5° to 10° of flexion for 3 weeks. [57]

An injury to the DIP joint that may be confused with a dislocation is the mallet finger (Fig. 52-35) . The injury is often caused by blunt trauma to the end of the finger (e.g., struck by a baseball). The patient presents with an inability to extend the fingertip. The joint appears normal on passive extension by the examiner. The injury is a rupture of the extensor tendon, with or without avulsion of a small piece of bone. Unless the injury is properly splinted or surgically immobilized, a permanent deformity will occur (see

**Metacarpophalangeal Dislocations**

The pathology and management of finger MCP dislocations are identical to those of the thumb, as discussed above. The same classification of simple and complex applies; the complex type requires operative repair. Dimpling on the palmar surface suggests the presence of a complex dislocation. It is important to remember that the application of traction alone for a simple MCP dislocation may convert it to a complex dislocation. After the wrist is flexed to relax the tendons, the initial maneuver in reduction should be hyperextension as far as possible. This is followed by pressure on the base of the proximal phalanx to effect reduction. [57] After reduction of a simple MCP dislocation, buddy taping is generally sufficient to secure the reduction. [57]

**Carpometacarpal Dislocations**

Carpometacarpal dislocations are rare injuries that are frequently misdiagnosed. The usual site of injury is the fifth carpometacarpal joint, which is dorsally dislocated. [58] The injury is usually the result of a high-energy mechanism, such as a motor vehicle crash or a fall. The diagnosis can be quite difficult, as it may be obscured even on the lateral radiograph. Associated fractures and other injuries are frequently present, and percutaneous fixation is usually required.

# HIP DISLOCATIONS

The hip is generally a stable ball-and-socket joint. The head of the femur is deeply situated in the acetabulum, and ligamentous and muscular support is very strong. Hip dislocations are therefore usually the result of significant forces, and a careful search for other limb- or life-threatening injuries must be undertaken. Conversely, in cases of severe multiple trauma in which the evaluation and treatment of other injuries take priority, a dislocated hip may be overlooked unless specifically sought after immediate life-threatening injuries have been addressed. Common mechanisms of hip dislocation include motorcycle crashes, car crashes, and falls.

Associated fractures are quite common with hip dislocations. In fact, up to 88% of hip dislocations present with an associated fracture. [62] When a fracture complicates the dislocation, orthopedic consultation is generally indicated. The emergency physician should be able to reduce type I hip dislocations, which are dislocations without an associated fracture or with a very minor fracture.   Hip dislocations may occasionally be missed in the setting of severe trauma, as other injuries garner more attention. A missed diagnosis can also occur when a femur fracture obscures the clinical picture of hip dislocation. [63] Sciatic nerve injuries are seen in 10 to 14% of posterior hip dislocations. One of the more disabling complications is avascular necrosis of the femoral head. Although it is generally stated that early reduction will reduce the frequency of this complication, evidence for this statement is hard to find. Dreinhofer and coworkers noted poor outcomes despite early (i.e., <6 hours) reduction of type I hip dislocations. [62] Yang and colleagues found that reduction beyond 24 hours was associated with a

worse prognosis, but they could not find a significant time factor for those reduced in <24 hours. [61] However, it is still advisable to reduce hip dislocations as soon as feasible, to decrease soft tissue distortion. If evidence of nerve injury exists, the dislocation should be treated as an emergency and should be reduced as early as possible.

## Radiographic Examination

Dislocation of the hip is generally obvious on the standard AP film that is often taken during trauma resuscitations as a pelvic film. The use of a lateral or oblique view may help clarify the type of dislocation, but this can usually be deduced through clinical examination.

## Analgesia and Anesthesia

The dislocation of a prosthetic hip can usually be managed with moderate amounts of IV premedication in the ED. Premedication recommendations for acute traumatic dislocations run the gamut from general anesthesia for all reductions [62] to IV sedation only. [64] Some IV premedication is recommended, and patients often require deep sedation if the procedure is to be successful in the ED. One should not hesitate to push for spinal or general anesthesia if a reasonable attempt at reduction fails in the ED.

## Posterior Hip Dislocations

A posterior hip dislocation is usually the result of a force on the flexed knee with the hip in varying degrees of flexion. The greater the amount of flexion of the hip at the time of the injury, the less the chance of an associated fracture. [63] The humeral head is forced out of the acetabulum and rests behind it (Fig. 52-36 (Figure Not Available) *A* and *B*). The sciatic nerve is located just behind the hip joint and may be injured with posterior hip dislocation. The clinical picture includes a shortened, internally rotated, and adducted leg.

### Reduction Techniques

Two basic methods for hip reduction have been reported in the literature. In the prone or gravity method of Stimson (Fig. 52-37) , the patient is placed so that the distal pelvis overhangs the edge of the stretcher. The hip, knee, and ankle are all flexed to 90°, and downward pressure is applied to the proximal posterior tibia. [63] The hip can be gently internally and externally rotated to facilitate reduction, and direct downward pressure may be applied to the femoral head by an assistant. An alternative and more comfortable way to provide downward pressure on the tibia is for the operator to grasp the patient's ankle and place his or her own knee on the patient's calf, applying the body weight for pressure. [64]

Other techniques involve placing the patient in a supine position with downward stabilization of the pelvis performed by an assistant. In the Allis technique (Fig. 52-38) , upward traction is exerted in line with the deformity, and the hip is flexed to 90°. The hip

can be gently rotated internally and externally until it is reduced. [63] Howard suggests modifying this technique by applying lateral traction to the flexed upper femur to disengage the head of the femur from the outer lip of the acetabulum. [65]

Once reduction is achieved, the legs are immobilized in slight abduction through the placement of pillows or another object between the knees. Reduction is confirmed by repeat radiographs, and the patient will require admission to the hospital.

## Anterior Hip Dislocation

Anterior hip dislocation is a less common injury than posterior dislocation, constituting 10 to 15% of all hip dislocations. There are three general types of anterior hip dislocations, which are defined by where the femoral head comes to rest (Fig. 52-39) (Figure Not Available) : the iliac or subspinous, the pubic, and the inferior or obturator dislocation. Anterior hip dislocations generally result from a forced abduction of the thigh, which may occur in a fall or motor vehicle crash. [63] The clinical picture varies with the type of dislocation. With the obturator (inferior) type, the leg is abducted and externally rotated with varying degrees of flexion. In the other types, the hip is usually extended and externally rotated. [63]

### Reduction Techniques

The Stimson gravity method may work for anterior hip dislocation, although it is not recommended for the pubic type. [63] Alternatively, the Allis maneuver is applied in a modified fashion (Fig. 52-40) . The patient is placed in a supine position and an assistant stabilizes the pelvis and applies lateral countertraction to the thigh. Traction by the operator is applied in the long axis of the femur with the hip slightly flexed. The leg is then gently adducted and internally rotated to effect reduction. As with posterior dislocations, admission to the hospital is required for patients with these injuries.

# KNEE (FEMUR/TIBIA) DISLOCATIONS

Although the knee is a simple hinge joint, dislocations are quite rare due to its strong ligamentous support. The major ligaments include the anterior and posterior cruciate and the collateral ligaments. The usual mechanism of a knee dislocation involves a great deal of force, such as a motor vehicle crash or a sporting injury. However, knee dislocation has been reported after minor mechanisms, such as stepping off a curb or into a hole. [66] There are five general types of knee dislocations, including anterior, posterior, medial, lateral, and rotatory (Fig. 52-41) (Figure Not Available) . Knee dislocations are described with respect to the position of the tibia in relation to the femur. [67]

## Clinical Assessment

Knee dislocations are usually clinically obvious; however, they may have been reduced at the scene. A grossly unstable knee is probably a reduced dislocation and carries the same risk of vascular and other complications as a dislocated knee. [68] The severely

unstable knee can be defined as one that has >30° recurvatum (hyperextension) [68] or one that has medial joint opening when stressed in full extension. [67]

An impressive effusion may not be present in knee dislocation, as the joint capsule is often disrupted and extravasation occurs into the surrounding tissue. [67] The most important part of the clinical assessment is the vascular status of the extremity (see below). Nerve injury is less common, but peroneal nerve injury is a recognized complication, particularly of a posterolateral dislocation. [69] Posterolateral dislocations may be irreducible, as the medial femoral condyle buttonholes through the joint capsule. [70] A clue to this injury is the presence of a dimple sign at the medial joint line. [70]

**Vascular Injury**

The most feared complication of a knee dislocation is severance of the popliteal artery. Vascular injury is frequent in knee dislocations due to the popliteal artery being relatively fixed both proximally and distally. [67] This complication can be seen with both anterior and posterior dislocations. [69] Varnell and others pointed out that vascular injury was as common in the severely unstable knee as in a dislocated knee. [68] The incidence of popliteal artery injury in a dislocated knee is around 20% in most series. The seriousness of this complication is largely due to the fact that collateral circulation about the knee is poor, [66] and amputation may be the end result of popliteal artery (or vein) injury.

It has been previously stated that popliteal artery disruption can occur with a full pulse. [70] Such statements have led to recommendations to perform arteriography or exploration in all knee dislocations. [67] Recent studies question that perspective. Varnell and coworkers reported a pulse deficit or absence in all patients with vascular injury. [68] Kendall and colleagues also reported clear clinical evidence for all popliteal artery injuries in knee dislocations. [71] This group recommended exploration for obvious ischemia, angiography for those with ischemia who have pulse restoration after relocation, and observation for all others. [71]

If vascular compromise is detected on clinical assessment, it is appropriate to reduce the knee dislocation without obtaining radiographs. [67] [69] The use of the Doppler flow meter for pulse checks and ankle-brachial pressure indices should also be considered in these injuries (see Chapter 70) .

## Reduction Technique

Premedication is dependent on the clinical situation, but it should be considered when possible. The basic initial approach for all types of knee dislocations is to apply traction to the extremity (Fig. 52-42) (Figure Not Available) . This alone is often all that is required for reduction. [70] For anterior dislocations the distal femur is lifted to effect reduction. For posterior dislocations the proximal tibia is lifted to complete the reduction. [67] For medial and lateral dislocations a similar approach is acceptable, with pressure being exerted as needed in the medial or lateral direction.

After reduction the extremity is splinted in 15° of flexion. The posterolateral dislocation may be irreducible, and operative intervention should be considered if reduction is not easily accomplished.

**Postreduction Care**

Appropriate aftercare in knee dislocations involves serial reassessment of the neurovascular status of the extremity. Postreduction radiographs are performed and the patient is admitted to the hospital. These injuries cause severe ligamentous and other derangements in the knee and generally require operative stabilization.

# DISLOCATIONS OF THE FIBULAR HEAD

The fibula can be dislocated at its proximal articulation in the knee joint. This is most commonly an anterior dislocation. [67] The fibular head is normally nestled in a stable manner behind the lateral tibial condyle with 2 supporting tibiofibular ligaments. [72] The tibiofibular joint has a separate synovial cavity, and therefore a typical knee joint effusion will not be seen with this dislocation. When the knee is flexed the stability of this joint is decreased due to relaxation of the fibular collateral ligament. [72] The typical mechanism of injury is a fall on the flexed, adducted leg, often combined with ankle inversion. This mechanism is seen in sports and parachute landings. [72] Posterior dislocations can occur from a direct blow to the area while the knee is flexed, such as in horseback riding. [67]

Anterior dislocation is accompanied by obvious prominence of the fibular head anteriorly; no associated neurovascular problems are noted. The more uncommon posterior dislocation often is accompanied by an injury to the peroneal nerve. [67] Patients present with varying degrees of disability, and some may walk on the leg with only mild discomfort. [72] On radiographic examination, the 3 cardinal signs of anterior dislocation are lateral displacement of the fibula on the AP film, a widened proximal interosseous space, and anterior displacement of the fibular head on the lateral view. [72]

**Reduction Technique**

The position for reduction of an anterior fibular head dislocation is to place the patient supine and flex the knee to 90° in order to relax the biceps femoris tendon. The foot can also be inverted to create further distraction at the site. [67] Direct pressure is then applied to the fibular head and reduction is usually signified by a snap. [72] The method for a posterior dislocation is the same except that the direct pressure is applied in forward manner.

# PATELLAR DISLOCATION

Patellar dislocations are fairly common, especially among adolescents. The usual mechanism is a powerful quadriceps contraction combined with a strong valgus and

external rotation component. [73] This may be seen in activities such as making a "cut" in sports or with dancing. The patella may also dislocate from a direct blow to the flexed knee. [67] Predisposing factors to patellar dislocation include genu valgum and femoral anteversion. [73] The patellar dislocation is described by the relation of the patella to the knee joint. Lateral dislocations are the most common by far. Other types include superior, medial, and intra-articular.

## Clinical Assessment

Lateral dislocation of the patella is generally clinically obvious (Fig. 52-43) . The knee is held in some degree of flexion and the patella can be easily seen and palpated on the lateral side of the knee. A tenting type action of the patella is often detectable unless significant soft tissue swelling is present.

The patella may be spontaneously reduced in the field with leg straightening. The patient will report that the leg "went out" and may describe actually seeing the lateral deformity caused by the displaced patella. Clinical clues to the spontaneously reduced patella include the presence of a knee effusion and tenderness along the medial edge of the patella. Fairbank's test or the patellar "apprehension" sign is elicited when the patella is pushed laterally and the patient grabs for the knee, indicating the sensation of repeat injury. [67]

## Radiographs

Prereduction films are difficult to obtain as the patient is usually in flexion. Some recommend prereduction films in all patients; [67] however, it is an easy matter to reduce these injuries prior to radiography. The diagnosis is usually obvious and there are no reports in the literature of complications from gentle reduction. Osteochondral fractures are detectable in about half of patients with patellar dislocations, but many of these are only visible on arthroscopy. [73] Postreduction radiographs are recommended, as are prereduction studies, when the diagnosis is uncertain. The clinical diagnosis of patellar dislocation in an older patient should be made with caution, as these are primarily injuries of the young.

## Reduction Technique and Postreduction Care

Reduction of a lateral patellar dislocation is usually quite simple. Premedication is often not required if the patient can be verbally reassured. If the patient is anxious or in great discomfort, premedication should be considered. The 2 basic maneuvers for patellar relocation are extension of the knee and gentle medial pressure applied to the patella, lifting the most lateral edge of the patella over the femoral condyle (Fig. 52-44) (Figure Not Available) . [67]

The leg is then immobilized in extension. This may be done by casting or application of a commercially available knee immobilizer. Orthopedic follow-up is necessary because of the need for physical therapy and the high rate of persistent instability. [73]

Patellar dislocations in other locations are often irreducible, and orthopedic consultation should be sought in these circumstances. Hospitalization is not required for routine lateral dislocations of the patella.

## ANKLE DISLOCATIONS

The ankle joint is a modified hinge-type joint in which the talus is nestled in the mortise formed by the distal tibia and fibula. [74] The ligamentous support of the ankle is quite strong, and pure dislocations are uncommon. Usually there are associated fractures of the ankle joint (Fig. 52-45) (Figure Not Available) . Ankle dislocations

are described by the relation of the talus to the tibia. Posterior dislocations of the ankle are more common than area anterior dislocations, and they usually result from a fall on a plantar flexed foot. Patients with posterior dislocations often have an associated fracture of the posterior tibial margin (Fig. 52-46) (Figure Not Available) . [74] The clinical picture is usually one of significant deformity and disability.

Anterior dislocations generally result from forced dorsiflexion or a posterior blow to the distal tibia while the foot is fixed. The talus is prominent anteriorly and the dorsalis pedis pulse may be lost secondary to pressure from the talus. [74] Superior dislocations are uncommon and result in diastasis of the tibiofibular joint. These injuries are usually the result of a significant axial force. Lateral dislocations of the ankle are always fracture dislocations.

### Radiographic Examination

Because of the high rate of associated fractures and the clinical difficulty in assessing for the presence or the nature of a dislocation, it is recommended that prereduction radiographs be obtained in all suspected ankle dislocations. An AP and lateral view usually suffice for emergency management, and other views can be ordered if necessary after the joint is relocated.

### Analgesia

Unless a strong contraindication is present, it is advisable to administer IV premedication to patients with ankle dislocation early in their care, preferably before conducting any manipulations or radiologic studies. Reduction is always painful, and sufficient premedication must be administered.

### Reduction Techniques

For posterior dislocations, the patient is placed supine and the knee is flexed to relax the Achilles tendon. This can be done by an assistant, or the patient can be brought to where the knee hangs over the end of the bed. The operator then grasps the foot with both hands, placing one hand on the heel and the other on the forefoot (Fig. 52-47)

(Figure Not Available) . The foot is flexed slightly plantar, and traction is applied to the foot. A second assistant can then apply downward pressure on the distal tibia as the operator moves the heel anteriorly to effect reduction. [75]

For anterior dislocations, the initial steps and positioning are the same as for posterior dislocation (Fig. 52-48) (Figure Not Available) . However, instead of plantar flexion, the foot is dorsiflexed to free up the talus. The second assistant applies upward pressure to the distal tibia while the operator pushes the foot in a posterior direction. [74]

Lateral dislocations are really fracture-dislocations, and orthopedic consultation is generally required. The emergency physician will often need to reduce these injuries due to the extreme lateral deformity and the occasional compromise of the dorsalis pedis artery by stretch. Open dislocations (in the absence of vascular compromise) may be better handled by cleaning in the operating room prior to attempts at reduction. If a lateral fracture-dislocation is to be reduced in the ED, the approach is quite similar to that for posterior ankle dislocation. However, instead of pressure in the AP direction, the foot is moved medially after application of traction.

### Postreduction Care

The ankle is splinted at 90° with a long-leg posterior splint; necessity for admission to the hospital will need to be determined in consultation with orthopedic surgeons. Many patients with these injuries have associated fractures that necessitate surgical intervention.

## DISLOCATIONS OF THE FOOT

The importance of the foot is recognized by anyone who has had to spend time ambulating with an injury to this area. For the purposes of discussion, injuries to the foot can be divided into those of the hindfoot and those of the forefoot.

### Hindfoot Injuries

Injuries to this area are uncommon and usually require high-energy transfer. The major dislocations are the subtalar and talar dislocations, and midtarsal fracture-dislocations (Lisfranc's injury). Lisfranc's injury is complex and is always a fracture-dislocation because of the rigid nature of the region. These injuries require orthopedic management and are not discussed.

#### Subtalar Dislocation

This uncommon injury generally occurs secondary to sports, falls from heights, or motor vehicle crashes. The calcaneus, navicular, and forefoot are displaced from the talus. [75] The primary process is severe inversion causing a medial dislocation, or severe eversion resulting in a lateral dislocation. It occurs so commonly during basketball that it has been termed *basketball foot.* [76] This is an injury usually seen in young adult males. Medial dislocations constitute the majority (85%) of these injuries, with lateral

dislocations making up the rest. [76]

The diagnosis is usually obvious, as the talus is prominent and often tents the skin of the proximal foot. The medial type has been termed an "acquired clubfoot," whereas the lateral appears as an "acquired flatfoot." [76]

Some authors recommend spinal or general anesthesia for all such injuries; however, it is commonly possible to reduce these injuries with IV premedication. The patient is positioned in a supine fashion and the knee is flexed much as in posterior dislocation of the ankle. One hand is placed on the forefoot and the other grasps the heel. Firm longitudinal traction is required to effect reduction, and dangling the leg over the end of the bed allows the operator to use his or her body weight to assist in traction. Once traction is applied, the deformity is initially increased (inversion for medial; eversion for lateral) and then reversed to effect reduction. [76]

### Dislocation of the Talus

In this extremely rare injury, the talus is essentially extruded from its normal position, coming to lie anteriorly. This injury is not amenable to closed reduction and virtually always progresses to avascular necrosis. [77] Talectomy is generally required, and orthopedic referral should be emergently undertaken if vascular compromise of the foot is present.

## Forefoot Dislocations

Much of what is pertinent to the diagnosis and management of forefoot dislocations has already been discussed in the management of dislocations of the fingers and hand MCP joints. Anatomically, the joints are quite similar.

### Metatarsophalangeal (MTP) Dislocations

These uncommon injuries are generally the result of hyperextension resulting in a dorsal dislocation of the MTP joint. [78] As with MCP dislocations, these can be simple or complex. Complex dislocations of the first toe can be suspected by the presence of sesamoid bones in the joint space in the prereduction radiographs. [76] Complex MTP dislocations are irreducible.

For simple MTP dislocations, reduction is accomplished by increasing the deformity through hyperextension and then applying traction while applying thumb pressure over the base of the dislocated proximal phalanx. Plantar flexion of the foot may be used to relax the flexor tendons. [75] Operative intervention is required after reduction if crepitus is present on motion, the joint is unstable, or an intra-articular loose body is noted on the postreduction radiographs. [76]

### Interphalangeal Dislocations

In the foot, IP dislocations result from an axial load to the toe, such as from kicking a

wall. These dislocations are generally dorsal and can be reduced as in the hand. Dislocations of the first toe interphalangeal joint are usually buddy taped to the second toe for 2 to 3 weeks, whereas those of lesser toes can be taped for 10 to 14 days.

## CONCLUSION

The following points are important regarding the assessment and management of dislocations:

1. A search for other more serious injuries should be undertaken when there is a high-energy mechanism of injury.
2. A neurovascular assessment should be performed early in the evaluation and appropriately documented.
3. Radiographs and IV premedication are generally indicated prior to reduction attempts.
4. Reduction attempts should involve the gentle, gradual application of forces and patience on the operator's part.
5. After completion of reduction, the operator should recheck the neurovascular status, request postreduction radiographs, and, in certain circumstances, assess the stability and the range of motion of the joint.

A definite percentage of dislocations are irreducible, and the need for multiple attempts should prompt orthopedic consultation.

# Chapter 53 - Splinting Techniques

**Carl R. Chudnofsky**

Splints are frequently used in the emergency department (ED) for temporary immobilization of fractures and dislocations and for definitive therapy of soft tissue injuries. Immobilization is the mainstay of fracture therapy, but it is difficult to find firm scientific data that support the use of splinting for soft tissue injuries. Although the general principle of immobilizing sprains and contusions is strongly supported by custom and personal preference, its exact influence on healing, number of complications, and ultimate return to normal activity is not known. In most studies of ankle sprains, for example, the function and pain of the injured joint are similar at 6 weeks' follow-up regardless of whether treatment consisted of ad lib walking, a simple elastic bandage, a posterior splint, or a formal cast.Patients with acute gouty arthritis of the wrist acknowledge reduced pain with short-term use of a short arm splint while medical therapy is initiated.

Most splinting techniques are handed down from housestaff or experienced physicians, but the procedure is often suboptimal and haphazard. [7] This chapter presents guidelines for the adequate immobilization of injuries that are commonly encountered by emergency physicians.

Patients routinely present to the ED with injuries that are amenable to splinting to relieve pain and to augment

healing (Table 53-1) . Emergency physicians have virtually abandoned the use of circumferential casts in favor of premade commercial immobilizing devices or splints made from plaster of Paris or fiberglass. The impetus for this change is primarily related to the complications occasionally associated with circumferential casts, liability issues, and ease of application brought about by new technology. In most instances, properly applied splints provide short-term immobilization equal to that of casts while allowing for continued swelling, thus reducing the risk of ischemic injury. Other obvious advantages of splints are that patients can take them off when immobilization is no longer needed or can remove them temporarily to bathe, exercise the injured part, or perform wound care.

## INDICATIONS

Theoretically, immobilization facilitates the healing process by decreasing pain and protecting the extremity from further injury. Other benefits of splinting are specific to the particular injury or the problem that is being treated. For example, in the treatment of fractures, splinting helps maintain bony alignment. Splinting deep lacerations that cross joints reduces tension on the wound and helps prevent wound dehiscence. Immobilizing tendon lacerations may facilitate the healing process by relieving stress on the repaired tendon. The discomfort of inflammatory disorders such as tenosynovitis or acute gout is greatly reduced by immobilization. Deep space infections of the hands or feet as well as cellulitis over any joint should similarly be immobilized for comfort. Limiting early motion also may reduce edema and theoretically improve the immune system's ability to

combat infection. Hence, selected puncture wounds and mammalian bites of the hands and feet may be immobilized until the risk of infection has passed. Splinting large abrasions that cross joint surfaces prevents movement of the injured extremity and reduces the pain that is produced when the injured skin is stretched. Finally, patients with multiple trauma should have fractures and reduced dislocations adequately splinted while other diagnostic and therapeutic procedures (e.g., peritoneal lavage, computed tomography scan) are completed. Immobilization decreases blood loss, minimizes the potential for further neurovascular injury, decreases the need for opioid analgesia, and may decrease the risk of fat emboli from long bone fractures.

| **TABLE 53-1** -- Conditions That Benefit from Immobilization |
| --- |
| Acute arthritis |
| Severe contusions and abrasions |
| Skin lacerations that cross joints |
| Tendon lacerations |
| Tenosynovitis |
| Puncture wounds to the hands, feet, and joints |
| Animal bites to the hands or feet |
| Deep space infections of the hands and feet |
| Joint infections |

# EQUIPMENT

## Support Materials

### Plaster of Paris

Plaster of Paris is the most widely used material for ED splinting. [8] Its name originated from the fact that it was first prepared from the gypsum of Paris, France. When gypsum is heated to approximately 128 °C, most of the water of crystallization is driven off, leaving behind a fine white powder--plaster of Paris. When water is added to plaster, the reaction is reversed, and the plaster recrystallizes or sets by incorporating water molecules into the crystalline lattice of the calcium sulfate dihydrate molecules.

Today, plaster is impregnated into strips or rolls (2-, 3-, 4-, or 6-in. widths) of a crinoline-type material. The crinoline allows for easy application, helps keep the plaster molded to the proper form during the setting process and adds support to the finished splint. Plaster rolls and sheets are available in a variety of setting times and widths. The distinct advantage of plaster over commercially available, premade splints is that plaster can be more easily molded and tailored to the individual's anatomy, negating the "one-size-fits-all" approach. Also, plaster is generally less expensive than premade splints.

### Prefabricated Splint Rolls

The use of plaster splints in the form of prefabricated splint rolls (OCL, Specialist J-Splint) are popular among emergency physicians. These splint rolls have 10 to 20 sheets of plaster enclosed between a thick layer of protective foam padding on one side, and a thin layer of cloth on the other side. Like custom constructed splints, they are secured to the extremity with an elastic bandage. The major advantage of prefabricated splint rolls is that significant time is saved because the splint and padding are ready to apply. In addition, prefabricated splint rolls are ideal for intermittent splinting and can be removed and reapplied by the patient as needed. However, prefabricated plaster splint rolls are more expensive than simple plaster rolls, and they lack some of the versatility and custom-fit qualities of self-made plaster splints. The application of premade splints is shown in Figure 53-1 (Figure Not Available) *A- F.*

Recently, prefabricated splint rolls using layers of fiberglass between polypropylene padding (Ortho-Glass) have been introduced. These splint rolls offer the same time-saving aspect of prefabricated plaster splint rolls. In addition, splints made from prefabricated fiberglass rolls cure more rapidly (20 minutes), have no messy residue (i.e., can be hydrated using a conventional sink without a special trap), can be washed and reapplied, and are stronger and lighter than splints constructed from prefabricated

plaster rolls. Another advantage is the polypropylene padding, which wicks moisture away from the skin better than polyester, nylon, or cotton padding. [9] However, like prefabricated plaster splint rolls, prefabricated fiberglass splints lack some of the versatility and custom-fit qualities of self-made plaster splints.

## Protective and Miscellaneous Equipment

### Stockinette

A single layer of stockinette is commonly used under circumferential casts and splints. It protects the skin and, when folded back over the ends of the plaster, creates a smooth, professional-looking, padded rim. Stockinette is available in 2-, 3-, 4-, 8-, 10-, and 12-in. widths.

### Padding

Padding under the splint protects the skin and bony prominences and allows for swelling of the injured extremity. In general, the older thin cotton padding known as sheet wadding has been replaced by newer materials such as Webril (Curity) or Specialist (Johnson & Johnson) cast padding. Webril is soft cotton with a much coarser weave than sheet wadding; consequently it has greater tensile strength, adheres better, and can be applied more evenly. Specialist padding uses micropleated cotton fibers that relax when moistened. This results in uniform, felt-like padding that conforms to the surface being wrapped. Felt (0.5-in. thick) also may be used to pad bony prominences.

### Elastic Bandages

Elastic bandages are used to secure the splint in place. Elastic bandages are available in 2-, 3-, 4-, and 6-in. widths.

### Adhesive Tape

Adhesive tape is used to prevent slippage of the elastic bandages, to line the cut edges of a bivalved cast, and to "buddy tape" digits.

### Utility Knife and Plaster Scissors

A utility knife, a No. 10 scalpel blade, or plaster scissors can be used to cut and shape dry plaster.

### Bucket

A large bucket (preferably stainless steel) is used for wetting plaster. Plaster should not be prepared in the sink because the residue quickly clogs the drain. A special drain is required to accept plaster residue.

**Protective Gear**

Gowns or sheets prevent soilage of the both the patient's and the physician's clothing. Gloves (vinyl or latex) and safety glasses are recommended to prevent skin or eye damage from plaster dust, wet plaster, or uncured fiberglass polymer. Wearing gloves also decreases cleanup time for the physician.

## GENERAL PROCEDURE OF CUSTOM SPLINT APPLICATION

The following section refers to the application of custom-made plaster splints (Fig. 53-2 *A- E*), unless otherwise stated. If periodic wound care is required, a more easily removable splint (e.g., an OCL, Ortho-Glass, or a Velcro-type splint) should be applied in lieu of the standard splint to be described. The issue of removability should be addressed before the splint is applied. In addition, use of Webril (Curity) cast padding is described, but other suitable cast padding may be substituted.

### Patient Preparation

If the clinical situation permits, the patient should be covered with a sheet or gown to protect the clothing and the surrounding area from water and plaster. Nursing and housekeeping staff also appreciate this courtesy. The involved extremity should be inspected carefully before splinting. All skin lesions and soft tissue injuries should be examined and documented clearly on the emergency department record. All wounds should be cleaned, repaired, and dressed in the usual manner. When immobilizing open fractures or joints, the soft tissue defect should be covered with saline-moistened sterile gauze.

### Padding

When the splint involves the digits, padding must be placed between the fingers and toes to prevent maceration of the skin. This can be done with pieces of Webril or gauze cut to the appropriate length.

Following placement of padding between the fingers and toes, stockinette is often used as the next protective layer in self-made splints (see Fig. 53-2 *A*). The stockinette should extend at least 10 to 15 cm beyond the area to be splinted at both ends of the extremity. Later, after plaster has been applied, the stockinette can be folded back over the ends of the splint to create smooth, padded rims. Folding back the stockinette can also help hold the splint in place when applying elastic bandages (see Fig. 53-2 *C*). Care is needed to avoid pressure damage from pulling the stockinette too tightly over bony prominences, such as the heel. Wrinkling over flexion creases should also be avoided by slitting and overlapping the stockinette at bony prominences. One may also use two separate pieces of stockinette (one at each end of the splint) to produce the smooth padded rims. As a general rule, 3-inch-wide stockinette is used for the upper extremity, whereas 4-inch-wide is used for the lower extremity.

After the stockinette has been properly positioned, Webril should be wrapped around the entire area that will be exposed to plaster. The Webril should be at least 2 to 3 layers thick and each turn should overlap the previous turn by 25 to 50% of its width (see Fig. 53-2 *B*). In addition, the Webril should extend 2.5 to 5.0 cm beyond the ends of the splint so it too can be folded back over the splint to help create smooth, well-padded edges (see Fig. 53-2 *C*). Extra padding should be placed over areas of bony prominence, such as the radial condyle or the malleoli (Table 53-2) . If significant swelling is anticipated, 3 to 4 layers of Webril should be applied as padding. Care should be taken to avoid wrinkling, because it can result in significant skin pressure when a tight splint is used for a long period. Wrinkles can be eliminated by proportionately stretching or even tearing the side of the Webril that must wrap around the bigger portion of an extremity. Joints that must be immobilized in a 90° position, such as the ankle, make continuous Webril wrapping difficult. To avoid wrinkles in the area of the ankle, the joint should be placed in the proper position before padding. Webril is then wrapped around the malleolar and midtarsal regions first. The bare calcaneal region can then be covered with overlapping vertical and horizontal Webril strips until the entire heel region is evenly padded. The same approach can be used in similar areas, such as the elbow. The width of Webril that should be used varies depending on the extremity to be splinted. In general, the 2-in. width should be used for hands and feet, the 3- to 4-in. width for the upper extremity, and the 4- to 6-in. width for the lower extremity.

A final caveat when using Webril is to be aware of the potential for ischemic injury. This rare complication is most likely to occur in an extremity that continues to have significant swelling after the patient is released from the emergency department. Ischemia may result because the concentrically placed Webril can become a constricting band. If this situation is anticipated, it can be prevented easily by cutting through the Webril along the side of the extremity opposite to the plaster splint. The splint is then secured to the extremity in the usual manner. Alternatively, 2 to 3 layers of Webril (the same diameter as the plaster) are placed directly over the wet plaster (Fig. 53-3) . The Webril-lined splint is then positioned over the area to be immobilized and secured with an elastic bandage.

**Plaster Preparation**

The choice of plaster setting time depends on the nature of the injury and the expertise of the physician. Extra-fast-setting plaster is typically used when rapid hardening is desired to help maintain alignment of an acutely reduced fracture. However, for the majority of ED splints, plaster with slower setting times (e.g., Specialist fast-drying) is recommended. [10] Plaster that sets more slowly is easier for some physicians to use, because the longer setting times allow more leeway in applying and molding the splint. Furthermore, plaster with a longer setting time produces less heat, thus reducing both patient discomfort and the risk of serious

| TABLE 53-2 -- Bony Prominences of the Upper and Lower Extremity That Require Additional Padding |
|---|
| **Upper Extremity** |
| Olecranon |
| Radial styloid |
| Ulnar styloid |
| **Lower Extremity** |
| Upper portion of the inner thigh |
| Patella |
| Fibular head |
| Achilles tendon |
| Medial and lateral malleoli |

burns. [11] Table 53-3 lists the setting times for commonly used plaster. These setting times are created by adding different substances to the plaster during the production process (Table 53-4) . Given plaster with equal setting times, the most important variable affecting the rate of crystallization is water temperature. *Warm water hardens a splint faster than cold water* and should be avoided when extra time is desired for splint

application.

The ideal length and width of plaster depends on the body part to be splinted and the amount of immobilization required. The best way to estimate length is to lay the dry splint next to the area to be splinted. It is best to use a generous length, because wet plaster shrinks slightly from its dry length. Also, if the wet splint is too long, the ends can be folded back easily. The plaster width varies according to the type of splint being made and the body part that is injured, but generally it should be slightly greater than the diameter of the limb to be splinted. Specific recommendations regarding splint length and width are made in sections describing individual splints.

The thickness of a splint depends on the size of the patient, the extremity that is injured, and the desired strength of the final product. An ankle splint may crack quickly and become useless if only 8 layers are used, but this thickness may be ideal for a wrist splint. In general, it is best to use the minimum number of layers necessary to achieve adequate strength. Thicker splints are heavier and more uncomfortable. It is important to note that plaster thickness is a major determinant of the amount of heat given off during the setting process. With more than 12 sheets of plaster, there is an increased risk of significant burns, especially when using extra-fast-drying plaster, using dipping water with a temperature

| **TABLE 53-3** -- Setting Times of Fast- and Extra-Fast-Drying Plaster | |
| --- | --- |
| **Plaster** | **Setting Time (min)** |
| Fast-drying | 5-8 |
| Extra-fast-drying | 2-4 |

greater than 24 °C, or placing a pillow under or around the extremity for support during the setting process (Table 53-5) . In an average-sized adult, upper extremities should be splinted with 8 sheets of plaster, whereas lower extremity injuries generally require 12 to 15 sheets. This layering usually gives the strength necessary for adequate immobilization while reducing the patient's discomfort and the risk of significant burns. In a 300-lb patient, however, up to 20 layers may be required to make a durable ankle splint.

The dipping water should be kept clean and fresh. The use of water that has been used

previously for wetting plaster increases the amount of heat given off during crystallization and causes plaster to set more quickly. As a rule of thumb, the temperature of the water should be kept around 24 °C. This temperature allows for a workable setting time and has not been associated with an increased risk of significant burns. As the temperature of the dipping water approaches 40 °C, the potential for serious burns increases, even at splint thicknesses of less than 12 plies. It is interesting to note that water temperature has been shown to be only a minor consideration in heat production in some studies (see Table 53-5) .

## Splint Application

The dry splint should be completely submerged in the water until bubbling stops. The splint is removed and excess water is gently squeezed out until the plaster has a wet and sloppy consistency. The splint is placed on a hard table or countertop (a protective covering is recommended to prevent water or plaster damage) and smoothed out to remove any wrinkles and to ensure uniform lamination of all layers. Lamination helps to increase the final strength of the splint. The splint is placed over the Webril and gently smoothed over the extremity. Plaster is usually somewhat adherent to Webril, but an assistant may be required to hold the splint in place. Once the splint has been properly positioned over

**TABLE 53-4** -- Effect of Water Temperature and Different Additives on Setting Time of Plaster

| Accelerates Setting Time | Slows Setting Time |
|---|---|
| Reusing dip water | Cool dip water |
| Higher dip water temperature | Glue |
| Salicylic acid | Gum |
| Zinc | Borax |

| | |
|---|---|
| Magnesium | |
| Copper | |
| Iron | |
| Aluminum | |
| Salt | |
| Alum | |

| TABLE 53-5 -- Variables That Increase Heat Production During Crystallization | |
|---|---|
| **Major** | **Minor** |
| Increased splint thickness | High humidity |
| Setting time * | High ambient temperature |
| High dip water temperature | Reusing dip water |

| | |
|---|---|
| Wrapping the extremity for support while drying | |

* Faster setting times produce more heat.
Dip water temperature has been a minor determinant of heat production in some studies.

the extremity, folding back the underlying stockinette and Webril also helps hold it in place. The splint is secured with an appropriately sized elastic bandage by wrapping in a distal to proximal direction. Finally, the extremity is placed in the desired position and the wet plaster is molded to the contour of the extremity using only the palms of the hand. Finger indentations may cause a ridge that will produce a pressure point.

Molding the wet splint to conform to the body's anatomy is probably the most important yet the most frequently overlooked step to ensure adequate immobilization (see Fig. 53-2 *E*). The act of molding may cause some pain, and the patient should be forewarned. All manipulation of the wet plaster should be completed before it reaches a thick, creamy consistency. Any movement after this time, also known as the critical period, results in an imperfect crystalline network of calcium sulfate molecules and greatly weakens the ultimate strength of the splint. *While the plaster is setting*, a pillow or blanket should *NOT* be wrapped around the extremity for support (Fig. 53-4) . This leads to inadequate ventilation around the splint and greatly increases the amount of heat produced (see Table 53-5) .

If an elastic bandage is applied directly over wet plaster, the elastic bandage may be incorporated into the drying plaster, making subsequent removal of the bandage difficult. To make it easier for patients to remove and reapply the splint, a single layer of Webril or roll gauze can be wrapped around the wet plaster loosely before application of the elastic bandage. This prevents the wet plaster from becoming stuck to the elastic bandage. Only one layer of Webril should be used over the plaster because multiple layers have been associated with high drying temperatures.

Before the patient is released from the ED, the splint should be checked for adequate immobilization, and the patient should be observed for any evidence of vascular compromise or significant discomfort. If either occurs, the elastic bandage should be loosened. If the discomfort persists, additional padding should be placed over the painful areas. If this measure, too, is unsuccessful, a new splint should be made, and special attention should be paid to proper molding so that the wet plaster does not become indented. By resting tender tissue, splinting usually relieves discomfort quickly, and patients generally say that they feel better immediately after the splint has been applied. Never release a patient who complains of increased pain after a splint has been placed.

After a proper-fitting, comfortable splint has been applied, one may place 2 strips of tape along each side of the splint to prevent the elastic bandage from slipping. Tape should be applied over the metal fasteners used to secure the elastic bandages. Note that

these objects can be easily swallowed or aspirated by infants and small children. Finally, a sling should be provided for upper extremity injuries, and, if required, crutches should be dispensed (and instructions given for their proper use) for lower extremity injuries.

## Patient Instructions

Patients should receive verbal and written instructions on splint care and precautions. The importance of elevation in helping to decrease pain and swelling should be stressed and demonstrated (most patients do not understand the medical definition of elevation). *At night*, a pillow wrapped and secured around a hand or foot will help the patient keep the injured extremity satisfactorily elevated (see Fig. 53-4) . If the injury is less than 24 hours old, the application of ice bags or cold packs also should be encouraged. It is useless to apply cold packs over plaster, but it can be beneficial if it is applied over Webril and an elastic bandage or directly over an injury if the splint is removed. In theory, cold therapy stiffens collagen and thus reduces the tendency for ligaments and tendons to deform. Cold therapy also decreases muscle spasm and excitability, decreases blood flow (thus limiting hemorrhage and edema), increases the pain

threshold, and decreases inflammation. Because the thermal conductivity of subcutaneous tissue is poor, cold packs should be applied for at least 30 minutes at a time. This guideline is in contrast to the popular recommendation of "ice 20 minutes on, 20 minutes off," which does nothing more than cool the skin. Cold packs should not be applied for more than the first 24 to 48 hours because cold can interfere with long-term healing. The patient should be instructed not to stress the splint for at least 24 hours because plaster does not approach optimal strength until evaporation has reduced the water content of the plaster to approximately 21% of its initial hydrated level. This process of removing excess water by evaporation is called *curing,* and it generally takes several days to be completed. However, by 24 hours the water content of the plaster has usually been reduced enough to produce a strong resilient splint. In addition, because the chemical process involved in the formation of plaster is reversible, the patient should avoid getting the splint wet. However, if the injury permits, the splint can be removed for showering and then reapplied. Alternatively one or more plastic bags may be placed over a splint before showering.

Splints may crack, break, or disintegrate with wear, and a useless splint should be removed or replaced. Patients should be given general guidelines for length of immobilization and appropriate follow-up care. Long-term immobilization, particularly in the elderly, can produce permanent disability.

It is extremely important for the patient to continue to check for signs of vascular compromise. If the patient experiences a significant increase in pain, any numbness or tingling of the digits, pallor of the distal extremity, decreased capillary refill, or weakness, he or she should be instructed to return to the ED or to see the primary physician without delay. As with casting, increased pain after splinting is a warning sign that should prompt a return visit-- *not telephone advice*. Strong opioids should be avoided during the first 2 to 3 days after splinting to allow pain to prompt a follow-up visit.

# UPPER EXTREMITY SPLINTS

## Long Arm Splints

### Long Arm Posterior Splint

**Indications.**

The long arm posterior splint (Fig. 53-5 *A* and *B*) is used to immobilize injuries of the elbow and proximal forearm. It completely eliminates flexion and extension of the elbow, but it does not entirely prevent pronation and supination of the forearm. Therefore, it is not recommended for immobilization of complex or unstable distal forearm fractures unless used in conjunction with a long arm anterior splint. Alternatively, a double "sugar-tong" splint can be applied.

**Construction.**

The long arm posterior splint is constructed using 8 to 10 layers of 4- to 6-in. wide plaster. The splint starts on the posterior aspect of the proximal arm. It runs down the arm to the elbow and then continues along the ulnar aspect of the forearm and hand to the level of the metacarpophalangeal joints. The anterior splint is constructed in the same manner. It mirrors the posterior splint by running down the anterior aspect of the arm to the antecubital fossa, where it continues along the radial aspect of the forearm to the distal radius. The anterior splint is never used alone, but rather is used as an adjunct to the long arm posterior splint to improve immobilization by increasing stability and preventing pronation and supination of the forearm.

**Application.**

Stockinette and Webril are applied as described previously. A hole should be cut in the stockinette to expose the thumb, and extra padding should be placed over the olecranon to prevent pressure injury. The arm is positioned with the elbow flexed to 90°, the forearm neutral (thumb upward), and the wrist neutral or extended slightly (10° to 20°). It is helpful to have an assistant hold the wet splint in place, particularly when applying both a posterior and anterior splint. Once the splint has been properly positioned, the ends of the stockinette and Webril are folded back and the splint is secured in place using two 3- to 4-in. elastic bandages. Finally, the sides of the splint are folded up to create a gutter configuration, and the splint is carefully molded using the palms of the hand. The fingers and thumb should remain free to prevent stiffness from unnecessary immobilization.

### Double Sugar-Tong Splint

**Indications.**

Like the long arm posterior splint, the double sugar-tong splint (Fig. 53-6 is used to immobilize injuries of the elbow and forearm. However, because it prevents pronation and supination of the forearm, it is preferable for some fractures of the distal forearm and elbow.

**Construction.**

The splint consists of two separate pieces of plaster, a forearm splint and an arm splint. Each piece is constructed using 8 layers of 3- to 4-in. plaster. The forearm portion of the splint runs from the metacarpal heads on the dorsum of the hand, along the dorsal surface forearm around the elbow. It continues along the volar surface of the forearm to the palm of the hand, stopping at the level of the metacarpophalangeal joints. The arm portion of the splint begins on the anterior aspect of the proximal humerus. It runs down the arm over the forearm splint and around the elbow. It then continues up the posterior aspect of the arm, once again going over the forearm splint, until it reaches the starting point.

**Application.**

Use of stockinette and Webril and positioning are similar to those described for application of a long arm posterior splint. The two splints are secured in place using two 3- to 4-in. elastic bandages starting at the forearm splint at the hand. Once secure, the arm portion of the splint is wrapped beginning at the proximal end. The fingers and thumb should remain free to avoid stiffness.

## Forearm and Hand Splints

### Volar Splint

**Indications.**

The volar splint (Fig. 53-7 *A* and *B*) is used to immobilize a variety of soft tissue injuries of the hand and wrist. It is also used for temporary immobilization of triquetral fractures, lunate and perilunate dislocations, and second through fifth metacarpal head fractures. For these more serious injuries, some physicians prefer to add a dorsal splint to create a more stable bivalve effect (see Fig. 53-7 *B*). Because the volar splint does not completely eliminate pronation and supination of the forearm, it is not recommended for fractures of the distal radius and ulna.

**Construction.**

The splint is constructed using 8 to 10 layers of 3- to 4-in. wide plaster. The splint begins in the palm at the metacarpal heads and extends along the volar surface of the forearm to just proximal to the elbow. If there is an injury to any of the fingers, the splint

may be extended to incorporate the involved digit.

**Application.**

Stockinette and Webril should be applied as described previously. A hole should be cut in the stockinette to expose the thumb. In addition, Webril or gauze should also be placed between any digits that are going to be immobilized. The forearm is placed in the neutral position (thumb upward) with the wrist extended slightly (10° to 20°). Wrist flexion should be avoided. After the wet plaster has been properly positioned, the ends of the stockinette and Webril are folded back and a 3- or 4-in. elastic bandage is used to hold the splint in place. The sides of the splint are folded up, creating a gutter effect, and the plaster is carefully molded to conform to the contours of the palm and wrist. Some clinicians prefer to extend the splint to the fingertips and then fold the wet plaster back toward the palm, allowing the fingers to "grasp" the rounded distal end when at rest. In any event, the thumb and fingers should be free to move unless they are injured and are being intentionally immobilized by the splint.

## Sugar-Tong Splint

**Indications.**

The sugar-tong splint (Fig. 53-8 is used for fractures of the distal radius and ulna. The advantage of this splint over the volar splint is prevention of pronation and supination of the forearm. In addition, it immobilizes the elbow, which is desirable for the first few days following a distal forearm fracture.

**Construction and application.**

The splint is constructed and applied the same way as the forearm portion of the double sugar-tong splint, described above.

## Thumb Spica Splint

**Indications.**

The thumb spica splint (Fig. 53-9 is used to immobilize injuries to the scaphoid, lunate, and thumb and fractures of the first metacarpal. It is also used in the treatment of de Quervain tenosynovitis.

**Construction.**

The splint is constructed using 8 layers of 3-in. wide plaster. The splint extends from just distal to the interphalangeal joint of the thumb to the mid-forearm.

The forearm is placed in the neutral position with the wrist extended 25° and the thumb in the wine glass position (Fig. 53-10) . Stockinette and Webril are applied from the base of the palm to the mid-forearm. It may be difficult to place stockinette around the thumb. Instead, a hole can be cut in the stockinette to expose the thumb. The thumb is then padded with small vertical strips of Webril, or wrapped with 2-in. Webril. The dry plaster is then placed over the radial aspect of the forearm from just beyond the thumb interphalangeal joint to the mid-forearm. Once in position, the location of the first metacarpophalangeal joint is marked and a small (1 to 2 cm) perpendicular cut is made 1 cm distal to the mark on each edge of the plaster (see Fig. 53-9 inset). This will allow the splint to be molded around the thumb without creating a buckle in the plaster. The plaster is then dipped and secured in place with a 2- or 3-in. elastic bandage. It is important to carefully mold the wet plaster around the thumb and palm and to maintain the thumb in the wine glass position while the plaster is drying.

## Ulnar Gutter Splint

**Indications.**

The ulnar gutter splint (Fig. 53-11) is used to immobilize fractures and serious soft tissue injuries of the little and ring fingers and fractures of the neck, shaft, and base of the fourth and fifth metacarpals.

**Construction.**

The splint is made using 6 to 8 layers of 3- to 4-in. plaster. It incorporates both the little and ring fingers. It runs along the ulnar aspect of the forearm from just beyond the distal interphalangeal joint of the little finger to the mid-forearm.

**Application.**

Stockinette and Webril are applied as usual. Additional Webril or gauze should be placed between the little and ring fingers to prevent maceration of the skin. The forearm is in the neutral position with the wrist in slight extension (10° to 20°), the metacarpophalangeal joints in 50° of flexion, and the proximal and distal interphalangeal joints in slight (10° to 15°) flexion. When immobilizing a metacarpal neck fracture (i.e., boxer's fracture), the metacarpophalangeal joint should be flexed to 90°. Once in the proper position, the sides of the splint are folded up to form a gutter. The ends of the stockinette and Webril are then folded back to help hold the splint while it is secured in place with a 2- or 3-in. elastic bandage.

## Radial Gutter Splint

The radial gutter splint (Fig. 53-12 is used to immobilize fractures and serious soft tissue injuries of the index and long fingers and fractures of the neck, shaft, and base of the second and third metacarpals.

**Construction.**

The splint is made using 6 to 8 layers of 3- to 4-in. plaster. It runs along the radial aspect of the forearm from just beyond the distal interphalangeal joint of the index finger to the mid-forearm.

**Application.**

Stockinette (with a hole cut to expose the thumb) and Webril are applied as previously described. Additional Webril or gauze should be placed between the index and long fingers to prevent maceration of the skin. The forearm is in the neutral position with the wrist in slight extension (10° to 20°), the metacarpophalangeal joints in 50° of flexion, and the proximal and distal interphalangeal joints in slight (10° to 15°) flexion. When immobilizing a metacarpal neck fracture the metacarpophalangeal joint should be flexed to 90°. The dry plaster is placed over the extremity and the location of the thumb is marked. A hole is cut in the dry plaster to expose the thumb. The plaster is then dipped and positioned over the extremity. The ends of the stockinette and Webril are folded back to help hold the splint while it is secured in place with a 2- or 3-in. elastic bandage.

## Finger Splints

Fingers are splinted following sprains, fractures, tendon repair, or infection. Minor finger sprains can often be managed with dynamic splinting (e.g., buddy taping) (Fig. 53-13) or a commercially available foam splint with aluminum backing (one-surface splint) (Fig. 53-14) , but fractures, tendon repairs, and some soft tissue injuries benefit from formal splinting (e.g., thumb spica, ulnar and radial gutter splints). Specific conditions, such as mallet finger, require a specialized splint (Fig. 53-15) . When complete immobilization of a finger is required (e.g., unstable phalangeal fractures) an "outrigger" finger splint that incorporates the wrist may be used (Fig. 53-16) .

## Sling, Swathe and Sling, Shoulder Immobilizer

### Sling

The sling is used to maintain elevation and provide immobilization of the hand, forearm, and elbow. It is most often used in conjunction with a plaster splint or cast. There are a number of commercially available slings to choose from. Many of these are fairly economical and simple to use, whereas others are expensive and do not allow the versatility of a simple, inexpensive triangular muslin bandage. When applying a sling, it

is important to have adequate support of the wrist and hand (Fig. 53-17) . A sling that is too short will allow the wrist and hand to hang down (ulnar deviation) and can result in ulnar nerve injury.

### Swathe and Sling, Shoulder Immobilizer

The swathe and sling is the treatment of choice for most proximal humerus fractures and shoulder injuries, such as reduced dislocations. The sling supports the weight of the arm, and the swathe immobilizes the arm against the chest wall to minimize shoulder motion. In most EDs, the swathe and sling has been replaced by the commercially available shoulder immobilizer (Fig. 53-18) . Its advantage is that it may be removed for showering and range-of-motion exercises and it is easily reapplied by the patient (a desirable option in the care of a shoulder dislocation). If used for more than a few days, the axilla should be padded to absorb moisture and decrease skin chafing.

The Velpeau bandage is a sling-and-swathe device that positions the forearm diagonally rather than horizontally across the chest, with the hand elevated to the level of the shoulder. This offers no particular advantage over a standard sling and swathe, is difficult to apply, cannot be removed easily, and is not well tolerated for prolonged immobilization.

### Figure-of-8 Clavicle Strap

Clavicle fractures have been traditionally treated with an uncomfortable and complex figure-of-8 bandage. Despite its widespread use, this device has never been proved superior to a simple sling (in terms of cosmesis, functional outcome, or pain relief). [12] [13] Indeed, use of the figure-of-8 dressing should be discouraged because it may actually promote nonunion or increase the deformity at the fracture site; it is uncomfortable; it prohibits bathing, often causing chafing and discomfort in the axilla; and it may predispose to axillary vein thrombosis. Although some orthopedists continue to recommend the figure-of-8 bandage, a simple sling is sufficient to treat most clavicular fractures.

## Pitfalls of Upper Extremity Dressings

and Splints

The two most common problems with hand dressings are putting them on too tightly and leaving them on too long (Table 53-6) . One must be especially careful to avoid wrapping elastic bandages too snugly. The patient should be instructed to loosen an elastic bandage if it feels too tight. The patient should always have access to emergency follow-up care. It is often advisable to start patients on a regimen of early protected motion. This means that the patient removes the splint for a specified period, does a prescribed exercise, and then replaces the splint. A splint is not an all-or-none device, and the patient is generally weaned slowly from it before it is discarded entirely. A stiff hand is a nonfunctional one, and stiffness is often a consequence of prolonged immobilization. It is important for the patient to be made aware of his or her

responsibility for the injured hand.

# LOWER EXTREMITY SPLINTS

## Knee Splints

### Knee Immobilizer

**Indications.**

The knee immobilizer (Fig. 53-19) is commonly used for mild to moderate ligamentous and soft tissue injuries of the knee. It is removable and extremely easy to apply, making it popular among patients and physicians

| **TABLE 53-6** -- Useful Estimates of Splint Times for Various Hand Problems | | |
|---|---|---|
| **Injury** | **Splint Type** | **Immobilization Time** * |
| Mallet finger | FIN | 8 wk |
| Boutonniere deformity | FIN | 6 wk |
| Distal phalanx--soft tissue | FIN | 1 to 2 wk |
| Extensor tendon | DHWF | 3 wk |
| Sprain-strain § | | |
|   Interphalangeal joint | FIN | 1-2 wk |

| | | | |
|---|---|---|---|
| Wrist | | DHWF | 1-2 wk |
| Hand burn | | DHWF | 5-7 wk |
| Infection | | | |
| Digit | | DHWF | 5-7 days |
| Hand | | DHWF | 5-7 days |
| Severe hand contusion | | DHWF | 5-7 days |
| Fracture | | | |
| Distal phalanx | | FIN | 2-3 wk |
| Middle phalanx | | FIN | 2-3 wk |
| Proximal phalanx | | DHWF | 2-3 wk |
| Metacarpal | | DHWF | 2-3 wk |
| Carpal tunnel | | DHWF | Night only |

| | | |
|---|---|---|
| de Quervain disease | DHWF | 2-3 wk |
| Trigger finger | FIN | Night only |

* These are average times only. Every patient is treated as an individual when a splint is used. Clinical judgment is critical.
Finger splint.
Digit-hand-wrist-forearm splint.
§ The diagnosis of a sprain should be made only after a thorough effort has been made to rule out a fracture or a dislocation. This is particularly true in the wrist.

alike. In many EDs it has almost totally replaced the more bulky plaster splint. Its use should be restricted to injuries that do not require immediate surgical intervention, traction, or casting. For these injuries, in which temporary but more complete immobilization is needed, a plaster knee splint can be used because it provides better stabilization and costs much less than a knee immobilizer. The exact scientific benefit of the knee immobilizer is poorly studied and difficult to document. However, it clearly helps relieve pain and, at least theoretically, hastens healing.

**Application.**

The knee immobilizer is available in small, medium, large, and extra-large sizes. To choose the appropriate size, the knee immobilizer is placed next to the injured leg so that the tapered end lies distal to the patient's knee; if present, the cutout patellar area on the anterior aspect of the splint lies adjacent to the knee. In this position, the splint should extend distally to within a few inches of the malleoli and proximally to just below the buttocks crease. To apply the knee immobilizer, the open splint is slid under the injured extremity and firmly secured in place using the Velcro straps. The knee immobilizer can be applied directly over clothing, obviating the need to remove or cut the patient's pants.

**Posterior Knee Splint**

**Indications.**

In many EDs, the knee immobilizer has virtually replaced the plaster knee splint for mild to moderate

injuries to the knee. However, the plaster knee splint can be particularly useful in patients whose extremities are too large for the knee immobilizer, in the treatment of angulated fractures, or for temporarily immobilizing other knee injuries that require immediate operative intervention or orthopedic referral. The posterior (gutter) knee splint (Fig. 53-20 *A* and *B*) is the type most commonly applied, but as an alternative, two

parallel splints can be placed along each side of the leg and foreleg, creating a bivalve effect (see Fig. 53-20 *B*), or a long leg sugar-tong splint can be applied (see below). The bilateral knee splint is slightly more difficult to apply than the posterior knee splint, but it may provide better immobilization of the lateral and medial collateral ligaments and can be used for injuries to these structures.

**Construction.**

The knee splint is made with 12 to 15 layers of 6-in. plaster. It should run from just below the buttocks crease to approximately 2 to 3 in. above the malleoli. The sides of the splint are folded upward to form a gutter configuration.

**Application.**

A stockinette should be placed in the usual manner, and the leg should be well padded with 4- to 6-in. Webril. If available, an assistant can help elevate the leg and hold the splint in place while it is being secured with 4- or 6-in. elastic bandages. If no aide is available, the patient can be placed in the prone position and the splint laid on the posterior surface of the extremity. The leg is then wrapped in the usual manner without need for special support of the applied plaster. Also, while in the prone position, the patient's toes can elevate the lower part of the leg off the bed, allowing sufficient room to wrap the Webril and elastic bandages around the injured extremity.

**Jones Compression Dressing**

**Indications.**

A Jones compression dressing is commonly used for short-term immobilization of soft tissue injuries of the knee. It immobilizes and compresses the knee, reducing both pain and swelling. However, because it does allow slight flexion and extension of the knee, it should not be used for injuries that require strict immobilization. In addition, it is difficult to maintain the splint for more than a few days.

**Construction.**

A Jones dressing is made using 6-inch Webril and elastic bandages.

**Application.**

To apply a Jones dressing, the patient is placed on a stretcher, lying supine. If available, an assistant can elevate the patient's leg to facilitate wrapping. If no help is available, a pillow placed under the patient's heel should suffice. Webril is then wrapped around the extremity from the groin to a few inches above the malleoli. Two or 3 layers of Webril can be used, and each turn should overlap the previous turn by 25 to 50%. The elastic bandages (2 are usually required) are then wrapped around the Webril. If more support is required, the process can be repeated with another 2 to 3 layers of Webril held in

place by a second elastic bandage.

## Ankle Splints

### Posterior Splint

**Indications.**

The posterior ankle splint (Fig. 53-21 *A-C*) is one of the most common splints applied in the ED. It is used primarily to immobilize severe ankle sprains, fractures of the distal fibula and tibia, and reduced ankle dislocations. It can also be used for fractures of the tarsal and metatarsal bones or for other foot conditions that require immobilization. In particularly severe or unstable injuries, an additional anterior splint may be used to provide extra immobilization resembling that of a formal cast (Fig. 53-22) . For severe lateral or bilateral ligamentous injuries, a sugar-tong or stirrup splint (see below) may be added to the posterior splint for increased immobilization. With minor soft tissue injuries, patients may have partial weight bearing on ankle splints after 24 hours. If the patient will be bearing weight, a cast shoe worn over the splint makes it easier to walk. In addition, a cast shoe increases the longevity of the splint because walking on an unprotected splint quickly destroys the device. Generally, walking on the splint is prohibited if immobilization for more than 2 or 3 days is desired.

**Construction.**

The posterior splint is made using 4- to 6-in.-wide plaster strips. It should extend from the plantar surface of the great toe or metatarsal heads along the posterior surface of the foreleg to the level of the fibular head. If it hurts to move the toes, they should be incorporated into the splint (after padding is placed between the digits). It is a common mistake to apply a posterior splint that does not extend far enough to support the ball of the foot. Fifteen to 20 layers should be used if partial weight bearing is allowed, because this splint frequently breaks or cracks when walked on. [14]

**Application.**

The easiest way to apply a posterior splint is to place the patient in the prone position with the knee and ankle flexed at a 90° angle (see Fig. 53-21 *B*). Failure to place the ankle in a 90° angle results in a plantar-flexed splint. The supine patient may help maintain the ankle at a 90° angle by pulling up on the foot with a wide stockinette stirrup. Flexing the knee to a 90° angle relaxes the gastrocnemius muscle and facilitates ankle motion. With the knee and ankle in the proper position, stockinette may be applied and the foot and leg padded with Webril as described earlier. Extra padding is used over bony prominences, particularly the malleoli. Again, Webril or gauze is placed between the toes if they are to be included in the splint. The wet plaster is then laid over the plantar surface of the foot and secured in place by folding back the ends of the stockinette and Webril and then wrapping with one or two 4-in.-wide elastic bandages. The wet plaster is carefully molded around the malleoli and instep to ensure maximum comfort and immobilization. The toes should be left partially exposed for later

examination of color and capillary refill.  Posterior ankle splints are probably one of the most commonly used splints in the ED, and one's splinting prowess is often judged by other physicians when the patient returns for follow-up evaluation in the office. It therefore behooves the emergency physician to avoid some of the common mistakes shown in Fig. 53-21 *C*.

**Anterior-Posterior Splint**

**Indications.**

The anterior splint is never used by itself, but it can augment a posterior splint, creating a bivalve effect (Fig. 53-22) . It is used for serious fractures and soft tissue injuries of the ankle.

**Construction.**

A piece of plaster should be cut several centimeters shorter than the one used for the posterior splint, but because this splint does not bear weight, only 8 to 10 layers are required.

**Application.**

The patient should be positioned and padded as for the posterior splint. After the wet posterior splint has been applied, the anterior splint is placed over the anterior aspect of the ankle and foreleg parallel to the posterior splint. The two are then held in place with elastic bandages as described earlier for the posterior splint alone. An assistant is needed to apply the anterior-posterior splint because it is extremely difficult to hold both splints in place while wrapping the elastic bandages. Once secured, both splints are carefully molded over the instep and ankle joint.

**Sugar-Tong (Stirrup) Splint**

**Indications.**

The sugar-tong or stirrup splint (Fig. 53-23) is used primarily for injuries to the ankle. It functions like the posterior splint, and either of the two provides satisfactory ankle immobilization. In one study that compared these splints in normal volunteers, the sugar-tong splint allowed less plantar flexion and broke less often with plantar flexion than the posterior splint. [14] Also, because it actually covers the malleoli, the sugar-tong splint may protect the medial and lateral ligamentous area from further injury better than the posterior splint.

**Construction.**

The sugar-tong splint is made using 4- or 6-in.-wide plaster strips. The splint passes under the plantar surface of the foot from the calcaneus to the metatarsal heads and

extends up the medial and lateral sides of the foreleg to just below the level of the fibular head.

**Application.**

The patient is positioned, and the extremity is padded as described for the posterior splint. If both posterior and sugar-tong splints are used, the posterior splint is applied first. The wet plaster is laid across the plantar surface of the foot between the calcaneus and metatarsal heads with the sides extending up the lateral and medial aspects of the foreleg. The plaster is secured in place with 4-in. elastic bandages. The elastic bandage should be wrapped around the extremity starting at the metatarsal heads and continuing around the ankle using a figure-of-8 configuration. Once the ankle has been wrapped, another 4- or 6-in. elastic bandage can be used to secure the remainder of the splint in place. The splint should be carefully molded around the malleoli. The plaster may overlap on the anterior aspect of the ankle; this overlap does not interfere with the splint's ability to accommodate further swelling.

**Semirigid Orthosis**

**Indications.**

In patients with sprains of the lateral ankle associated with a stable joint, the use of a functional brace with early mobilization is frequently more comfortable and results in an earlier return to normal function than complete immobilization in a plaster splint or cast. Consequently, functional bracing with early mobilization has become the standard of care. However, it should be pointed out that there is no documented difference in long-term outcome between the two methods of treatment.

**Application.**

Most functional ankle braces resemble a sugar-tong splint with air bladders (Aircast, Inc., Summit, NJ) or foam padding (DeRoyal Inc., Powell, Tenn) for cushioning the malleoli. The braces are secured about the ankle by Velcro straps. The device is worn within the patient's shoe over a sock and appears to eliminate ankle instability.

**Hard Shoe (Cast or Reese Shoe)**

**Indications.**

A hard shoe can help reduce the pain associated with ambulation in patients with fractures or soft tissue injuries to the foot. This device can also be used over a splint or cast to allow partial weight bearing.

**Application.**

If the cast shoe is going to be used by a patient with a fractured toe, the injured digit

should first be buddy taped to the adjacent toe. After this is done, the patient merely slips on the hard shoe like a sandal or loafer. The shoe is then fastened with ties or Velcro straps.

**Soft Cast**

**Indications.**

A "soft cast" is basically a modified Jones compression dressing. It is useful for minor ligamentous and soft tissue injuries of the foot and ankle that do not require prolonged or complete immobilization. A soft cast can help reduce the pain and swelling often associated with mild ankle sprains and gives support for early weight bearing.

**Construction.**

A soft cast is made using 3- or 4-in. Webril and elastic bandages.

**Application.**

A soft cast is as simple to apply as the Jones compression dressing. To begin, the patient is placed in a supine position with the foot and ankle extending off the end of the stretcher. Alternatively, the leg can be elevated by an assistant or by placing pillows under the knee and foreleg. The ankle and foot are then wrapped with 2 to 3 layers of Webril, starting at the metatarsal heads and continuing around the ankle in a figure-of-8 configuration. The Webril should extend 5 to 7 cm above the malleoli and, as discussed earlier, should overlap by 25 to 50% of its width. After the Webril is in place, an elastic bandage is wrapped around the foot and ankle in a similar fashion. Additional layers of Webril and elastic bandages are seldom required.

**Unna Boot Dressing**

Ligamentous injuries of the ankle may be treated with a cloth dressing impregnated with calamine gelatin-zinc oxide (Dome paste or Unna boot) (Fig. 53-24) . [22] This moist dressing is applied like that for a rolled cast in a figure-of-8 design, from the metatarsal heads to the distal foreleg. This dressing remains moist for a few days (it should be covered with a gauze roll or an elastic bandage) and dries to a leathery consistency. It requires minimal maintenance or patient compliance, is lightweight, can be worn under a shoe, and is associated with few complications. It is cut off with scissors by the patient after 5 to 10 days, depending on symptoms. This dressing is non-yielding; hence, evaluation for ischemia is essential in the edema-prone extremity.

# COMPLICATIONS OF SPLINTS

## Ischemia

A compartment syndrome leading to ischemic injury and ultimately to a Volkmann

ischemic contracture is the most worrisome complication of cylindrical casts. Although the risk of ischemia is drastically reduced with splinting, Webril or elastic bandages can cause significant constriction. To reduce the likelihood of this occurring, the elastic bandage should not be excessively tight. If the patient has a high-risk injury, the Webril may be cut lengthwise before the plaster is applied. Elevation, no weight bearing, and application of cold packs should be stressed to each patient. Furthermore, signs and symptoms of vascular compromise should be explained carefully, and all patients whose injuries have the potential for significant swelling or loss of vascular integrity should receive follow-up in the first 24 to 48 hours. *Complaints of increasing pain under a splint should not be ignored by the physician and should not be treated with a telephone prescription for opioid analgesics*.

### Heat Injury

Many physicians are unaware of the potential for drying plaster to produce second-degree burns. [23] Thermal injury can occur with both cylindrical casts and plaster splints. Some physicians have reported a higher incidence of burns with the use of plaster splints, although the reasons for this are unclear. Table 53-5 lists factors that can increase the amount of heat that is produced during plaster recrystallization. Their effects are additive, and this fact should be taken

into account when applying a splint. For example, if 15 sheets of plaster are needed for strength in a particular splint, one should not increase the heat production further by using extra-fast-drying plaster or by reusing warm dip water. To avoid plaster burns it is prudent to use only 8 to 12 sheets of plaster when possible, to use fresh dip water with a temperature near 24 °C, and to never to wrap the extremity in a sheet or pillow during the setting process. Peak temperatures usually occur between 5 and 15 minutes after plaster wetting.

The patient should be warned that the hardening process produces warmth. The heat of drying may produce pain in patients with hemophilia-related hemarthroses. Splinting these patients may require that the plaster splint only be placed long enough to verify a proper fit; the splint is then reapplied after setting (and cooling) of the plaster. *If any patient complains of significant burning sensation while the plaster is drying, do not ignore this complaint!* Immediately remove the splint, and promptly cool the area with cold packs or cool water. Patients with vascular insufficiency or sensory deficits (e.g., diabetic neuropathy, stroke) are at high risk for plaster burns and require close observation during the drying process.

### Pressure Sores

Pressure sores are an uncommon complication of short-term splinting. [25] They can result from stockinette wrinkles, irregular wadding of Webril, incorrectly padded or unpadded bony prominences, irregular splint ends, plaster ridges, or indentations produced from using the fingers rather than the palms to smooth and mold the wet plaster. Attention to detail during padding and splinting reduces the incidence of pressure sores. However, whenever a patient complains of a persistent pain or a burning sensation under any part of a splint, the splint should be removed and the

symptomatic area inspected closely.

### Infection

Bacterial and fungal infections can occur under a splint. [26] [27] Infection is more common in the presence of an open wound but may occur with intact skin. The moist, warm, and dark environment created by the splint is an excellent nidus for infection. Also, it has been shown that bacteria can multiply in slowly drying plaster. To avoid infection, all wounds should be cleaned and debrided before splint application, and clean, fresh tap water should be used for plaster wetting. In some instances, it is preferable to apply a removable splint that allows for periodic wound inspection or local wound care.

### Dermatitis

Occasionally patients develop a rash under a cast or splint. Allergy to plaster is exceedingly rare, but there have been several reports of contact dermatitis when formaldehyde and melamine resins are added to the plaster. [30] [31] The rash is usually pruritic, with weeping papular or vesicular lesions. Because these resins are unnecessary for ED splints, their use should be avoided whenever possible.

### Joint Stiffness

Some degree of joint stiffness is an invariable consequence of immobilization. It can range in severity from mild to incapacitating and can result in transient, prolonged, or, in some cases, permanent loss of function. Stiffness appears to be worse with prolonged periods of immobilization, in elderly patients, and in patients with preexisting joint diseases such as rheumatoid arthritis or osteoarthritis. Thus splints should be left on only for the period of time necessary for adequate healing. Table 53-7 lists several injuries that commonly require splinting, along with some suggestions for length of immobilization. Fractures, dislocations, or other conditions that require prolonged immobilization (more than 7 days) should have orthopedic follow-up. Patients must be told that a splint is only a short-term device and that prolonged immobilization can be detrimental. For minor injuries, the physician can suggest that the patient use his or her own judgment about when to remove the splint, but a definite end point should be set.

## CAST PAIN

Cast-related pain is a common complaint that brings patients to the ED. Because of the potential for ischemia with circumferential casts, all complaints should be fully investigated, and vascular compromise must be ruled out. A detailed history and physical examination should be performed on all such patients. The nature and onset of the pain is of particular importance. A dull, nonspecific pain that has worsened gradually since the time of injury may be the only clue to an early compartment syndrome (see Chapter 58) . The sudden onset of throbbing pain associated with swelling and redness suggests a possible deep venous thrombosis. In both of these cases, rapid intervention is the key to decreasing morbidity and mortality. The physical examination should pay particular attention to the areas of tenderness and the effect of active and passive

movement of the involved muscle group on the severity of pain.

With a *compartment syndrome*, tenderness over the involved compartment is a common finding; stretching or

| TABLE 53-7 -- Suggested Length of Immobilization for Conditions That Frequently Require Splinting | |
|---|---|
| **Condition** | **Length of Immobilization (Days)** |
| Contusions | 1-3 |
| Abrasions | 1-3 |
| Soft tissue lacerations | 5-7 |
| Tendon lacerations | Variable * |
| Tendonitis | 5-7 |
| Puncture wounds and bites | 3-4 |
| Deep space infections and cellulitis | 3-5 |
| Mild sprains | 5-7 |

| Fractures and severe sprains | Variable |
|---|---|

* There is considerable controversy surrounding the length of immobilization for tendon lacerations, and duration therefore is best left to the orthopedic or plastic surgeon.
Usually requires prolonged immobilization; best determined by an orthopedic surgeon

contracting ischemic muscle also elicits significant pain. The examination should also evaluate the presence and quality of distal pulses, amount of edema fluid present, distal sensation, capillary refill, and color and temperature of the digits. The finding of pain, pallor, paresthesias, paralysis, and pulselessness (the five Ps) are said to be pathognomonic for ischemia. Unfortunately, they seldom occur simultaneously, and their presence together is usually a late finding that carries a poor prognosis. Hence, the emergency physician must maintain a high index of suspicion for possible ischemia and remove the cast if any possibility of vascular compromise exists. Almost any cast can be bivalved and reapplied after inspection without significant loss of short-term immobilization.

To loosen a cast, an oscillating cast saw is used to cut along the medial and lateral aspects of the cast (Fig. 53-25 *A* and *B*). This is called *bivalving* the cast, and it allows the halves to be spread and reapplied in a less constricting manner while still maintaining proper immobilization. To use the oscillating power saw, proceed with a series of downward cutting movements facilitated by wrist supination, removing the blade between cuts. The blade is removed between cuts to prevent it from getting hot enough to burn the skin. This is particularly important if synthetic materials have been used in the cast. Also, the blade should not be allowed to slide along the skin, and the saw should not be used on unpadded plaster. In the apprehensive patient, the physician can demonstrate that the cast saw blade only vibrates (it does not turn) and that it does not cut the skin. After completely cutting through the medial and lateral sides of the cast, the two halves are separated using a cast spreader, and the padding is cut lengthwise with scissors. This may be sufficient to relieve early ischemia if the problem is simple post-injury swelling, but both the padding and cast can be removed totally to inspect the injured area if necessary. If ischemia cannot be ruled out, compartment pressures should be measured, and an orthopedic consultation should be obtained. If vascular integrity is established and no other problems are found, the bivalved cast can be replaced. The extremity should first be padded in the usual manner using fresh Webril. The cut ends of the bivalved cast are then lined with white adhesive tape, and the cast is replaced around the extremity. Finally, the cast is secured in place using elastic bandages.

If plaster sores are the cause of the patient's discomfort, the physician who placed the cast should be consulted. In some cases, additional padding is all that is needed, but in others a window should be cut out over the problem area. Because pressure sores can lead to significant tissue necrosis, the patient should receive follow-up care within 24 hours.

If the patient's problem is plaster (or more likely, resin) dermatitis, treatment generally

consists of topical or oral steroids and antihistamines. Therapy should be done in concert with an orthopedic surgeon because the patient may require admission for other forms of immobilization until the cast can be replaced. With mild cases, changing the cast or splint and using antihistamines for symptomatic relief may suffice. All of these patients should receive close follow-up, and if the condition does not improve, the cast must be removed.

## CONCLUSION

Splinting represents an important means of temporary fracture immobilization and provides protection and comfort for a variety of soft tissue injuries. The clinician should be aware of potential complications including ischemia, thermal injury, and pressure sores, which can occur with improper splint application. Proper technique should minimize the risk of these adverse outcomes. The emergency physician also should be facile in the release of circumferential cast and splint materials when ischemia is suspected.

# Chapter 54 - Podiatric Procedures

**Lancing P. Malusky**

A painful foot frequently motivates a person to seek professional medical care more readily than other limited musculoskeletal conditions, because mobility is compromised. This chapter addresses some common presentations of and procedures for the painful foot. A number of procedures (e.g., local and regional anesthesia, foreign body removal, soft tissue injections) are addressed in detail elsewhere in the text. These procedures also are discussed here in a limited fashion when they are relevant to the unique anatomy or function of the foot. The management of a paronychia, felon, and subungual hematoma is discussed in Chapter 40 . Joint aspiration for synovial fluid analysis is discussed in Chapter 57 . Lower extremity splinting techniques are discussed in Chapter 53 . Joint reduction techniques for digital and ankle dislocations are discussed in Chapter 52 .

## LOCAL AND REGIONAL ANESTHESIA

Most invasive podiatric procedures require local or regional anesthesia. A digital or metatarsal block is administered in the same fashion as that for anesthesia of the hand (see Chapter 32) . Because the digital arteries are end arteries, anesthetic agents without epinephrine are recommended. [1]

Although field blocks and local infiltration are generally adequate for anesthesia of the dorsal surface of the foot, regional nerve blocks at the ankle are preferred for anesthesia of the volar (plantar) surface (see Chapter 32) . The plantar surface is thickly calloused, but it is also exquisitely sensitive. Distention of the tissues by local infiltration on the plantar surface can be quite painful.

A device that has been used to facilitate placement of a skin wheal before deeper soft tissue infiltration is the MadaJet (Gill Podiatry Supply Company, Middleburg Heights, Ohio) (Fig. 54-1) (Figure Not Available) . The device mechanically injects a skin wheal using 0.1 mL of anesthetic without a needle. The device should be loaded daily with fresh anesthetic. After cocking the MadaJet, the aperture is placed about 1 cm from the skin site for anesthetic placement. The patient is warned about feeling a momentary sensation similar to a rubber band snap. The raised wheal area is instantly numbed, and injection of the remainder of the anesthetic using a standard needle and syringe technique can be begun immediately. The MadaJet is not recommended for the thicker adult plantar skin.

The physician who intends to use a jet injector should review and practice current disinfection recommendations. These devices have been linked with hepatitis B [2] and *Mycobacterium chelonei*[3] spread.

An alternative technique is to use a vapocoolant (e.g., fluorimethane or ethyl chloride) to numb the overlying skin prior to initiating needle puncture. The liquid is sprayed from a distance so that it begins to vaporize shortly after making skin contact rather than

running down the skin surface prior to vaporizing. Once the skin frosts, the superficial nerve endings have been deadened, and the operator can then numb the deeper tissues. If a plantar area is to be anesthetized and a tibial nerve block is not efficacious, this technique may minimize the pain of plantar injection. However, whenever possible, a less sensitive lateral, medial or dorsal approach site should be used.

## SHAVING OF PLANTAR LESIONS

The plantar skin is the thickest skin of the entire body and is especially designed to adapt to environmental influences in its effort to protect the foot's internal structures. Shearing forces and high pressure points will create hyperkeratoses on the plantar skin. These hyperkeratotic areas further focus weight-bearing forces in the foot, hence producing discomfort.

When the verruca virus is introduced to the plantar skin, its presence also frequently induces a hyperkeratotic response (i.e., wart). Unfortunately, *the definitive treatment methods for a wart are at odds with the appropriate regimen for a callus*. The wart should be treated with some form of epidermal eradication (see Curettage of Verruca and Porokeratosis), whereas the hyperkeratosis requires simple debridement with podiatric rebalancing of the weight-bearing surface.

Most hyperkeratoses occur directly over an area of focal friction or pressure, such as beneath a metatarsal head, on the ridge of the calcaneal skin, or adjacent to a digital bony prominence that is experiencing excessive shoe pressure. Verrucae may occur on any site in the plantar skin; they are less likely to occur on the digits and are rare on the dorsal skin.

Other, less common skin lesions can mimic the wart. A common error is to assume that a macerated lesion between two digits, especially at the crease between the fourth and fifth toes, is a verruca. This lesion is more likely a "soft corn" (heloma molle), discussed later. Another common misdiagnosis is porokeratosis plantaris discretia ("poro"), which is a small cyst-like lesion found in plantar locations and is histologically a sweat duct filled with a cone of keratin. This lesion often has been given the misnomer of "seed wart."

A "hard corn" (heloma durum, hammertoe, clavus) is a hyperkeratosis that occurs adjacent to a bony prominence on a digit. Hammertoes occur when the intrinsic (foot) muscles lose their stabilizing influence on the interphalangeal joints. The extrinsic (leg) muscles then overpower, producing flexion at the interphalangeal joints. Friction and pressure over the bony prominence, usually the proximal phalangeal head, cause a keratinization process that attempts to protect the dermis from penetration. The heloma durum may become macerated when it occurs between the toes, therefore becoming a heloma molle (soft corn) that may even perforate to the fascia. The heloma durum may become so chronically inflamed from recurrent friction or infection that a small sensitive neurovascular bundle (heloma milliare) may form amidst the keratosis.

Application of alcohol to the plantar lesion enhances skin line visibility. The skin lines of a hyperkeratosis should pass through the lesion or, in the case of severe pressure or

scarring, may flow into and disappear in the lesion. In contrast, the skin lines should pass around a verruca, which has a cystic capsule. A plantar verruca frequently has a coexistent surrounding hyperkeratosis, a response to the additional friction from the foreign mass. Differentiating a plantar verruca from a plantar hyperkeratosis also may be enhanced by observing the patient's response to pressure on the lesion. Direct pressure on a keratosis often elicits a more painful response than a squeezing maneuver of similar force. The reverse is true for a verruca.

## Indications

The lesion type may be more definitively determined by debriding the keratinized layers of skin. The clinician examines the shaved surface, seeking specific diagnostic signs of verrucae. Warts display the minicauliflower-like appearance of the papillomatous formation and the small capillary ends, which appear as black dots in the lesion. The process of debridement also affords a measure of temporary relief from the discomfort of the lesion. Should the shaving reveal a keratosis, more definitive shaving should be performed. If a wart is found, definitive curettage or podiatric referral should be provided.

## Equipment

Although a No. 10 or 15 Bard Parker scalpel blade on a handle may be used for debridement, these instruments are frequently difficult to handle when trying to debride thin layers of tissue from an oblique angle. Safer, more convenient instruments are the Beaver miniblades and the Gill chisel blade No. 312 and 313 with appropriate handles (Gill Podiatry Supply Company, Middleburg Heights, Ohio) (Fig. 54-2) (Figure Not Available) . When these instruments are held like a pencil, the physician can achieve a cutting angle more parallel to the skin.

## Technique

After the hyperkeratotic area is softened in warm tap water for 10 to 15 minutes, the patient is placed in a chair, and the foot is elevated to a comfortable eye level for the clinician. Heel lesions are often debrided with the patient in the prone position. After appropriate anesthesia for very sensitive lesions, the lesions are shaved under a bright examination light. A fresh, sharp blade is used for each patient.

The lesion is further moistened with an alcohol swab, and the skin is grasped above the area to be debrided using the fingers and below the area with the thumb of the nondominant hand. With the skin under traction, the Beaver blade handle is held like a pencil and placed at a 10° to 40° angle to the skin surface. The clinician rests or presses the knuckles or fingers of the dominant hand on the patient's foot and begins debridement tangential to the area of skin traction (Fig. 54-3) (Figure Not Available) . The rhomboid-shaped blade is held so that the end of the cutting edge distal to the clinician is wider than the proximal edge. Short, superficial strokes toward the clinician are used until a feel for the tissue and a feel for the patient's reactions are established. This hand placement technique provides a safety buffer for the patient, because if the

patient jumps, all materials move with him or her.

The skin surface to be pared should buckle outward to some extent. Using only the middle two thirds of the cutting edge, the layers are pared, and the site is repeatedly examined. A fresh swipe of an alcohol swab aids inspection. The clear, plastic-like appearance of keratinized skin over or around the shaved area identifies the safe debridement zone. No nerves or capillaries should exist in this tissue. However, even an experienced clinician occasionally inadvertently opens an end capillary, which bleeds into the field and obscures the diagnosis. The topical astringent Lumicain, which contains aluminum chloride (Medical Products Laboratories, Philadelphia) seals these bleeders off, but its application may be momentarily uncomfortable for the patient who has not had the lesion anesthetized before shaving. After initial shaving, the lesion should be more easily identified.

Plantar skin that thickens beneath a palpably prominent metatarsal head or heads is termed a *callus*. When this occurs in combination with a discrete, centrally bruised plug of keratin, it is called an *intractable plantar keratosis*. These lesions may require periodic podiatric evaluation for debridement, protective padding, and balancing of the weight-bearing surface with orthotics.

The routine corn is treated with periodic debridement and felt corn pads with an aperture to transfer pressure to the surrounding healthy skin (Fig. 54-4) (Figure Not Available) . The perforated soft corn is frequently misdiagnosed as an interdigital verruca by visual inspection. After a regional block, the lesion should be debrided with a mini-Beaver knife and a No. 64 blade for clearer diagnosis and relief of pain. Subsequent home care for this lesion includes astringent boric acid soaks twice daily, antibiotic cream or ointment applied topically, and a small, opened gauze pad laid into the web crease. The toes should be separated during healing with a cotton ball placed appropriately distal or proximal to the lesion or a small gauze pad taped in place. The soft corn may later require a foam interdigital cushion. All of these hyperkeratotic lesions are candidates for podiatric referral for bony correction if conservative care does not relieve pain adequately.

Definitive management of verrucae and "poro" requires treatment by curettage, acid, or freezing. Considerable comfort can be obtained before podiatric referral, however, by debridement under regional anesthesia.

## CURETTAGE OF VERRUCA AND POROKERATOSIS

### Indications

The preferred treatment regimen for the common verruca plantaris (plantar wart) varies among physicians and specialties, but all procedures concentrate on separating the verruca and epidermis from the basement membrane. Salicylic acid lyses the tissue interface, liquid nitrogen blisters it with cold, and the hyfrecator and the low-dose $CO_2$ laser blister it with heat. The curette mechanically separates the 2 skin layers. When the pain of a wart is severe because of its prominence, curettement may provide definitive

therapy.

## Technique

After placement of a regional nerve block (i.e., posterior tibial nerve block), several sterile curettes (Fig. 54-5) (Figure Not Available) , tissue nippers, and a hyfrecator needle are readied. An oval curette with a more pointed end is initially chosen. The skin distal to the site of the verruca is stretched taut with the physician's nondominant hand, and the handle of the curette is rested in the palm of the dominant hand. The dominant hand index finger guides the curette in a sweeping motion, directed opposite to the skin-stretching vector of the stabilizing hand (Fig. 54-6) (Figure Not Available) .

If the skin surrounding the verruca is still thick, the curette may face too much resistance, and the clinician may wish to give the curette a head start by making a snip 2 or 3 mm deep in the skin, just distal to the lesion in normal epidermis, using tissue nippers (see Fig. 54-5 (Figure Not Available) *B*). Larger lesions may require larger curettes; choosing the right size curette initially will simplify the process. Warts are cystic in structure and generally separate easily from normal tissue once the lesion begins to break away (Fig. 54-7) (Figure Not Available) . The clinician will appreciate the basement membrane when the curette scrapes on the skin lines. This process may mimic a metallic chime when encountered. The basement membrane is the protective layer of the dermis, and by design, the normal curette does not penetrate this zone unless undue force is used.

Some clinicians hyfrecate the lesion first to blister the wart and make curettement through the epidermis easier. Once the verruca has been thoroughly curetted with the various curettes, the hyfrecator may be used to sweep the area to loosen or destroy adjacent skin and discourage regrowth. If any purulence is uncovered, the base of the lesion should be inspected for the existence of a foreign body, which may be the original source of the virus.

Postoperatively, the treated area looks and feels like a blister to the patient, and 2 to 4 weeks of topical antibiotics and a gauze pad are commonly prescribed. Periodic debridement of the eschar may be needed to assist wound drying and resist plug formation. The recurrence rate is about 25% for most verruca procedures, and the patient should be warned to watch for regrowth.

The *porokeratosis* curettement process is similar to the verruca curettement process, but a sharper, smaller curette is needed. Whereas the verruca provides a relatively mild, broad-based pressure on the basement membrane, the porokeratosis creates a narrower, deeper pressure that may depress, scar, or even obliterate the dermis. The porokeratosis excision is begun with the same hand motions, but the narrow, cone-shaped lesion is found and followed into the basement membrane. The base appears bruised, and no hyfrecation is needed. A recurrence rate of 30% to 40% is common, and recurrence is more likely if the lesion is over a weight-bearing metatarsal head. Many podiatrists use a $CO_2$ laser on this lesion, and reported recurrence rates are <15%. Postoperative care is similar to that for wart curettage, but postoperative

debridement of the eschar plug may be needed during follow-up care.

# FOOT PAD USE

Foot pads are used to redistribute pressure applied to a tender, inflamed area of the foot. The type of pad used and its placement are dependent on the underlying problem. A number of commercial aperture pads are available for protection of bony prominences (Fig. 54-8) . These pads are helpful for management of corns and hyperkeratoses secondary to hammertoe deformities (Figs. 54-4 (Figure Not Available) and 54-9) (Figure Not Available) . The use of these shields for management of bunions is discussed subsequently.

## Metatarsalgia

Firm palpation that elicits pain directly beneath the metatarsal head can be diagnosed as a metatarsal bruise or contusion if it involves 1 head, or metatarsalgia if it involves more than 1 head. The metatarsalgia patient complains of discomfort that comes on during walking, becomes progressively worse during the day, and is relieved only by rest. The patient can often identify the painful spot for the clinician. Frequently the pain is felt beneath the second or the second and third metatarsal heads.

The physician palpates each of the lesser metatarsal heads and identifies the site of pain under the metatarsal head or heads. A hyperkeratosis may be associated with this condition, either diffusely under the central lesser metatarsus or discretely beneath a metatarsal head. Debridement of the hyperkeratosis (as discussed earlier) is important for diagnosis and treatment. A discrete lesion may be the real origin for discomfort, and adequate debridement and podiatric follow-up to address the source of the hyperkeratotic reaction may be sufficient emergency department therapy.

### Therapy

Any hyperkeratosis should be cushioned with a pad (e.g., Spenco [Gill Podiatry Supply Company, Middleburg Heights, Ohio] or PPT [Langer Biomechanics Group, Inc., Wheeling, Ill.]). For discrete pain beneath the second metatarsal head, which may be a flexor hood capsulitis (see below), a sub-first metatarsal head raise is accomplished using a 1-cm-thick PPT pad placed beneath an innersole (Fig. 54-10) (Figure Not Available) . Nonsteroidal anti-inflammatory drugs and a quality athletic or walking shoe should also be recommended. Continued symptoms require podiatric follow-up.

## Hallux Valgus (Bunion)

The basis for a bunion is complex and represents a dynamic imbalance between the structures on the lateral side of the first metatarsophalangeal (MTP) joint and the medial side of the MTP joint (Fig. 54-11) (Figure Not Available) . [4] [5] When there is lateral displacement of the distal hallux, a bunion begins to form. A number of anatomic, hereditary, physiologic, and footwear elements contribute to bunion development.

The typical bunion deformity develops in a woman wearing heels occupationally. She may state she has numbness on the distal medial aspect of the hallux (from compression of the terminal branch of the medial dorsal cutaneous nerve). She may recall a time when the intermetatarsal space was tender or when the area of the sesamoid apparatus was tender for a short time. Other foot complaints, such as lesser metatarsalgia, intermetatarsal neuroma, hammertoes, ingrown toenails, and corns and callus, may be common. First MTP joint bursitis commonly occurs over the medial bunion bump. Finally, gout should be considered, even in women.

**Therapy**

Removal of the offending footwear is advised, or a hole may be cut in the shoe to allow the sensitive area freedom from pressure. A precut bunion shield (Figs. 54-8 *C* and *D* and 54-12 (Figure Not Available) ) may be dispensed or constructed from 1-cm-thick felt. Nonsteroidal anti-inflammatory drugs are also commonly used. When inflammation is severe, aspiration of the bursal fluid with a 20-ga needle followed by instillation of 0.5 mL of a local anesthetic and a short-acting steroid (e.g., 4 mg of dexamethasone) can be performed.

## Flexor Hood Capsulitis

This condition is a common overuse presentation that is sometimes mistaken for metatarsalgia. Direct palpation of the dorsal or lateral metatarsal head will *not* be painful, but tenderness will be elicited directly beneath the proximal phalangeal shaft. The MTP joint is similarly nonpainful when palpated from the dorsum or axially loaded, thus ruling out an arthritis.

The flexor hood mechanism is a thin retinacular band that holds the flexor digitorum longus/brevis tendon complex close to the digit's proximal phalanx. A poorly functioning bunion foot, with its ineffective medial column, may overuse the second and, rarely, the third digit. The toe may hammer, causing the flexor plate that normally articulates beneath the metatarsal head to be pulled distally. Edema of these tissues may cause the involved toe to abduct from the neighboring digit. Treatment is the same as for metatarsalgia (see Fig. 54-10) (Figure Not Available) ; in addition, reinjury should be reduced by using footwear that restricts digital dorsiflexion at the MTP joint. A well-cushioned, hard-soled Reese shoe is useful for this condition.

## Heel Pain Syndromes

In-depth discussion of the multiple causes of heel pain is beyond the scope of this text. The use of foot pads as adjunctive therapy for these conditions is addressed later. Other supportive therapies for most of the entities include modification of physical activity, use of nonsteroidal anti-inflammatory drugs, local injection of anesthetic and steroid agents, and use of orthotic footwear.

**Heel Spur Syndrome**

The patient presents with heel pain that gradually worsens over several months. Palpation elicits pain about the medial border of the calcaneus, possibly extending distally at the radiographic site of a bony spur ledge (Fig. 54-13) (Figure Not Available) . Supportive therapy is helpful, including a local steroid injection (e.g., 10 to 20 mg methylprednisolone). Injections are performed from the medial aspect of the calcaneal spur, thus avoiding the sensitive plantar area. The medication is distributed along both the medial calcaneal tubercle and the central plantar calcaneus beneath the spur fascia junction. Shoe support in the form of a heel raise of 1.25 cm bilaterally reduces heel weight bearing. A temporary donut-shaped heel cushion cut from 1-cm-thick PPT or a similar high-density foam rubber also suffices for this purpose. Podiatry follow-up, especially for the severely pronated or supinated foot, should be considered.

**Retrocalcaneal Bursitis**

With this condition, pain is present at the specific site of the Achilles tendon insertion. Either or both bursae may be sensitive, and the clinician can palpate for either (see Fig. 54-13) (Figure Not Available) by relaxing the Achilles tendon with plantar flexion of the foot. Tenderness when a squeezing force is applied anterior to the tendon in the depression above the calcaneus suggests inflammation of the retrocalcaneal bursae. Tenderness with thumb pressure on the back of the insertion of the tendon behind the calcaneus suggests inflammation of the postcalcaneal bursa. Treatment is identical to that for heel spur syndrome, with the exception that steroids, when used, are injected into the inflamed bursae. A small steroid dosage is recommended to reduce the risk of atrophy of the Achilles tendon insertion. A PPT heel pad lift or varus pad in the shoe is also helpful. All shoes worn at home and work by the patient should have a uniform heel height with a nylon cleat on the lateral side.

**Calcaneal Apophysitis (Sever Disease)**

This condition commonly occurs in active children between the ages of 8 and 14 years old. As a result of incomplete fusion, the apophysis at the plantar posterior aspect of the juvenile's heel is stressed at its cartilaginous connection by the Achilles tendon. Great tenderness is evident with palpation directly over the apophysis. There should be no significant tenderness of the Achilles tendon, and there is usually no edema. Treatment is identical to that for retrocalcaneal bursitis, with the exception that steroid injections are not recommended.

**Other Conditions**

A painful heel may be due to other conditions, including a partially ruptured Achilles tendon, gouty and psoriatic arthritis, and calcaneal stress fractures. These conditions are generally identified by history and physical examination; specific therapies for these entities are discussed in general emergency medicine and orthopedic references.

# TARSAL TUNNEL SYNDROME AND SINUS TARSITIS

## Tarsal Tunnel Syndrome

The tarsal tunnel syndrome (TTS) is similar to carpal tunnel syndrome in the upper extremity. TTS is caused by compression of the posterior tibial nerve in a restricted anatomic channel while traveling around the medial malleolus. The other structures in the tibiotalocalcaneal canal are the posterior tibial, flexor digitorum longus, and flexor hallucis longus tendons; the double posterior tibial veins; and the posterior tibial artery. Inflammation or disease of any of these structures may cause secondary inflammation of the nerve. These structures are held in place by a flexor retinaculum. The posterior tibial nerve trifurcates, sending off first the medial

calcaneal branch (this branch may become involved in the heel spur syndrome) and then splitting into the larger medial and lateral plantar nerves. These plantar nerves travel deep to the abductor hallucis muscle belly and into the deep fascia of the sole of the foot. There they serve motor, sensory, and proprioceptive functions.

TTS may present with a sensation of burning at the ball of the affected foot, usually in the second through fifth sub-metatarsal head areas. Percussion over the posterior nerve posteromedial to the medial malleolus may reveal a positive Tinel's sign. Because this condition may be due to a variety of causes, referral to an orthopedist or podiatrist is recommended. Emergency department management may include inversion position taping or Unna boot application to reduce nerve compression or use of nonsteroidal anti-inflammatory drugs. Severe cases may be treated with short-acting corticosteroids (e.g., 1.5 mL of dexamethasone acetate suspension [8 mg/mL]) and local anesthetic (e.g., 2 to 3 mL of 0.25% bupivacaine) injected into the canal at the level of the medial malleolus.

## Sinus Tarsitis

This condition, also known as *tarsal canal syndrome,* consists of inflammation within the subtalar (talocalcaneal) joint. This anatomic canal runs obliquely from proximal medial to distal lateral. The only intrinsic dorsal foot muscle, the extensor digitorum brevis, takes origin from the calcaneus here. Trauma, inflammation, or mass (e.g., ganglion) in the canal may produce a tight band sensation obliquely across the ankle below the level of the malleoli.

The diagnosis may be made by injecting a local anesthetic agent directly into the canal. The insertion point is palpated as a depression just anterior to the tip of the lateral malleolus. Injection of 3 mL of 2% plain lidocaine should be sufficient to resolve symptoms. Further therapy with a repository steroid, such as 2 to 8 mg of dexamethasone acetate, can be used. Consultant referral is recommended to differentiate this condition from a variety of disorders, including peroneal spastic flatfoot, tarsal coalitions, Charcot joint, and degenerative joint disease.

# INJECTION THERAPY FOR A NEUROMA

A forefoot neuroma, also known as a *Morton neuroma,* is a painful condition that is most likely to affect the female adult, especially if she wears heels occupationally. The neuroma is usually unilateral, but it may also occur bilaterally. This lesion frequently occurs in the third intermetatarsal space but may occur in the second interspace; it rarely occurs in the first or fourth interspace. Its preponderance for the third space is thought to be due to dual innervation of this interspace by the lateral plantar nerve and the medial plantar nerve and to the fact that the fourth metatarsal is the most mobile. An intermetatarsal ligament (deep plantar aponeurosis) connects each metatarsal head on its plantar aspect, and the sensory nerve passes beneath this ligament (Fig. 54-14) (Figure Not Available) .

In high heels, the sensory nerve is forced to bend around this ligament and is compressed against it during weight bearing. The perineural covering becomes fibrotic from chronic irritation. Although the normal sensory nerve is the size of a pencil lead, the neuroma is the size of a pencil. The damaged nerve trunk no longer conducts normal impulses, and a burning pain, numbness, or other paresthesia is reported in the third or fourth toes. A feeling of a lump, cord, or rolled up sock is often expressed. A classic diagnostic feature is when the patient states that he or she had to sit down on a curb and massage the foot for relief.

The condition is diagnosed using careful forefoot palpation to rule out arthritis, metatarsalgia, capsulitis, ganglion, or stress fracture. The space between and just distal to the metatarsal heads is squeezed, and the resultant pain suggests a neuroma. A tender spot may be noted in the depression just behind and between the involved metatarsal necks, where the nerve first encounters the intermetatarsal ligament. Pain produced with palpation distally in the intermetatarsal sulcus also suggests a neuroma. The pain is reproduced by squeezing the metatarsal heads together. The differential diagnosis includes ganglion, MTP joint capsulitis, and stress fracture. A common diagnostic maneuver is to instill 2 mL of 1% or 2% lidocaine in the intermetatarsal space, just proximal to the ligament and in the fascia just deep to the plantar skin. The injection is done from dorsal to plantar. If the pain is totally relieved on repeat palpation maneuvers, a Morton's neuroma is likely.

## Therapy

A serial treatment plan may include a reduction in heel height, a cushioned innersole such as PPT or Plastizote, sub-fourth metatarsal shaft raise (Fig. 54-15) (Figure Not Available) , and local injection. After skin preparation, the tender neuroma is injected with a 1- to 2-mL mixture of a long-acting local anesthetic (e.g., 0.5% bupivacaine) and steroid (e.g., 5 to 10 mg methylprednisolone). The injection is made either directly into the lesion or indirectly into its fascial plane, as described above. The previous local anesthetic diagnostic block will make this steroid injection more tolerable. The dorsal venous arch is frequently encountered when performing this injection; thus, careful aspiration prior to injection should be performed. The patient also should be warned that a harmless ecchymosis is common following this procedure. Podiatric follow-up for

additional care is warranted.

## ASPIRATION AND INJECTION OF A GANGLION

A ganglion is histologically similar to a synovial sheath and frequently contains synovial fluid. The ganglion is considered traumatic in origin and usually has a stalk traceable to a tendon sheath or joint capsule. Although the ganglion is easily diagnosed when it occurs over a tendon on the dorsum of the foot, it can be a challenging diagnosis when present among the compact forefoot structures. This lesion may mimic the neuroma or MTP joint capsulitis.

When the clinician finds edema in the forefoot, the location of this swelling is a key diagnostic consideration. A neuroma usually does not cause edema of the intermetatarsal space unless it is unusually large. An MTP joint capsulitis causes swelling at the MTP joint, perhaps brawny in nature, but careful palpation confirms its location. A small ganglion causes swelling directly beneath the flexor apparatus of the involved digit or perhaps directly beneath a metatarsal head or the tibial sesamoid. The larger ganglion grows in the path of least resistance, which is to an available intermetatarsal space. If painful, a ganglion associated with the lesser metatarsal structures will provide a localized stinging or burning sensation on palpation, whereas the neuroma's pain travels out into the involved toes. The ganglion should roll under the examiner's fingers. If the lesion is painless and nonmovable, other soft tissue neoplasms should be considered. An extremity coil magnetic resonance image enhanced by gadopentetate may aid in this diagnosis and help rule out or stage the rare malignancy.

### Therapy

When the clinician suspects that the lesion is a ganglion, the diagnosis may be confirmed and treatment begun by aspiration of synovial fluid. The physician, with local or regional anesthesia, blocks the tender area and anticipated skin penetration site. After skin preparation, a 20-ga needle is inserted, and under negative pressure, the suspected ganglionic structure is explored for fluid. When fluid is encountered, the lesion should be drained. The fluid should have a yellow, thick, gelatinous character. Further aspiration and exploration with rotation of the bevel of the needle should be performed. Upon needle withdrawal, the lesion may be further squeezed to drain the rest of the fluid. After fluid drainage, infiltration with a steroid solution and local anesthetic is appropriate to enhance resolution. Although a small volume (<0.5 mL) will reexpand the cyst somewhat, the medication is generally absorbed within 24 hours. Fluid is not always found, but if the clinician is certain of the diagnosis, steroid infiltration may still be entertained. The ganglion frequently remains deflated and asymptomatic, although recurrences are common and warrant specialist follow-up.

## SPLINTING TOE FRACTURES

Although many clinicians and laypersons shrug their shoulders at a fracture of a toe and state that nothing can be done, something can and should be done to relieve the pain

and encourage healing. As with any other fracture, attention should be paid to the possibility of breach of joint cartilage, hypermobility of fracture segments, and malposition and malunion of the fracture segments.

Aggressive fracture reduction is indicated for great toe proximal phalanx fractures, as it represents the main propulsive segment of the forefoot. A plaster cast is *insufficient* treatment *except* in cases of a simple nondisplaced fracture through the body of a great toe phalanx. Any displacement should raise concern regarding the possibility of an axial rotation or a deviation in the way the hallux interacts biomechanically within its own interphalangeal or MTP joint. In the acute setting, a non-weight-bearing ankle splint with attached plantar component extending beyond the great toe provides protection until the patient with a complicated great toe fracture can obtain follow-up. Open fractures obviously need immediate referral for debridement and continued antibiotic therapy.

A fracture of the first MTP joint *sesamoid* bone may result from a jump from a height. The great toe sesamoid bones lie in grooves on the bottom of the metatarsal head. Each bone lies within the tendon of its respective flexor hallucis brevis muscle belly. With a sesamoid bone fracture, there is localized pain on the plantar aspect of the first metatarsal head. Bipartite sesamoids (tibial more frequently than fibular) are common, and bilateral radiographs are helpful to clarify whether the radiographic changes represent a true fracture.

Fractures of the lesser toes usually result from jamming

the toe into a nightstand or bedpost while barefoot. Radiographs of the phalanges will confirm the clinical suspicion.

**Therapy**

For a tibial sesamoid injury, an aperture bunion-type pad, reinforced medially, of 0.5- to 0.75-cm-thick felt protects the sesamoid and transfers weight bearing to the surrounding structures (Fig. 54-16) (Figure Not Available) . A Reese shoe and nonsteroidal anti-inflammatory drugs are also helpful. Subsequent radiographs rarely show bony consolidation, but the fracture interface appears smoother.

The treatment for closed lesser phalangeal fractures is "immobilization" for 6 weeks. The patient is ideally put in a less restrictive, stiff-soled shoe or a Reese orthopedic shoe. As the fracture is reduced, the injured toe is secured against an adjacent noninjured toe. A soft corn pad is placed between the toes, and the toes are held together with an adherent wrap around both digits (Fig. 54-17) (Figure Not Available) . Thus, the normal toe splints the injured toe. The procedure is demonstrated to the patient or family and materials are dispensed so that the splint can be changed every 2 to 3 days at home.

# PLANTAR PUNCTURE WOUNDS

Plantar puncture wounds often present a diagnostic and therapeutic dilemma for the

clinician. [8] (See also Chapters 36 and 39.) Considerable controversy exists regarding the proper initial management of these wounds. Although up to 98% of such wounds are produced by nails, [9] various other objects have been reported as causing these wounds, including other metal objects, wood, and glass. The host response to the injury is dependent on the penetrating material, location of the wound, depth of penetration, footwear, time from injury until presentation, and underlying health. Because superficial puncture wounds generally do well, the depth of penetration may be a primary determinant of outcome. This may in part explain why distal wounds in the MTP joint area that are associated with increased weight bearing during ambulation are often more serious. [10]

Because most minor puncture wounds are not seen in the emergency department, the true risk of infection, and in particular osteomyelitis, remains speculative. One review of these wounds suggests that no more than 2% to 8% of puncture wounds become infected, and only a small percentage of these go on to develop osteomyelitis. [8] The retention of foreign material (e.g., a portion of a tennis shoe sole) in the wound is an important factor in persistent infection. One prospective series suggests that only the presence of symptoms (e.g., redness, tenderness, increased swelling) at 48 hours was associated with the risk of infection or potential retained foreign body. [11]

**Therapy**

The approach to the patient is dependent on several factors, including the time from wounding to presentation, the suspicion regarding a foreign body, and the presence of infection (Fig. 54-18) (Figure Not Available) .

Patients presenting within 24 hours and without signs of infection generally require only simple wound care. [11] Although irrigation of all exposed dermal tissue is recommended, high-pressure irrigation of deep tissues with distention of soft tissue is unlikely to be helpful.

The extent to which a foreign body workup is pursued depends on the history and physical findings. When a patient clearly states that the puncture was produced by a new nail that was removed intact, a radiographic series or local wound exploration for a retained metallic foreign body is not needed. When the wound is large and the patient was wearing rubber-soled shoes, however, local wound exploration may be warranted to exclude the possibility of a retained portion of the shoe. Exploration should be performed under a regional anesthetic block after an ellipse of tissue has been removed from around the puncture site. Blunt probes should be used. General management of suspected foreign bodies is discussed in further detail in Chapter 39 .

Patients who delay presentation for medical attention have an increased risk for a retained foreign body, since delayed presentation is often due to the development of inflammation. Unless the patient presents without infection or with a clearly superficial cellulitis expected to respond to simple oral antibiotics, local wound exploration is warranted, as discussed earlier. Recurrent infections, deep soft tissue tenderness, and increasing soft tissue swelling indicate the probability of a retained foreign body or deep space infection. Such patients require prompt specialty referral. Consideration of the

need for additional diagnostic studies (e.g., wound cultures, ultrasonography, or computed tomography of the puncture area) should be made.

# DIABETIC FOOT CARE

The diabetic patient is at special risk for serious cutaneous injury and infection as a result of microcirculatory insufficiency and neuropathic changes. The diabetic often does not appreciate the development of pathologic conditions such as hyperkeratotic lesions, corns, ingrown toenails, and ulcers. Often these conditions are exacerbated by the patient's choice of footwear and activities.

**Therapy**

In general, the diabetic patient with foot lesions is instructed not to stand for longer than 20 minutes at a time. The patient is also cautioned to avoid extreme temperatures when cleansing the feet.

A hole or slit may be cut in the shoe over any obvious non-weight-bearing prominence. Hyperkeratotic lesions should be shaved and shielded with an aperture pad (see Fig. 54-9) (Figure Not Available) . For a hammertoe lesion, the toe is covered with an aperture pad. For the bunion area, a large commercial adhesive bunion pad or bunion shield cut from felt may be applied.

Ingrown toenails and mycotic toenails can be managed as for nondiabetic patients. The only caveat is that these conditions can become serious lesions before the patient is aware of the problem. The diabetic with advanced disease who has an infected or progressive lesion also should have differential blood pressures checked in all four extremities to assess large artery patency (see Chapter 70) . Evidence of large artery insufficiency requires urgent vascular referral. The diabetic patient's metabolic status also should be closely monitored.

When ulcers develop, the necrotic rim should be debrided. Antibiotic ointments or biologic membranes as used for burn patients (see Chapter 41) are also helpful. These lesions should be shielded with aperture pads, and the patient should be followed closely. A radical reduction of walking, perhaps to total non-weight-bearing status, should be stressed for *plantar* ulcerations. A pillow placed under the patient's calf that allows the heel to overhang permits heel ulcers to repair (Fig. 54-19) (Figure Not Available) . A foot cradle keeps bedding pressure off the toes.

# MANAGEMENT OF FUNGUS TOENAILS (ONYCHOMYCOSIS)

The mycotic toenail can occur at any age but increases in frequency with increasing age. The condition is caused by the usual tineal fungi, most frequently members of the *Trichophyton* genus. Onychomycosis can also occur posttraumatically and is more often seen in the hallux and fifth toenails, where the greatest shoe friction occurs. Spontaneous resolution is rare and is less likely as the nail plate becomes more extensively involved and thickened. Occasionally a traumatic avulsion leaves an

individual free of the hypertrophic mycotic toenail temporarily, but the matrix retains its acquired predilection for growing a hypertrophied toenail. A new tinea subsequently colonizes the nail in most cases.

The tinea is contained in the nail plate, and the body usually maintains the integrity of the viable dermis of the nailbed (Fig. 54-20) (Figure Not Available) . The fungus feeds on the nail plate and deposits its waste products within the nail plate and on the nailbed. The dusty, brownish-yellow debris encountered in the mycotic nail consists of fungus spores and hyphae, desiccated toenail, and desquamated skin from above the nailbed. During the initial stages of the fungus infection, only the free edge or the surface of the nail plate is involved. As time proceeds, the fungus extends proximally and more deeply into the nail plate along longitudinal lines of stress. Once the entire free edge of the toenail is mycotic, the fungus works its way back to the central nailbed in a series of striations (see Fig. 54-20) (Figure Not Available) .

Any mechanical or shearing force causes microtrauma to the invaginated nailbed folds, and a resultant lysis of the nailbed-nail plate interface occurs. A local, transient, primary fungal infection of the nailbed may be followed by a secondary bacterial infection. Eventually the fungus reaches the nail matrix, which responds by growing a hypertrophic nail plate. The mycotic nail subsequently causes greater friction or pressure on the nailbed, and recurrent inflammation and infection may occur.

**Therapy**

When the fungal infection is limited to the distal nail, simple surface nail plate filing with topical anti-tinea therapy is often effective. When the involvement is more extensive, avulsion of the nail plate is performed, and an oral antifungal agent is prescribed for 1 month, along with local wound care. Topical antifungal therapy is applied twice daily as the new nail plate develops. Therapy for secondary bacterial infections should incorporate broad-spectrum antibiotics (e.g., second-generation cephalosporin agents).

Once the fungus reaches the level of the nail matrix as visualized by obliteration of the lunula, the hypertrophy becomes fixed, and the condition is usually irreversible. Surgical approaches to treatment of chronic onychomycosis include nail plate avulsion with nailbed debridement (with or without phenol nail matrix destruction, described later) and periodic limited debridement of the involved nail using a Dremel tool (Gill Podiatry Supply Company, Middleburg Heights, Ohio) and nail burs (Fig. 54-21)

## INGROWN TOENAIL (ONYCHOCRYPTOSIS)

The ingrown toenail represents a progressive nail curvature or static widening of the lateral sides of the toenail such that the nail impinges on the skin of the nail groove. Normally, the cuticle and periungual nail structures undergo a progressive maturation and sloughing process that permits the nail to slide forward easily as it grows anteriorly from the nail matrix. Additional frictional forces are present in the nail groove when the nail is severely incurvated or hypertrophic, when the patient habitually wears tight shoes, or when the patient has a digit that is axially rotated against an adjacent digit or

the bottom of the shoe. These frictional forces may stimulate a hyperkeratotic response in the nail groove.

The vast majority of ingrown toenails occur in the halluces. Discomfort occurs when the nail impingement causes hyperkeratosis of the nail groove with localized nerve irritation or when a foreign body reaction is generated. The foreign body response may be simply a mild erythema and edema of the nail fold, or it may progress to a purulent granulomatous formation. If the patient cuts the nails in a curved rather than transverse manner, there is also the potential for a spicule of nail to remain in the nail fold and impinge upon the hyperkeratosis (Fig. 54-22) (Figure Not Available) .

**Evaluation**

In the presence of predisposing nail changes, the decision to treat is primarily based on the subjective complaints of the patient. The offending margin in the nongranulomatous ingrown toenail must be examined closely for determination of the exact location of the problem. With this condition, the patient will complain of pain reproduced by fingertip pressure over the nail groove. The clinician also should apply pressure to the central portion of the nail plate, the proximal cuticle, and the opposite nail margin to rule out other potential sources of pain. Other causes for discomfort include a stress fracture of the distal phalanx and a subungual exostosis, a proximal bony bump on the dorsum of the distal phalanx that can produce central nailbed pain. Other considerations producing referred pain include diabetic neuropathy and shoe pressure-induced neuritis of the dorsal medial cutaneous nerve.

The clinician should continue diagnosis and treatment by cleaning the nail groove with an alcohol pad. Hyperkeratosis, mycotic debris, and impacted periungual debris can be visualized with this technique. The nail groove should be inspected with a mini-curette. The free edge of the nail should have a clear path to exit the nail groove. The clinician should curette the extraneous periungual debris with the mini-curette until the nail and nail groove are separate and visible. This debridement may be facilitated by a 5-minute topical application of a 1% phenol solution (made by adding distilled water to a 70% to 88% aqueous phenol solution in an 80:1 ratio [e.g., 80 mL distilled water to 1 mL of phenol]). The physician must be careful not to put a great amount of pressure directly on the nail groove with the sharp edge of the curette, because the area is quite sensitive. The blunt curve of the back of the mini-curette can be used for gentle probing of the nail groove to locate the hyperkeratotic plug. This plug will elicit tenderness and will be visible as yellowish to gray translucent tissue after being cleansed with alcohol.

Further treatment will generally require a digital block (see Chapter 32) . At this point, the clinician's options are (1) temporary removal of the presumed nail spicule and/or debridement of the periungual hyperkeratotic plug or (2) permanent correction of the toenail deformity using the phenol technique (see below). The decision is primarily based on the chronicity of the complaint, the degree of nail curvature, the subjective discomfort of the patient (without a concurrent desensitizing neuropathy), and the history of infection or current infection. Because this procedure can be time consuming, the clinician also will need to factor concurrent patient care demands in the emergency

department.

**Removal of Nail Spicule and Debridement of Hyperkeratosis**

This procedure is primarily reserved for those ingrown toenails with minimal inflammation, a small amount of incurvation, no concurrent infection, and limited discomfort. Spicule removal can also be used where the nail is loosely adherent to the nail groove and can be freed without causing any capillary bleeding.

Following a digital block, the toe is cleansed with a standard skin preparation. An oblique portion of the offending nail (about one third to two thirds of the way back to the posterior nail fold) is removed (Fig. 54-23) (Figure Not Available) . Ideally, an English anvil nail splitter or nail forceps is used (Fig. 54-24) (Figure Not Available) . If the nail is found to be spiculated more than two thirds of the way back, the clinician can switch to the permanent phenol correction technique described below.

Following removal of the oblique nail portion, the free edge of the remaining nail should be smoothed so that it will glide freely out of the nail groove as it grows. Loose keratinized debris exposed beneath the removed side of the nail plate can be curetted off the nailbed. Next, the keratotic plug in the nail groove is debrided (preferably with a mini-Beaver handle and blade No. 64 or 67). The blade should be sharp to facilitate debridement and reduce the risk of trauma and resultant scarring of the nail groove. The nondominant hand should firmly grasp the toe while the clinician's thumb or index finger plantarly rotates the nail groove open. The keratotic plug is debrided down to normal epidermis or dermis, which may be ecchymotic in character. The clinician should attempt to prevent patient bleeding during the procedure. Should bleeding occur, a digital tourniquet (see Chapter 36) or a commercial Tourni-cot (see below) can be used for maintaining a dry field during the procedure. The area is then dressed with antibiotic ointment, Adaptic (or similar nonadherent material), and a dry dressing. A dressing change should occur at 48 hours with nail fold reevaluation. Follow-up podiatric care with consideration for permanent correction should be advised.

**Technique of Partial Toenail Removal**

The decision to permanently remove a portion of a toenail is based on the following criteria. Ingrown toenails associated with chronic inflammation, severe pain, or infection (with the exception of simple paronychia [i.e., paronychia without a hyperkeratotic reaction]) should be considered for nail removal. Phenol is applied to the exposed nailbed to produce neurolysis of nerve endings and necrosis of the germinal matrix.

After a digital block on the involved hallux or toe (see Chapter 32) , the digit is squeezed or wrapped for exsanguination of the blood currently in the toe, and a digital tourniquet is applied (see Chapter 36) . One commercial tourniquet is the Tourni-cot (Mar-Med Company, Grand Rapids, Mich), which both exsanguinates as it is applied and provides excellent hemostasis. Hemostasis is mandatory for subsequent phenol application. The device is rolled onto the digit from the tip to the base of the toe. It is wise to leave the device's warning tag in place during surgery as a reminder that the device must be

removed at the completion of the procedure and prior to release of the patient.

While stabilizing the toe in the nondominant hand, one uses English nail nippers to clip the involved lateral quarter of the nail approximately one half to two thirds of the way back to the cuticle, following the longitudinal lines of the nail (Fig. 54-25) . Care must be used to avoid cutting or tearing the nail further because of the danger of phenol seepage onto the desired remaining portion of nail matrix. The clinician proceeds by splitting the nail the rest of the way back with a mini-Beaver No. 61 blade; a No. 11 or 15 scalpel blade on a handle can also be used but is less effective. An angulated nail splitter (or straight scissors, although these are less desirable) may be used for thicker toenails. The end of the chosen instrument is placed in the palm of the clinician's pronated dominant hand for stability. The blade is placed into the formerly created nail defect. The index finger of the dominant hand guides the blade as the palm forces it into the defect. Any residual blood pooled in the toe now appears in the wound; the blood should be removed by daubing with dried gauze. If minor hemorrhage persists, the toe tourniquet should be reapplied and tightened. The index finger of the dominant hand should press against the patient's toe or the clinician's opposite index finger to provide a stabilizing counterpressure as the blade is slowly forced into the nail and beneath the cuticle. One should gently rock the splitter to ease it proximally. The nail is cut until the sensation of resistance is gone after passing several millimeters beneath the posterior nail fold. Care must be taken to keep the blade directed slightly downward and perpendicular to the nail plate, or the cut may angle obliquely into the corner of the posterior nail fold. If this happens, a portion of toenail may remain directly over the unwanted matrix, thus blocking the phenol cautery and reducing the correction.

Once the nail is split, a blunt angulated nail splitter is then used to separate the unwanted toenail from the nail groove, nailbed, and posterior nail fold. The nail remnant is then grasped with a small straight hemostat and removed from the wound by rolling (twisting) the hemostat overhand toward the remaining nail plate. The clinician inspects the remnant to ensure that all of the desired nail is removed. The exposed nail groove is then inspected for the keratotic plug; the lateral nail groove is curetted to remove the plug. The cuticle that is tucked back under the exposed posterior nail fold also should be curetted with counterpressure on the skin above; this tissue may not initially yield to curettement until it has been exposed to phenol lysis, but removal should be attempted before dressing the operative site to prevent a possible foreign body reaction postoperatively. Some clinicians prefer to debride the lateral nail matrix prior to phenol treatment.

The wound must be freed of hemorrhage or pooled blood by daubing the area with gauze or a dry cotton-tipped applicator. Again, if even minor hemorrhage persists, the toe tourniquet must be tightened. When using a Tourni-Cot, a smaller diameter device may be required if seepage persists.

The wound is now ready for phenol cautery. A 70% to 88% aqueous phenol solution is applied by means of cotton-tipped applicators. If the cotton-tipped applicators are too bulky to fit into the nail defect, they should be debulked prior to use. Microtipped applicators also are commercially available (Gill Podiatry Supply Company, Middleburg Heights, Ohio). The applicators should be thoroughly moistened, but not saturated, with

phenol solution, and one should make three 30-second applications to the nail groove, concentrating on the matrix beneath the posterior nail fold. For small toes in children, a reduced dosage of three 20-second applications by means of a cotton wisp on a toothpick should be used. The applicator is twisted toward the top of the nail--that is, when facing the tip of the toe, the applicator is turned counterclockwise if the remaining nail is to the left of the applicator and clockwise if the remaining nail is to the right of the applicator (Fig. 54-25 *D*). One uses this technique to avoid forcing phenol under the remaining nail plate.

The chemical cautery should turn the nail groove tissue

to a brown-tinged grayish color. If fluid should drip from the wound or migrate along the posterior nail fold, it must be dabbed quickly with gauze to prevent it from lysing normal tissue. Upon completion of the third phenol application, the wound is lavaged with a small stream of sterile water. While the stream is flowing over the defect, the clinician rubs his or her latex-gloved finger over the wound until a greasy feeling is replaced by a squeaky friction sensation, indicating removal of the phenol. There should be no gross loss of nail groove tissue or matrix observed after the cautery; the tissue will merely appear gray and dry.

After chemical treatment, the area is inspected for any remaining cauterized cuticle or dead skin, which is snipped away with tissue scissors. The wound is dressed with an antibiotic ointment, Adaptic (or other nonadherent dressing), small gauze pads, and tape or Kling wrap. If a Tourni-cot or other tourniquet has been applied, it must be removed before or immediately after application of a dressing. The Tourni-cot device is released by cutting its narrowest groove. Some clinicians prefer to remove the tourniquet prior to application of a dressing, thus avoiding the potential for a tourniquet to be left in place under a dressing. Removing the tourniquet immediately after application of a dressing permits the dressing to be placed on a bloodless field. When significant underlying infection is present, a wound check is appropriate in 24 to 48 hours.

Postoperative care includes twice-daily scrubs, antibiotic ointment, and dry dressing changes for several weeks. Oral antibiotics generally are not required unless the patient is immunocompromised. The early wound will display red, moist granulation tissue. Wound drainage is often present for the first 2 weeks and appears clear and straw colored. A dry scab will remain in the wound of the uneventful phenol correction for 1 month and may need to be debrided if there is a complaint of tenderness persisting in the former ingrown corner of the nail fold.

This surgery is approximately 90% to 95% successful in preventing lateral nail regrowth after partial removal. In addition to nail regrowth, infection and postsurgical foreign body reaction to remaining cuticle or nail may occur. An inclusion cyst may also form around the nail remnants, and curettement of the nail groove and posterior nail fold may be necessary.

**Total Toenail Removal**

The entire nail is sometimes removed when there is extensive infection with medial and lateral symptoms. If infection has been present for >1 month, osteomyelitis should be considered and digit radiographs obtained. Periosteal abnormalities limited to the affected digit suggest concurrent osteomyelitis and warrant consultation with an orthopedist or podiatrist. The presence of osteomyelitis is not a contraindication to nail removal when removal is otherwise indicated. The patient should be warned that osteomyelitis is possible despite normal radiographs, especially if the toe still has a generalized ache or swelling after the nail has healed. With significant evidence of infection or in an immunocompromised patient, a wound culture should be obtained from the nail fold after nail removal and a sensitivity test performed.

The digit is prepared in a fashion similar to that used for partial nail removal. The Tourni-cot or digital tourniquet is applied. Rather than splitting the nail, the open arm of a small hemostat is placed under the nail with the toothed surface upward toward the nail. The hemostat is pushed straight back toward the proximal nail fold while elevating pressure is kept on the arm of the hemostat to avoid tearing the nail bed as the instrument is forced proximally. The hemostat is similarly inserted in bordering zones beneath the nail plate until the toenail is completely loosened from side to side. An alternative method is to insert a small straight hemostat sideways beneath the central nail plate and open the hemostat repeatedly. After the nail is removed, all debris is curetted, and the entire nail bed is treated with phenol as outlined above, concentrating on debriding the area beneath the posterior nail fold. The phenol solution is flushed from the wound after the third treatment, as previously discussed. Postoperative care and complications are similar to those for partial nail removal.

# Chapter 55 - Trigger Point Therapy

**Anders E. Sola**

Myofascial pain is probably the most common pain problem faced by physicians. [1] It may be the primary complaint or a crippling adjunct to other problems, and it is often accompanied by local or generalized fatigue. Unless it is properly diagnosed and treated, persistent symptoms haunt patients and often result in misdiagnoses, unnecessary pain, and debilitation.

Treatment of myofascial pain is an effective way of sorting out pain problems. Elimination or reduction of myofascial pain often unmasks other painful disorders and therefore is useful diagnostically. Myofascial pain is a common complaint in the emergency department, and affected patients often present with torticollis, headache, or lower back pain. Highly localized, exquisitely sensitive areas, usually found within or near the painful region, are recognized as a common feature of myofascial pain. Pressure on these areas, known as *trigger points* (TPs), causes local pain, referred pain, or both.

The local or referred pain associated with TPs may not follow segmental neurologic patterns and therefore may not occur with a well-defined dermatome distribution. Actions taken at TPs, such as application of heat or cold, electrical stimulation, injection with local anesthetic or saline, or simply stimulation of the sensitive point with a needle, have proved that TPs are, in many cases, the key to control of the painful experience. [5] A somewhat similar needling effect is associated with acupuncture points (many of which are located in areas in which TPs are commonly found). The exact mechanisms by which TPs are involved in and contribute to myofascial pain, as well as the response to intervention at TPs, indicate a continuous, cyclic relationship between TP activity and the pain phenomenon.

## BACKGROUND

### Mechanism

The TP phenomenon has its western origin in Germany, the Scandinavian countries, and Great Britain (the "rheumatic" countries). [10] [11] The Germans first reported on these painful muscular problems ("Muskel schmerzen") and "hard muscles" in the mid-19th century. Most of the investigations were directed at the microscopic findings of these hard nodules, or so-called *rheumatic lesions of muscle.* These sensitive areas in muscle caused local pain or tenderness on palpation with or without a predictable pain reference away from the local point of maximal tenderness. The British were unable to identify faulty microanatomy or pathophysiology with these elusive pain problems, commonly referred to as *fibrositis, myalgia,* or *nonarticular rheumatism.* Gower introduced the term *fibrositis,* [12] and it became fixed in the English literature when Llewellyn and Jones's massive text was published in 1915.

Travell described TPs caused by acute or chronic overload of a muscle as a palpable, firm, tense band in the muscle. The TP is characterized by a local twitch response to tapping of the muscle, restricted range of motion, weakness without atrophy, and no sensory deficit. Subjectively, patients complain of pain in a predictable pattern, stiffness and fatigue, and deep tenderness at the TP. [14]

In addition to muscular overload, a variety of stress-inducing stimuli--emotional or physical--may be implicated in the onset of myofascial pain (Fig. 55-1) (Figure Not Available) . The power of these stressors to induce pain in a particular individual is moderated by the genetics, personality, conditioning, and physiologic state of that individual. Recurrent injuries in a specific location may sensitize the tissue to development of a TP and discomfort out of proportion to the most recent insult. [15] This "injury pool" concept is consistent with current theories of pathophysiologic pain.  Once established, however, a painful event may sustain itself in spite of control or elimination of the initiating stimuli through a characteristic internal cyclic process of self-stimulus and response. Furthermore, the painful TP itself may become the stressor that involves other muscles in the event. Thus, TPs may act both as translators of stress to pain and, secondarily, as stressors that perpetuate pain.

While the exact physiologic mechanism of TPs is unknown, TPs may be "weak points" within the muscle or fascia that are particularly sensitive to stress-induced change. In the absence of stress, the TP may remain quiescent, only to become activated by a number of positive-feedback cycles that involve sensory motor reflexes, autonomic responses, vascular changes, and numerous other ill-defined events that ultimately lead to muscle tension, fatigue, and pain. [14] Hence, the resultant pain can be more intense, out of proportion, or of a longer duration than anticipated for the recent injury.

Both myofascial pain syndromes and fibromyalgia syndrome can be associated with painful TPs. While fibromyalgia is considered a form of nonarticular rheumatism and is associated with diffuse musculoskeletal involvement (multiple bilateral TPs), the myofascial pain syndrome is considered a regional pain syndrome associated with TPs. [20] While these conditions overlap somewhat and clinicians debate their definitions, most agree that TPs of recent onset associated with unilateral tenderness or referred pain are most amenable to therapy.

**Distribution**

A TP can occur in any muscle or muscle group in the body. Since the stresses involved in the onset of myofascial pain can commonly affect not only single muscles, but also entire muscle groups, TPs tend to cluster. In the upper trunk, a common TP cluster involves the muscles of the neck and the shoulder area--the trapezius, the levator scapulae, and the infraspinatus. In the lower trunk, an often-involved group of muscles consists of the quadratus lumborum, the gluteus medius, and the tensor fascia lata. As individuals age, there is an increase in the potential for nerve root irritation problems and subsequent pain, as suggested by Gunn.  Although nerve root irritation cannot be resolved by treatment of TPs, myofascial disturbances that may arise as a result of the

irritation may be reduced through such treatment. [22]

Painful TPs in a given muscle often affect all other muscles innervated from the same spinal segments. Therefore, the search for TPs and subsequent treatment is usually directed at those muscles innervated by *both* the *posterior* branch of the spinal nerves and the *anterior* spinal branch (Fig. 55-2) . Pain may be felt in a muscle innervated by an anterior spinal nerve, whereas little or no pain may be initially felt in the erector spinae muscles innervated by the posterior branch of the same spinal nerves. Palpation of the erector spinae muscles, however, may reveal evidence of active TPs: hypersensitive areas or, more importantly, a tight band-like local spasm plus hypersensitivity.

Although TP pain can be bilateral, it is frequently seen on 1 side. Besides pain in the muscles of the anterior and posterior branches of a specific segmental spinal nerve, there may be pain in the common musculature for several spinal segments above and below innervation of the major TP and in even more remote muscle groups. Thus, muscles in the erector group in the cervical area can be the source of noxious impulses that give rise to headaches (including migraine attacks) and can cause pain to be transmitted as far as the midthoracic area.

Common TPs abound in the lower trunk, gluteal muscles, abductors and adductors of the thigh. Their presence in the gluteal or lumbar muscles may often be related to hypersensitivity in the muscles of the upper trunk group. Therefore, a patient who is suffering muscle tension headaches on 1 side should be checked not only for TPs in the ipsilateral upper trunk muscles, but also for hypersensitive areas (TPs) in the gluteal and lumbar musculature (Fig. 55-3) . If TPs are found, they should be treated to reduce both the hyperactivity in the lower segment and the sensitivity of the upper muscle group. If hyperactive TPs are ignored in the lumbar and (especially) the gluteal region, treating only the cervical TPs can precipitate a painful low back

spasm. This may be severe enough to cause another trip to the emergency department.

**Therapy**

In 1937 Kraus published an innovative study on the use of ethyl chloride spray to relieve myofascial pain. [24] Travell and Rinzell subsequently identified common pain patterns and emphasized the use of procaine injections and the coolant spray Flouri-Methane (Gebaur Chemical Company, Cleveland, Ohio), instead of the more dangerous ethyl chloride.  Jaeger and Reeves quantitated clinical improvement with the "stretch and spray" technique. [26]

Travell and Rinzler also demonstrated that dry needling of the TP could bring relief. Sola and Williams discovered that normal saline injections could relieve myofacial pain. [27] In fact, Kibler found little difference in effect between type of anesthetic or amount and concentration of anesthetic injected into TPs. [28] Frost and colleagues, in a double-blind comparison of local anesthetic (mepivacaine) and saline injections, found that saline produced more consistent pain relief of longer duration. [29] However, Hameroff and colleagues found bupivacaine and etidocaine to be superior to saline

injection.  Interestingly, the immediate analgesia produced after TP injection can be reversed with IV naloxone.

Lewit postulated that injection therapy is most effective when an intense pain is produced at the exact site of maximal tenderness. [32] While sounding like torture, this brief "shot of pain" reduces TP pain in >80% of cases and provides persistent relief for months or even permanently in about 50% of cases.

## PATIENT PROFILE

All parts of the musculoskeletal system are susceptible to myofascial pain syndromes. The patient presents with localized or diffuse pain ranging from intense, burning, and debilitating to dull and bothersome. The affected areas are often described as stiff, heavy, or numb. The pain frequently affects a joint, and range of motion may be inhibited.

Many patients attribute their symptoms to overuse of muscles, sitting in drafts, or a "cold" in the muscle. Physical examination fails to elicit any sensory deficits or atrophy, although there may be some weakness of involved muscle groups. Thorough examination of the affected areas may reveal edema, coolness in an affected extremity, or exaggerated pilomotor reflexes.

In sedentary populations, TPs are common and are usually associated with chronic strain and stress. They tend to occur with great regularity in the same anatomic areas. In my clinical experience, TPs are less common in laborers and athletes and, when present, are usually the result of overuse or injury rather than chronic strain or stress. This suggests that regular exercise is of therapeutic value in prevention, as well as treatment, of myofascial syndromes, which start at an early age. Conversely, the presence of TPs inhibits the effectiveness of a well-designed exercise program.

Patients with prolonged or complicated myofascial pain syndromes may present to the emergency department after unsuccessful attempts to achieve relief from their symptoms. They may have consulted numerous physicians, chiropractors, or both; tried home remedies or vitamin therapy; or received treatment with a variety of muscle relaxants, anti-inflammatory medications, or potent analgesics. Their symptoms may have been diagnosed vaguely as overuse injuries, chronic bursitis, arthritis, or sciatica, or they may have been dismissed because there were no radiographic or laboratory confirmations of their complaints. Many patients labeled as "chronic complainers" have myofascial syndromes that, when treated, result in reduced symptomatology.

Younger patients are more responsive to TP injection than older individuals. In younger patients, TPs are more easily located and are less complicated in their pain referral patterns. As people age, muscle weakness and shortening and the effects of degenerative processes frequently confuse the pain referral pattern. Furthermore, patients seen in pain clinics tend to have more associated depression and less frequently have a medical explanation for their pain than patients seen in private

practice.

# EXAMINATION FOR TRIGGER POINTS

Any treatment of myofascial syndromes must be preceded by a history and physical examination to rule out other causes of apparent myofascial pain. Once this is accomplished, a systematic search for TPs is carried out, with special emphasis placed on the painful muscles and their segmental associates.

Suspected TP areas should be compared with their contralateral counterparts as a guide to their relative sensitivity. There are 4 general patterns of TPs [15] :

1. A single TP with minimal sympathetic involvement.
2. Clusters of TPs within a muscle group with segmental involvement (e.g., TPs isolated to cervical muscles).
3. Combined upper and lower trunk syndrome (e.g., ipsilateral TPs in both cervical and lumbar muscle groups).
4. Diffuse bilateral TPs--usually with extensive involvement of hyperanalgesic paraspinal skeletal muscle tissue. This pattern is often associated with "abnormal" or neuropathic pain. [22]

The most reliable method of locating TPs is by searching in the painful area with the tip of the finger. Pressure applied to the hypersensitive area in the muscle reproduces or accentuates the pain. This is usually accompanied by an involuntary "wince" by the patient. The hypersensitive area may feel rope-like, indurated, or tight, depending on which muscle is examined. The muscles should be examined in both relaxed and stretched positions. Although there may be a halo area or a surrounding zone of tenderness, one should search for the area of maximal tenderness or response.

It is helpful to mark the point of maximal tenderness with a pen and then repeat the search, varying the palpation. Variability in the site of maximal tenderness speaks against a true TP. If several areas of exquisite tenderness are found, it is important to treat the most sensitive point first.

Although the pain referral pattern may help to isolate the hypersensitive area, one should remember that the pattern differs according to severity and longevity of the TP injury, body build, state of health, gender, and degree of injury or weakness.

When searching for TPs, one should carefully examine the entire area of all muscles that may be involved. For

example, in treating torticollis, one should check the levator scapulae, trapezius, sternocleidomastoid, and posterior strap muscles (supplied by the posterior spinal ramus), especially the splenius and semispinalis muscles. The muscles are often innervated from many vertebral segments--for instance, innervation of the splenius starts at the C2 vertebral level and reaches to the midthoracic level. Thus, hypersensitivity at one level may readily involve other muscles with overlapping

segmental innervation. When treating neck pain problems, one must look for tender TPs along the entire spinal insertion of these muscles. The same principle of extended search is also important in the treatment of myofascial shoulder pain and headache pain. A common finding is an ipsilateral pattern involving cervical shoulder, lumbar, and gluteal muscles. The patient frequently complains of symptoms on 1 entire side of the body. When headache or neck pain is severe, often the patient is aware of stiffness, discomfort, or pain in the hip area.

## INDICATIONS AND CONTRAINDICATIONS

Local treatment of sensitive TPs is often quite effective for pain relief when the TP is the primary source of myofascial pain. Such treatment may reduce pain and speed recovery at sites of trauma in the same segment. Treatment of TPs that result from specific painful stimuli, such as nerve root lesions and nerve compression, is never more than moderately successful in relieving pain. Nonetheless, it is often difficult to ascertain the source of the stress that leads to the myofascial involvement until TP therapy has been tried. Therefore, in many cases the response to TP therapy may be valuable diagnostically.

The use of a vapocoolant for "stretch and spray" is contraindicated in the setting of cold hypersensitivity. Contraindications to injection treatment include the presence of a systemic illness (especially with fever), bleeding diathesis or anticoagulant therapy, cellulitis overlying the area of planned injection, high anxiety or emotional stress levels (including "needle" anxiety, manifestations of psychosis, and abuse of drugs or alcohol), and a history of hypersensitive or syncopal reactions to injections. If there is significant doubt concerning the diagnosis of myofascial pain caused by TP involvement, as opposed to such possible causes as a nerve root lesion, one should postpone injection therapy until other causes have been ruled out. Finally, injection of TPs should be avoided when malingering is suspected. Although the possibility of injection treatment may discourage some malingerers, it is less effective as a screening tool than acute observation of the "too-perfect" account of symptoms often given by such patients.

## EQUIPMENT

TPs may be treated with needling, injection therapy, or "stretch and spray" techniques. Little equipment is necessary for the injection procedure. A simple tray may contain a 5-mL syringe, a 25- or 27-ga needle, an antiseptic preparation, and a local anesthetic (1% lidocaine, 0.25% bupivacaine, or 1% procaine hydrochloride) or physiologic saline for injection. Some physicians use a repository corticosteroid preparation (e.g., 0.1 to 0.2 mg dexamethasone per 10 mL saline solution). [23] The stretch-and-spray technique uses a vapocoolant. Ethyl chloride (a volatile anesthetic agent) and fluorimethane spray have been used for this purpose.

## GENERAL PROCEDURE

**Injection Therapy**

Injection of the hypersensitive TP often provides dramatic pain relief. A body of evidence indicates that at least a portion of the benefit is derived from needle stimulation of the TP, regardless of the substance injected. [32] Although local anesthetics are often used, I have used normal physiologic saline for many years, with good results. [27]

Patients with marked pain and mild to moderate anxiety may benefit from an oral anxiolytic 30 to 60 minutes prior to therapy (e.g., 5 mg diazepam). When injection therapy is performed in the emergency department in patients with moderate to severe pain, the supplemental use of a local anesthetic may provide more immediate relief, although good results have been obtained, even in cases of severe pain, with saline injection alone. Some clinicians routinely inject small doses of a long-acting steroid preparation into painful TPs in conjunction with saline or a local anesthetic. Although steroids are of proven value in the treatment of inflammatory conditions, their role in TP therapy is unknown.

Prior to TP injection, the therapeutic plan and rationale should be explained to the patient. In particular, it is important to explain why the TP, rather than any site of pain referral, will be treated. It may be necessary to apply pressure over the TP to reinforce the need to treat the TP. It is wise to perform injection with the patient placed in a supine position to minimize vasomotor syncopal reactions. If the upper back, neck, or shoulder is being injected, a pillow is placed under the hips to rotate the trunk.

The hypersensitive point is located by palpation (with the affected muscle relaxed), and the area for injection is prepared in the usual manner. The point of entry should be in the area of maximal tenderness. One inserts the needle and aspirates to confirm a nonvascular site. If severe cramping pain or paresthesia occurs, suggesting nerve penetration, the needle should be repositioned. After proper positioning, inject 0.5 to 2 mL of fluid using a "fanning" technique, in which the needle is repeatedly withdrawn part of the way and redirected (Fig. 55-4) . This ensures maximal coverage of the area of the TP. Each TP is treated in this fashion. Generally, no more than 20 mL of saline total (for all sites combined) should be injected per day. Response to the injection should be observed within 10 minutes.

In most cases a short series of 2 to 5 injections of symptomatic TPs over a period of days to weeks is enough to treat myofascial pain caused by muscle strain or injury. After injection, a warm moist pack applied to the area relieves the temporary discomfort of the injection.

Follow-up treatment for patients with myofascial pain may include 1 or more of the following: repeated injection therapy; adjunctive physical therapy; and other special techniques, including intermittent cervical or lumbar traction, relaxation techniques, ultrasound, electrical stimulation, massage (especially deep friction), application of coolant sprays or ice packs, and, routinely, therapeutic exercise. Should the patient not respond to a brief course of TP therapy, suspicion for other structural phenomena should increase.

**Stretch-and-Spray Therapy**

The stretch-and-spray therapy technique [5] [34] seems most helpful for mild cases of myofascial pain syndromes. The patient is first informed of the therapeutic actions to be undertaken and informed as to why the therapy is directed to the TP rather than any pain referral zone. This can be reinforced by applying pressure to the TP. The expectation of some rebound pain exacerbation following treatment (see above) also can be discussed at this time.

The patient is placed in a comfortable position with the affected anatomy supported and the involved muscles relaxed. The vapocoolant is sprayed on the skin overlying the affected muscle TP, using a fine spray with the bottle approximately 45 cm from the skin. The spray is directed at an angle of 30° onto the skin overlying the TP. The spray is moved (in 1 direction) toward the zone to which any referred pain was previously noted. After a few initial sweeps of the spray over the referral zone, a warm moist compress is applied over the sprayed area to help relax the muscle. The affected muscle is then passively stretched with enough force to elicit pain and discomfort, followed by continued stretch with concurrent spray (Fig. 55-5) (Figure Not Available) .

As the spray is continuously applied over the muscle, progressive passive stretch is applied to the muscle. The spray is applied repeatedly in 1 direction, moving from the TP to the referral zone using slow, even, parallel sweeps over the entire muscle at a rate of about 10 cm/sec. This process is repeated 4 times, avoiding freezing of the overlying skin. After therapy, the patient is asked to perform a few sets of 5 repetitions demonstrating active range of motion with the treated area. The patient is given instructions for stretching and active range-of-motion exercises at home. Moist heat applications prior to these home activities may be helpful for some patients.

## COMPLICATIONS

Some patients are hyperreactors. These individuals may experience a vasomotor response with even a small injection of saline and may have an excessive flare-up of pain after treatment (generally within 24 hours, most commonly within 6 hours). Seated patients are more likely to faint than those kept supine. Those patients with an allergy history (e.g., allergies to foods and drugs and sensitivity to the sun), fair-skinned redheads, albinos, or blue-eyed blondes are more likely to faint. I monitor patients during injection therapy using the back of my hand, touching their skin to detect excessive sweating.

If complications occur, one should stop injecting. Some physicians inject as much as 5 to 10 mL of fluid at a given TP. I have found that this volume is excessive. The patient frequently develops a flu-like syndrome, characterized by malaise and myalgias, the next day. I have experienced few complications using saline with a normal pH. The use of local anesthetics, a standard procedure in emergency departments, slightly increases the risk of "-caine" allergies. However, the risks of excessive drug administration and IV

injections also are increased when local anesthetics are injected.

Particular care must be used when injecting the neck, the intercostal muscles, and the periscapular area, because these areas contain many large vessels and nerves, and the pleura is in close proximity to the injection sites. This risk is greatest if the patient is agitated or confused. Generally, TP injection should be avoided in the latter situations.

Ethyl chloride vapocoolant spray is highly volatile. Electrical sparks and flames should be avoided during its use. The agent also has general anesthetic properties and should be used in a well-ventilated room to avoid patient or operator toxicity.

# EMERGENCY DEPARTMENT TREATMENT OF COMMON PRESENTATIONS OF MYOFASCIAL PAIN

### Shoulder Disorders

A painful shoulder will frequently have TPs located in the posterior scapular muscles. Other muscles that are often involved include the supraspinatus, the infraspinatus, and the pectoralis major. Less commonly, the teres, the deltoid, and the triceps are involved. Some TPs in the splenius, the semispinalis, and the gluteal muscles also may contribute to shoulder pain and should be treated if found in conjunction with myofascial shoulder pain. Such pain often prompts the misdiagnosis of "bursitis." Patients may have had numerous prior unsuccessful treatments with muscle relaxants and potent anti-inflammatory medications.

When injecting the scapular and periscapular muscles, one should take extreme care to stabilize the scapula. The patient should lie on a bed with the arms along the sides and a pillow under the chest to round the shoulders and facilitate injection. A small pillow may be used to rest the forehead. The scapular boundaries (borders) should be noted before injection, and the patient should be warned not to move the shoulder. If the lower portion of the scapula moves, one can easily miss the muscle and pierce the pleura. Therefore, one should "fix the scapula" before injecting and should always double-check the anatomy. I frequently hook my thumb on the medial border while injecting TPs in the infraspinatus muscle. Myofascial pain is commonly associated with a number of muscles at the medial border of the scapula. These include the rhomboids; the serratus anterior; the subscapular muscles; and, at the superior edge, the levator scapulae muscle. It is best to inject the levator at an oblique angle to the muscle (nearly parallel to the thorax), because patients frequently flinch during injection, and the needle could puncture the pleura.

If the TPs are on the lower medial border, then the patient should place the hand behind the back. This causes a "winging" of the scapula, allowing easier and safer injection. A tangential laterally directed needle reaches much of the scapular undersurface (Fig. 55-6) . The rhomboids and the serratus anterior are easily treated with this maneuver. On occasion, the subscapularis muscle is involved, and the only complaint besides poorly localized scapular pain may be a sensation of "sleeping on a marble." When injecting along the lateral border (teres major and minor and latissimus dorsi), the

clinician should fix the scapula and warn the patient not to move.

## Headaches

Commonly, TPs contribute the muscle component of most headache syndromes. These TPs are usually found in the sternocleidomastoid, levator scapulae, and trapezius muscles and, not infrequently, in the scalp and facial muscles. In addition, the posterior strap muscles are often involved, particularly the semispinalis and splenius muscles (Fig. 55-7) (Figure Not Available) . When preparing to treat a patient with headache, one should carefully examine the entire back for hypersensitive areas, paying special attention to the thoracic paraspinal muscle group. These thoracic TPs are frequently associated with migraine as well as muscle tension headaches. As previously discussed, TPs located in the quadratus lumborum and the gluteus medius also may contribute to headache problems. These muscles seem to have particular significance if the headache is unilateral. When found, they should be treated concurrently.

## Back Pain

Unilateral back pain is usually responsive to TP injection. TPs are most commonly found in the quadratus lumborum, gluteus medius, and tensor fascia lata muscles. Hip pain caused by gluteal TPs may mimic trochanteric bursitis. Sciatica, by far the most frequently diagnosed back pain syndrome, is commonly caused by gluteal TPs (Fig. 55-8) (Figure Not Available) . In addition, ischial and sacral iliac syndromes may be seen.

The lumbosacral muscles are commonly involved in lower back pain. Here the TPs are frequently secondary to vertebral or nerve root irritation or spondylosis, which can cause neuropathic pain. Treatment of TPs may be of only temporary benefit. However, deep paraspinal (lumbar/thoracic) and hamstring muscle TPs are often overlooked. Nonetheless, if no better after a short course of TP treatment, affected patients should be referred for further evaluation and treatment of possible neuropathic pain. Back pain patients have many options other than TP therapy that should be considered before surgery. [35]

## Torticollis

Torticollis usually is a simple problem involving 1 to 3 muscles. The trapezius, sternocleidomastoid, and levator scapulae are the main offenders. A careful search of the posterior strap muscles, however, may reveal exquisite tenderness of the splenius and semispinalis muscles. If the trapezius is involved, particular care must be taken with the injection technique (Fig. 55-9) (Figure Not Available) .

The apical pleura in some individuals is much higher than normal. If there is a sudden upward flinch of the shoulder during injection, the pleura could be punctured. If possible, one should inject transversely, with the TP located between index finger and thumb.

**Somatic Visceral Reflex Phenomenon**

Skeletal muscle TPs may contribute to visceral pain by induction or continuation of spinal reflex arcs. Visceral sympathetic afferents converge on the same dorsal horn neurons as somatic nociceptive afferents. Reflex sympathetic efferent activity may result in spasm of visceral sphincters as well as of cutaneous nociceptors (leading in part to referred cutaneous pain). The rectus abdominis muscle is particularly prone to TP development in conjunction with visceral pain. Right upper quadrant TPs can be seen with gall bladder disease; left upper quadrant TPs can be seen with esophageal and ulcer disease, right lower quadrant TPs can be seen with dysmenorrhea, and left lower quadrant TPs can be seen with intestinal disorders.

While a full discussion of this phenomenon is beyond this text, treatment of abdominal wall muscle TPs can provide significant somatic and visceral pain relief in appropriate patients. Therapy for abdominal wall TPs should be considered when they are identified and when TP palpation or placing the muscle under tension (e.g., performing sit-ups) produces characteristic visceral discomfort. Painful abdominal TPs also are accompanied by TPs in the posterior paraspinal muscle segment. These TPs must be treated along with the abdominal muscle TPs. Esophageal spasm is often associated with TPs on the left posterior thorax at levels T3 through T6.

Visceral discomfort that persists following TP therapy warrants further diagnostic evaluation. Somatic and visceral pain differ in the following aspects:

> Somatic pain tends to be well localized and generally follows the distribution of somatic nerves, whereas visceral pain is more diffuse.

> Somatic pain is sharp and definite, whereas visceral pain may be dull and difficult to describe.

> Somatic pain is reproduced by external stimuli, whereas visceral pain is not, but may have associated referred pain or symptoms (e.g., nausea, vomiting or diaphoresis).

## SPECIFIC MUSCLE SYNDROMES

Some of the muscles that are commonly involved in pain problems are listed in the following paragraphs. Although the pain referral sites are fairly consistent, they may differ because of involvement of more than 1 muscle. Figures 55-7 (Figure Not Available) through 55-16 (Figure Not Available) demonstrate common TP sites and common pain referral zones (inserts).

**Infraspinatus**

Because of its multiple functions, this muscle is subject to earlier degeneration than

other muscles of the rotator cuff and therefore is more vulnerable to TPs in association with many types of shoulder lesions. The TPs in the infraspinatus invariably cause sympathetic hyperactivity and may be a major contributor to dystrophy-like syndromes of the upper extremity (Fig. 55-10) (Figure Not Available) . Careful palpation is necessary to locate TPs. It is useful to search the entire muscle along the length of the muscle bundles as well as across the "grain" of the muscle. Related pain is usually located on the posterior and lateral aspect of the shoulder and occasionally may return to the anterior chest. All of the scapular muscles are frequently involved, either singly or in concert with each other.

## Levator Scapulae

Painful sensitive foci may occur at the origin on the superior medial aspect of the scapula, along the entire flat muscle belly, or on the insertions on the transverse processes of the first 4 cervical vertebrae (Fig. 55-11) (Figure Not Available) . Invariably, the levator scapulae muscle is involved in chronic cervical conditions as well as in torticollis. The pain is usually referred to the posterior cervical region, the posterior scalp, and the area around the ear.

## Quadratus Lumborum

This muscle is a hip "hiker" and a lateral flexor of the spine; in addition, it assists respiratory function by anchoring the 12th rib for the pull of the diaphragm. It frequently signals its distress on deep inspiration with 12th-rib pain. Pain can be local or referred to the anterior abdominal wall. These TPs may accentuate postoperative pain or painful abdominal scars over the lower quadrant. TPs occur on the 12th rib, on the iliac crest, and along the lateral border of the entire muscle (Fig. 55-12) (Figure Not Available) .

## Gluteus Medius

TPs in the gluteus medius may well be the most critical TPs in the lower extremity. Like the infraspinatus, the gluteus medius is associated with sympathetic hyperactivity. Activity of these TPs often involves activation of TPs in the quadratus lumborum, the tensor fascia lata, and the other gluteal muscles, thus inducing widespread lower back discomfort (see Figs. 55-8 (Figure Not Available) and 55-12) (Figure Not Available) . There also is an interaction between the TPs of the gluteus medius and those in the cervical area, sometimes involving this remote muscle in cervical pain and headache. Although this muscle seldom causes pain without involving other muscles, the pain pattern most often attributed to the gluteus medius is along the iliac crest and into the posterior thigh and calf. It is a frequent cause of hip pain in the later stages of pregnancy and simulates sciatica. TPs most commonly are found along the iliac shelf, and with extensive involvement, the entire gluteal ridge (including also the gluteus minimus and maximus muscles from the sacroiliac joint to the anterior superior spine) may contain painful TPs. It is estimated that 10% of people have legs that differ in length by at least 1 cm. This difference can cause unilateral back pain and TPs of the gluteus, erector spinae, and quadratus lumborum muscles.

### Tensor Fascia Lata

This muscle is easy to examine, and it is also easy to locate the common TPs that are present in the muscle belly. Pain is usually referred to the lateral aspects of the thigh as far as the knee (see Fig. 55-8) (Figure Not Available) .

### Anterior Tibialis

Pain in the anterior ankle is usually experienced when the TPs of this muscle flare up, although in severe cases the entire muscle may be painful. These TPs are most commonly found in the upper one third of the muscle, and pain is referred to the anterior portion of the leg and into the dorsal portion of the ankle (Fig. 55-13) (Figure Not Available) .

### Gastrocnemius/Soleus

Myofascial pain related to this muscle group is felt behind the knee, over the muscle bellies, and along the Achilles tendon near the heel. The TPs are usually found on the medial and lateral margins of the muscle group and along the midline of the group. These TPs often flare up when a patient is experiencing vascular problems of the lower extremities. One report has suggested injecting these TPs for the relief of pain associated with intermittent claudication. [39] The pain is referred to the Achilles tendon and the heel (Fig. 55-14) (Figure Not Available) .

### Splenius Capitis/Semispinalis Capitis

Pain resulting from involvement of these muscles may be located over the muscles themselves. Both the splenius and the semispinalis can mediate pain to the head and the face, however, and both are commonly involved in headache. Occasionally, dizziness accompanies involvement of these muscles. Because TPs are difficult to pinpoint in these muscles, patient cooperation in pointing out positions of maximal tenderness is extremely helpful (see Fig. 55-7) (Figure Not Available) .

### Rectus Abdominis

These muscles are frequent sites of anterior abdominal wall pain. The TPs are best located with the patient in the supine position and with the head and neck flexed so that the abdominal rectus muscles are under tension. These TPs frequently flare up after abdominal surgery and can be one of the chief constituents of postoperative pain. The TPs are most commonly found in the upper 3 segments of abdominal rectus muscle, and the pain is usually localized over the muscle (Fig. 55-15) (Figure Not Available) . The lower segments also are associated with low back problems, and the posterior spinal segments L4-L5-S1 also must be treated.

**Pectoralis Major/Pectoralis Minor**

The pectoralis major muscles are a frequent site of myofascial pain in the area of the muscle insertion on the anterior medial shoulder. The inferior belly of the muscle is a common area of TPs; however, the entire muscle must be searched diligently. The clavicular portion of this muscle usually refers pain to the uppermost part of the muscle. On occasion, there is some referral into the arm (see Figs. 55-15 (Figure Not Available) and 55-16) (Figure Not Available) .

**Intercostals**

One should routinely examine the intercostals for chest pain by palpating the intercostal spaces with the fingers. The intercostals are frequently involved after any chest surgery or trauma. In treating chest pain when intercostal blocks are not successful, one must take care on injection to avoid entry into the pleural space. Pain from the exterior intercostal muscle is usually localized near the site of the TP and is emphasized during inspiration (see Fig. 55-16) (Figure Not Available) .

**Trapezius**

The trapezius is a frequent source of muscle pain and headache, especially at the angle of the neck or at the occipital insertions of the muscle, where TPs are most commonly located. When injecting TPs at the angle of the neck, one must take care to avoid the apical pleura (see Fig. 55-9) (Figure Not Available) .

**Sternocleidomastoid**

This muscle is often the source of neck pain and headache. In addition, dizziness and ipsilateral ptosis, lacrimation, and reddening of the conjunctiva have been reported in association with involvement of these muscles. [40] These TPs are most commonly located at occiput insertion in the upper two thirds of the muscle and frequently on its sternal and clavicular origins. The pain pattern may involve the muscle or may refer to the ear region, the face, or the frontal area (see Fig. 55-9) (Figure Not Available) .

## CONCLUSION

In summary, TPs may be involved in any painful event. They may play either a primary role (translating stress to pain) or a secondary role (supporting and intensifying a painful stimulus). They can complicate any type of pain and may mimic underlying visceral disorders. Obviously, interventions that directly affect the TP have a much greater chance for complete pain control if the TP is acting in the primary role of translator than if it is acting in the secondary role of pain intensifier. Even in the secondary role, however, there is the potential for benefit from properly administered treatment. The importance of TP therapy for differential diagnosis cannot be overemphasized.

Stretch-and-spray techniques, injections of local anesthetics, injection of saline solution, and dry needling are all useful in treating TP pain syndromes. However, use of local anesthetics is most appropriate when immediate reduction of severe pain is sought. Treatment considerations in addition to TP injection include administration of muscle relaxants, anesthetics, and analgesics; referral for physical therapy; reduction of stress; and prescription of therapeutic exercise following resolution of the event.

# Chapter 56 - Injection Therapy of Bursitis and Tendinitis

**David H. Neustadt**

*Bursitis* and *tendinitis* are terms frequently used to describe a variety of regional musculoskeletal conditions that are characterized chiefly by pain and disability at the involved site. Bursitis of the shoulder may be considered the prototypical disorder. All too often, in ill-defined regional soft tissue rheumatic problems, bursitis or tendinitis is used as a "wastebasket" diagnosis. For purposes of this chapter, in consideration of the accurate diagnosis that is necessary to institute appropriate therapy, the terms are reserved for well-defined, specific clinical entities.

## GENERAL ANATOMIC CONSIDERATIONS OF BURSAE AND TENDON SHEATHS

Bursae are potential spaces or sacs, subcutaneous (subcutaneous) or deep, that develop in relation to friction and facilitate the gliding motion of tendons and muscles. There are approximately 78 bursae on each side of the body. These were well described in the classic atlas of anatomy by Monro [1] in 1788 and were later elaborated in greater detail in the atlas of Spalteholz. [2]

The normal bursal wall is lined with a thin layer of synovial cells that appear to be similar to those of joint synovial membrane when examined by electron microscopy. [3] When a bursa becomes subacutely or chronically inflamed, the normally thin surface of sparse cells may thicken to 1 to 2 mm. The cause of bursitis may be trauma, infection, crystal deposition, chronic friction, or a systemic inflammatory arthropathy. In addition, so-called adventitial bursae may form in response to abnormal shearing stress at sites subjected to chronic pressure; an example is a bunion over the head of the metatarsal bone of the great toe.

Involvement of the synovial lining of bursae and tendon sheaths may also result from underlying systemic diseases, including rheumatoid arthritis, ankylosing spondylitis, psoriatic arthropathy, and gout. The most common bursal lesions in these systemic inflammatory arthropathies involve the olecranon at the elbow and the trochanter region of the hip. Smaller bursae, especially those around the Achilles tendon, also may be affected.

*Septic bursitis* usually affects the superficial bursae, such as the olecranon and the prepatellar regions. [4] Factors that predispose to infected bursae include trauma, steroid therapy, uremia, diabetes mellitus, and alcoholism. [5] Tuberculosis may affect any bursa but is rare, whereas other types of mycobacteria, such as *Mycobacterium kansasii*, are occasionally reported. [6] Another uncommon cause of involvement of superficial bursae and tendon sheaths is sporotrichosis, which can be contracted by gardeners and farmers. In addition, tendon sheaths at the hands, the wrists, and the ankles may be

affected by acute bacterial infections, such as gonorrhea.

*Tendinitis* and *tenosynovitis* are useful terms that describe inflammatory reactions in tendons and tendon sheaths. Tendon sheaths are relatively long and tubular, whereas bursae are round and flat. Except for their shape, however, the structures are similar. Common sites of tendinitis in the body are depicted in Figure 56-1 (Figure Not Available) .

Flexor tenosynovitis ("trigger," or "snapping," finger) is a frequent extra-articular manifestation of rheumatoid arthritis and occasionally may be the presenting symptom.

In *calcareous* (or calcific) tendinitis of the shoulder, there is a calcific deposit in and about one of the rotator cuff tendons (commonly the supraspinatus). The musculotendinous rotator cuff is composed of the supraspinatus, infraspinatus, teres minor, and subscapularis muscles, which insert as the conjoined tendon into the greater tuberosity of the humerus. The bursae in relation to the greater tuberosity and the subdeltoid (subacromial) bursa are the most common sites of calcific deposits. The nidus of the pathologic process is considered to be the calcific deposit (hydroxyapatite) within the substance of one or more of the involved tendons. The process has been likened to a chemical furuncle, or the so-called calcium boil. Calcific tendinitis may be hyperacute or acute, and release of the pressure from the inflammatory edema with rupture into the contiguous bursa (e.g., the subacromial bursae) provides prompt relief.

Bursitis and tendinitis embrace a variety of conditions that may be grouped together on a regional basis for the sake of a simple and convenient classification (Table 56-1)

## RATIONALE FOR STEROID INJECTIONS

The management of pain resulting from bursitis and tendinitis may be greatly enhanced by the proper selection and administration of local injections. Successful application of local injection and intrasynovial therapy requires an understanding of the diagnosis, accurate localization of the pathologic condition, and the appropriate choice of suitable injection techniques. Not infrequently, injections of lidocaine or corticosteroid preparations provide the additional aid that, alone or as an adjunct to the management program, overcomes the refractory pain.

Although local injection intrasynovial therapy is essentially palliative, it may provide striking and lasting relief. Restoration of function may follow a single injection, especially in a self-limited painful soft tissue condition. The precise mechanism of the lasting analgesia and the beneficial therapeutic effects have not been clarified. Explanations that have been considered include induction of local hyperemia, relaxation of reflex muscle spasm, generalized response from systemic absorption, pain relief allowing controlled activity or rest, favorable influence on local tissue metabolism, and mechanical benefit. The increased mobility permitted as a result of pain relief certainly accelerates recovery and restoration of function. Finally, the "power of suggestion" of the needle must not be underestimated (placebo therapy). Some observers believe the

pain relief may result from stimulation and release of the patient's endorphins.

Some physicians prefer to follow local injection with a short course of salicylates, oral corticosteroids, or nonsteroidal anti-inflammatory medications. Others prefer to prescribe simple analgesics and to evaluate the response to injection. In selected cases, short-term splinting or other forms of immobilization are beneficial. Cases must be individualized according to the specific pathologic condition and patient variables.

## INDICATIONS AND CONTRAINDICATIONS

Local injection therapy with corticosteroids or local anesthetics may provide valuable aid in a variety of acute or subacute bursitides and other painful soft tissue conditions. Abolition of symptoms confirms the localization of the involved site or structure, even if the response is not lasting. Visceral disease must be ruled out as a source of referred pain. Appropriate injection therapy is indicated when there are local accessible signs that are likely to respond to direct therapeutic infiltration. Acute localized bursitis or tendinitis warrants immediate direct injection for rapid relief.

Contraindications are relative and include infections, either local or in the vicinity of the site of involvement, and hypersensitivity to any preparation or substance that might be injected. The procedure is also contraindicated in patients receiving anticoagulants or those with any bleeding disorder. The patient with a pre-existing tendon injury may be subject to tendon rupture, which can inhibit full activity when the corticosteroid injection removes pain. Hence, partial tendon rupture is a relative contraindication. [9] Poorly motivated, "needle-shy," and severely neurotic patients obviously are considered poor subjects for this type of treatment. Active

| **TABLE 56-1** -- Classification of Bursitis and Tendinitis (Regional) |
|---|
| **Upper Extremity Disorders** |
| *Elbow* |
|    Radiohumeral bursitis, olecranon bursitis, epicondylitis |
| *Shoulder* |

| |
|---|
| Bicipital tendinitis, calcareous tendinitis (subacromial, subdeltoid bursitis), rotator cuff tendinitis |
| *Wrist and Hand* |
| Stenosing tenosynovitis ("trigger finger" syndrome), de Quervain syndrome |
| **Lower Extremity Disorders** |
| *Hip* |
| Trochanteric bursitis, ischiogluteal bursitis |
| *Knee* |
| Prepatellar, suprapatellar, and anserine bursitis |
| *Ankle, Foot, and Heel* |
| Ankle tendinitis, bunion bursitis, calcaneal bursitis (with heel spur) |

herpes simplex infection and tuberculosis are generally considered contraindications.

## HAZARDS AND COMPLICATIONS

Local anesthetics are often mixed with a corticosteroid preparation to increase volume, decrease postinjection pain, and assess the accuracy of the injection. Local anesthetics may also be used before injection of the corticosteroid. The major hazards in the use of local anesthetics are hypersensitivity and accidental IV or intra-arterial introduction.

Serious (possibly even fatal) hypersensitivity to procaine and other regional anesthetic compounds is encountered very rarely; the possibility is usually suggested by a history of previous reactions. Lidocaine or one of the newer "caine" derivatives may be used to avoid sensitivity reactions to procaine. Bupivacaine is an excellent choice because of its safety and long duration of action. If a past history of a reaction is suggested, one should proceed cautiously and use small amounts of dilute anesthetic solutions. When there is a definite history of sensitivity, it is wise to avoid the use of anesthetic agents during injection therapy.

In the event of accidental IV injection of a "caine" drug or if symptoms of hypersensitivity arise from these compounds (e.g., a significant slowing of the pulse rate, or even a seizure, is an indication of a major reaction to IV administration), a benzodiazepine anticonvulsant, or rapidly acting barbiturates (e.g., sodium pentobarbital) should be readily available and should be given promptly in accordance with the reaction and response (see Chapter 31) . In addition, a clear airway should be maintained, and oxygen should be administered. Severe reactions from accidental vascular injection of these drugs in the doses usually given are very rare.

A repository corticosteroid given IV by accident has been reported. [10] No serious reaction to a depot corticosteroid preparation given IV has been reported. The possibility of an allergic reaction caused by corticosteroids is highly unlikely. However, an unusual skin rash following an intra-articular methylprednisolone injection has been reported that appears to be consistent with a delayed type of hypersensitivity. [11]

Minor reactions occasionally seen after injection of "caine" preparations include light-headedness or dizziness, pallor, weakness, sweating, nausea, and (rarely) fainting and tachycardia. These symptoms usually disappear within a few minutes after the injection and rarely require any treatment except reassurance and a cold compress to the forehead of the patient. Often it is difficult to decide whether the symptoms are the result of sensitivity to the drug or a fright reaction (vasovagal). *The patient should always be in a supine, prone, or reclining position during the injection to minimize the effect of any vasovagal reaction.*

The obvious ways to prevent entering a blood vessel are an awareness of the local anatomy and aspiration after every 1 to 2 mL of solution are injected. Penetration into or striking a nerve may cause sharp pain or paresthesias, and the patient should be warned of this possibility in advance.

Complications are listed in Table 56-2 . Although the possibility of introducing infection is the most serious potential complication, in a review of my extensive experience and that of others, I found that infections occurring as an aftermath of intrasynovial injections are extremely rare.  I do

---

**TABLE 56-2** -- Complications of Injection Therapy

| |
|---|
| Infection |
| Postinjection flare |
| Afterpain |
| Bleeding (local) |
| Cutaneous atrophy (local) |
| Depigmentation |
| Tendon rupture |

not recommend routine prophylactic antibiotic administration unless the patient has had a significant recent systemic infection. Although the problem of infection is usually avoided with meticulous attention to aseptic technique, the patient should be cautioned to report the development of any significant pain, redness, or swelling after any local injection.

Local undesirable reactions are usually minor and reversible. A postinjection "flare," manifested as a significant increase in pain, may begin within a few hours after steroid injection and usually tends to subside spontaneously in up to 24 hours. Rarely, it can continue for as long as 72 hours. This transient increase in inflammation is considered to represent a true crystal-induced "synovitis" caused by precipitation of the microcrystalline steroid ester suspension. [16] Usually, the reaction is mild and can be adequately controlled with application of ice or cold compresses and analgesics as needed. Rarely, "afterpain" lasting for a few to several hours may occur following injections. Although the cause is obscure, this phenomenon may result from the trauma of needle insertion, penetration of inflamed tissue, or pressure on adjacent nerves from local swelling or bleeding. Afterpain usually is relieved by application of moist or dry heat and analgesics until the pain abates but is best handled by mixing a long-acting anesthetic, such as bupivacaine, with the steroid preparation.

Occasional subcutaneous bleeding at the site of injection may occur with penetration of

a venule, an arteriole, or a capillary. The patient should be warned that this may occur and should be reassured that the discoloration or hematoma will disappear spontaneously. Ice packs or cold compresses applied to the involved area for the first 24 hours are commonly advised.

Another relatively minor complication is localized subcutaneous or cutaneous atrophy at the site of the injection. [8] [17] This problem is chiefly of cosmetic concern and is recognized as a small depression in the skin frequently associated with depigmentation, transparency, and occasionally the formation of telangiectasia. These changes in the skin occur when injections are made near the surface and some of the injected steroid leaks back along the needle track. The skin depression usually recedes and the skin returns to normal with time, when the crystals of the steroid have been completely absorbed. Careful technique (avoiding any leaking of the steroid suspension to the skin surface) prevents this complication. A small amount of lidocaine or normal saline can be used to flush the needle of the suspension before removing it.

The potential danger of "spontaneous" tendon rupture (especially of Achilles tendons) following local corticosteroid injections in the Achilles bursal area must be given serious

consideration. Cautious administration with infiltrations around and beneath the tendons to keep any of the material from entering the substance of the tendon minimizes the occurrence of this complication. In general, *the injection of major stress-bearing tendons*, such as the Achilles and patellar tendons, *should be avoided in the emergency department*. Treatment with oral anti-inflammatory medications and splinting is preferred.

## AVAILABLE PREPARATIONS AND CHOICE OF COMPOUND

Hydrocortisone and a variety of available corticosteroid repository preparations are described in Table 56-3 . Local anesthetics, such as lidocaine or bupivacaine, can be mixed with the corticosteroid preparation in the same syringe. All corticosteroid suspensions, with the exception of cortisone and prednisone, can produce a significant and rapid anti-inflammatory effect (in synovial spaces). Unfortunately, soluble corticosteroids are absorbed and dispersed too rapidly, having only a brief duration of action locally. The tertiary butyl acetate (TBA) ester prolongs the duration of local tissue effect because of decreased solubility. The decreased solubility probably causes dissociation of the corticosteroid by enzymes to proceed at a delayed rate.

No single steroid agent has demonstrated a convincing margin of superiority, with the exception of triamcinolone hexacetonide. Prednisolone tebutate, however, simply by virtue of price advantage and long-term usage, is generally the drug of choice. Triamcinolone hexacetonide is the least water-soluble preparation currently available. Triamcinolone hexacetonide is 2.5 times less soluble in water than prednisolone tebutate, providing the longest duration of action. There is minimal systemic absorption, or "spillover," with this preparation, but because of its high potency and higher cost, it is usually reserved for use in conditions in which prednisolone tebutate or one of the other compounds has shown an inadequate response. Because of the long duration of action and its greater potential for subcutaneous atrophy, some authors use this preparation

only for intra-articular injections.

## DOSAGE AND ADMINISTRATION

The dose of any corticosteroid suspension used for intrasynovial injection must be arbitrarily selected. Factors that influence the dosage and expected response include the size of the affected area, the presence or absence of synovial fluid or edema, the severity and extent of any synovitis, and the steroid preparation selected for injection.

A useful guideline for estimating dosage follows: For relatively large spaces such as subacromial, olecranon, and trochanteric bursae, 20 to 30 mg of prednisolone tebutate or equivalent; for medium- or intermediate-sized bursae and ganglia formation at the wrists, knees, and heels, 10 to 20 mg; and for tendon sheaths, such as flexor tenosynovitis of digits and the abductor tendon of the thumb (de Quervain disease), 5 to 15 mg. Sometimes it may be necessary to give larger doses for optimal response. Intrabursal therapy of elbow (olecranon) or knee (prepatellar) bursae containing considerable fluid may require 30- to 40-mg doses.

Unlike intra-articular injections for synovitis in chronic joint disease, repeat infiltrations for soft tissue conditions such as bursitis and tendinitis frequently are not required. If only a partial response occurs or if recurrence develops, however, a single repeat injection can be given; the length of the interval between injections should not be a source of undue concern. In contrast with intra-articular injections, in which the hazard of "overworking" an injected joint is usually not a problem, following intrasynovial injection we recommend a reduction in activity with rest or splinting of the involved extremity. Limiting motion also delays somewhat the systemic absorption of the steroid. Anecdotal evidence suggests that those patients with inflammatory soft tissue lesions who follow a postinjection modified rest regimen obtain a more rapid and lasting resolution of the painful disorder. Frequency of injections is considered further in the discussion of techniques for the specific entities.

## PREPARATION OF THE SITE

Preparation of the site before injection requires meticulous adherence to aseptic technique. Anatomic landmarks are outlined with a black or red skin pencil. Tincture of iodine

| TABLE 56-3 -- Injectable Corticosteroids | | | |
| --- | --- | --- | --- |
| **Intrasynovial Preparations** | **Trade Name** | **Strength per mL** | **Range of Usual Dosage** |
| | | | |

| | | | |
|---|---|---|---|
| Hydrocortisone tebutate | Hydrocortisone TBA | 50 mg | 12.5-75 mg |
| Prednisolone tebutate | Hydeltra-TBA | 20 mg | 5.0-30 mg |
| Methylprednisolone acetate | Depo-Medrol * | 20 mg | 4.0-40 mg |
| Triamcinolone acetonide | Kenalog-40 | 40 mg | 4.0-40 mg |
| Triamcinolone diacetate | Aristocort Forte | 40 mg | 4.0-40 mg |
| Triamcinolone hexacetonide | Aristospan | 20 mg | 4.0-25 mg |
| Betamethasone acetate and disodium phosphatate | Celestone Soluspan | 6 mg | 1.5-6.0 mg |
| Dexamethasone acetate | Decadron-LA | 8 mg | 0.8-4.0 mg |

* Supplied in 20-mg/mL, 40-mg/mL, and 80-mg/mL preparations.
Long acting, best used for intra-articular injection only.
Available as 3 mg acetate, 3 mg phosphate.

or thimerosal (Merthiolate) applied with a sterile swab can be used in place of the skin pencil. The point of entry is cleansed with povidone-iodine (Betadine) and alcohol. Sterile drapes are not generally considered necessary, especially after sufficient skill and experience with the procedure have been acquired. For operator protection, latex examination gloves (nonsterile) should be worn.

## TECHNIQUES

### General Considerations

Materials required for local injection procedures include needles, syringes, and items for

preparation of the injection site. Disposable needles and syringes are convenient and adequate for emergency department use. Special trays may also be stocked for this purpose. The usual sizes of needles for various approaches are as follows:

*Intradermal skin wheal:* 1.3 cm (0.5 inch), 25 ga

*Tendinitis in elbow and shoulder inflammation:* 3.9 to 5.0 cm (1.5 to 2.0 inch), 23 or 25 ga

*Digital tenosynovitis:* 2.2 cm (7/8 inch), 25 ga

*Bursitis with fluid:* 2.5 to 3.9 cm (1.0 to 1.5 inch), 23 to 25 ga

*Deep gluteal bursitis:* 7.6 to 10.2 cm (3.0 to 4.0 inch), 20 to 22 ga

Table 56-4 lists appropriately sized needles for various injection sites.

Once the point of entry has been determined and the site is prepared, either a superficial skin wheal is made with 1% lidocaine (Xylocaine) or 0.25% bupivacaine (Marcaine, Sensorcaine) or the skin is sprayed with a refrigerant such as dichlorotetrafluorethane (Frigiderm). Some physicians do not use a skin wheal when the steroid is mixed with a rapid-acting local anesthetic. Ordinarily, preanesthesia is not necessary, but occasionally, in highly nervous or agitated individuals, it may be advisable to give opioids or benzodiazepines IV or to administer a nitrous oxide-oxygen mixture before beginning the procedure. Thus, an anxiety-provoking injection can be carried out with patient cooperation.

Local injections can be administered with corticosteroids and local anesthetics mixed together in the same syringe. Because the steroid tends to precipitate or layer in the barrel of the syringe during the injection, the syringe should be agitated immediately prior to use to optimize its distribution. The local anesthetic can be given alone. Generally, when injecting synovial spaces, the steroid is introduced without anesthetic, but often syringes are changed, and lidocaine is used to flush out the needle, frequently causing injection and deposition of several milliliters of the local anesthetic. When injecting a painful soft tissue structure directly, it is best to administer a mixture of corticosteroid and anesthetic with the same syringe. This both relieves pain immediately and confirms the accuracy of the injection. The duration of action of lidocaine is approximately 100 minutes, whereas bupivacaine may last for a few hours. The patient should be cautioned that *the local anesthetic effect may "wear off" within a couple of hours* and that *the beneficial effects of the corticosteroid may be delayed*.

The most important aspect of a successful technique is accurate positioning of the needle. The needle must "hit the mark," or the results will be disappointing. Injecting an inflamed synovial space, such as a bursa-containing fluid, may be as simple as puncturing a balloon. Aspiration of the fluid confirms that the needle has correctly entered the sac. On the other hand, direct injection into a painful soft tissue lesion requires additional skill that can be acquired only with experience. When a bursa is

injected, as much fluid as possible is aspirated before instillation of the corticosteroid suspension to reduce the dilution factor. Sometimes it is advisable to reaspirate and reinject several times within the barrel of the syringe, so-called *barbotage*, to obtain heterogeneous mixing and maximal dispersion of the steroid throughout the synovial cavity.

While an accurate injection is desirable, using a generous

**TABLE 56-4** -- A Guide for Needle Size and Dosage for Injection of Common Regional Disorders

| Disorder or Injection Site | Needle Size | Usual Dosage of Prednisolone Tebutate |
|---|---|---|
| Bicipital tendinitis | 1.5-2.0 in., 22-25 ga | 20-30 mg |
| Calcareous tendinitis | 1.5-2.0 in., 22-25 ga | 20-40 mg |
| Subacromial bursitis | | |
| Radiohumeral bursitis | 1.5 in., 22-25 ga | 20-30 mg |
| Epicondylitis | | |
| Olecranon bursitis | 1.0-1.5 in., 20 ga | 15-30 mg |
| Ganglia on wrist | 1.0 in., 18-20 ga | 10-15 mg |
| de Quervain syndrome | 7/8 in., 22-25 ga | 10-20 mg |

| | | |
|---|---|---|
| Carpal tunnel syndrome | 1.0-1.5 in., 22-25 ga | 20-40 mg |
| Digital flexor tenosynovitis | 7/8 in., 22-25 ga | 5 mg |
| Trochanteric bursitis | 1.5-2.0 in., 22-25 ga | 20-40 mg |
| Prepatellar bursitis | 1.0-1.5 in., 22-25 ga | 15-20 mg |
| Anserine bursitis | 1.0-1.5 in., 22-25 ga | 20-40 mg |
| Bunion bursitis | 1.0 in., 22-25 ga | 5-10 mg |
| Calcaneal bursitis | 1.0 in., 22-25 ga | 10-20 mg |

volume of anesthetic (3 to 6 mL) to dilute and hence disperse the steroid can compensate for some less than perfect injections. As a general rule, one should allow the patient to localize the area of maximum tenderness; the injection should begin in that area. Limiting the patient to 1 finger to localize the area of maximum pain and tenderness is the best way to ensure the most accurate positioning of the needle. If pain persists despite the use of a local anesthetic, an additional injection may be required.

## Specific Regions and Clinical Entities

Table 56-1 and Figure 56-1 (Figure Not Available) list the areas of involvement by region. In addition, certain nonarticular disorders that may require local injection are included.

### Shoulder Region

Pain associated with disability may result from any of the intrinsic shoulder disorders, including bicipital tendinitis, calcareous tendinitis, and subacromial bursitis (Fig. 56-2) (Figure Not Available) . These areas are frequently injected because it is safe and easy to do so, one gets a consistently good response to therapy, and to avoid the danger of

an untreated persistent inflammation resulting in a "frozen shoulder."

**Bicipital tendinitis (tenosynovitis).**

This is a nonspecific low-grade inflammation or irritation of the long head of the biceps tendon sheath. [21] The tendon courses through the joint and along the bicipital (intertubercular) groove. Pain at the shoulder is accompanied by restricted motion and disturbed scapulohumeral rhythm. Efforts to elevate the shoulder, reach the hip pocket, or pull a back zipper all aggravate the symptoms. "Rolling" the bicipital tendon produces localized tenderness (Lipman test), and the Yergason test may be positive. The Yergason test elicits pain along the bicipital groove when the patient attempts supination of the forearm against resistance, holding the elbow flexed at a 90° angle against the side of the body (Fig. 56-3) (Figure Not Available) . Radiographs are normal.

**Approach.**

The point of maximal tenderness of the bicipital tendon is located. Entry is made with a 22- or 25-ga, 3.9- to 5.0-cm needle through a lidocaine skin wheal (Fig. 56-4) (Figure Not Available) . It is wise to *avoid an actual intratendinous injection,* which may cause weakening of the tendon and predispose the patient to tendon rupture. The needle is brought in along the side of the tendon, aimed at one border of the bicipital groove to give a *peritendinous infiltration*. One third of the injection is administered at this point. The needle is then withdrawn slightly but is kept subcutaneous. It is redirected upward approximately 2.5 cm for another one third of the injection, withdrawn again, and redirected downward, touching the bicipital border gently; the remainder of the drug is deposited at this point. Usually the corticosteroid suspension, 1 to 1.5 mL of prednisolone tebutate, is instilled at the maximum area of tenderness, and the lidocaine is injected along the upper and lower borders of the tendon. Two to 4 repeat injections may be required at 1- to 2-week intervals.

**Calcareous tendinitis, supraspinatus tendinitis, and subacromial bursitis.**

These inflammations are so clinically similar that their symptoms and signs are difficult to differentiate. The acute irritative inflammation of the bursa is a secondary reaction produced by the calcific tendinitis of the supraspinatus or one of the other rotator cuff tendons. After the calcific material ruptures into the subdeltoid bursa (Fig. 56-5) , spontaneous relief usually is obtained within a few days. During the acute or hyperacute stage, the patient holds the arm in a protective fashion against the chest wall. Pain may be incapacitating, and all ranges of motion are disturbed, with internal rotation markedly limited. Tenderness is often diffuse over the perihumeral region. Constitutional symptoms are rare, but sometimes in the hyperacute form, actual swelling may be visible, and even fever and an accelerated sedimentation rate may develop. When shoulder radiographs demonstrate a calcific deposit, the shadow appears "hazy," with lightening of the periphery caused by the pressure of inflammatory edema. Night pain may be intolerable, requiring opioids for control.

**Anterior approach.**

In calcific tendinitis or supraspinatus tendinitis without calcification, the injection is given by the anterior (subcoracoid) or lateral approach, below the acromion (Fig. 56-6) (Figure Not Available) . If the tenderness is not localized, a point is selected over the depression that is palpable between the anterolateral or anteromedial border of the acromion and the head of the humerus.

**Posterior approach.**

With the patient sitting and the lower part of the extremity resting on the lap, a lidocaine skin wheal is made at a posterolateral point under the acromion. A 3.9- to 5.0-cm, 22- or 25-ga needle is then directed toward the center of the head of the humerus and upward at an angle of approximately 10°. After the site has been penetrated 2 to 3 cm, aspiration is carried out for any fluid or calcific material. The syringe is then removed, leaving the needle in position. Another syringe containing 20 to 40 mg of prednisolone suspension or equivalent is attached, and the medication is instilled. This injection can be followed with 1 to 5 mL of 1% lidocaine (or a similar volume of 0.25% bupivacaine), or local anesthetic can be given combined with the steroid in the same syringe. One should be generous with the volume of local anesthetic to ensure adequate dispersion of the steroid. A single treatment relieves the majority of acute disorders, but occasionally it may have to be repeated once or twice.

Sometimes a painful reaction may follow when the analgesic has worn off. To avoid severe pain, the patient should be warned about this possibility and given appropriate analgesia. A sling may provide additional relief, and short-term use of opioids is appropriate.

**Acromioclavicular joint inflammation.**

Pain arising in the acromioclavicular joint is frequently an aftermath of an acute injury. With this injury, all ranges of motion of the shoulder cause pain, and the joint is tender but rarely swollen.

**Approach.**

Entry is made through a cutaneous lidocaine wheal over the interosseous groove at the point of greatest tenderness (Fig. 56-7) (Figure Not Available) . The joint line is relatively superficial, and a 2.2- to 2.5-cm, 22- or 25-ga needle is adequate. One to 2 mL of lidocaine and 5 to 10 mg of a prednisolone suspension are injected. It is not necessary to advance the needle beyond the proximal margin of the joint surface.

**Elbow Region**

The elbow is subject to frequently occurring characteristic extra-articular disorders.

These include radiohumeral bursitis, epicondylitis ("tennis elbow" and "golfer's elbow"), and olecranon bursitis ("barfly's elbow").

**Radiohumeral bursitis.**

This occurs at the juncture of the radial head and the lateral epicondyle of the elbow. Radiohumeral bursitis is commonly found in combination with lateral epicondylitis. The symptoms of the 2 adjacent problems are indistinguishable, but tenderness in bursitis overlies the site of the radiohumeral groove, whereas tenderness in tennis elbow occurs chiefly at the lateral epicondyle (Fig. 56-8) (Figure Not Available) . A clinical sign supporting the diagnosis of tennis elbow is the provocation of pain when the patient attempts elevation of the middle finger against resistance, with the wrist and the elbow held in extension.

**Approach.**

The entry site is the point of maximal tenderness, usually found at a point slightly distal to the lateral epicondyle. The radial head can be palpated and confirmed by rotation of the patient's forearm (Fig. 56-9) (Figure Not Available) . The injection is made through a skin wheal with a 3.9-cm, 22-ga needle, depositing 20 to 30 mg of prednisolone mixed with lidocaine. Alternatively, the steroid is followed with 1 to 3 mL of local anesthetic. Part of the repository preparation may be instilled into the radiohumeral bursa and part at the lateral epicondyle (Fig. 56-10) (Figure Not Available) . Generally, it is wise to limit repeat injection attempts to 2 or 3. In medial epicondylitis (golfer's elbow), a similar technique can be used, but it is important to remember to avoid the ulnar nerve, which lies in a groove behind the medial epicondyle.

**Olecranon bursitis.**

"Student's elbow", or "miner's beat elbow," is a common condition that is frequently idiopathic or provoked by minor trauma (Fig. 56-11 *A* and *B*). It is common for patients to present following some minor, often unrelated, trauma to the elbow region. The bursa also can be involved in rheumatoid arthritis and gout. Although most cases of olecranon bursitis are sterile, the olecranon bursa is the most frequent site of septic bursitis. The diagnosis of olecranon bursitis is obvious when the elbow is inspected and examined during flexion and extension. Radiographs are normal, but bony spurs (of questionable relationship) may be noted at the olecranon process, and amorphous calcific deposits may be seen. Occasionally in rheumatoid arthritis and gout, nodules, or tophi, may be readily palpated within the bursal sac. The olecranon bursa that is most commonly involved lies between the skin and the olecranon process. Motion at the elbow joint remains complete and painless unless there is also "true" elbow joint involvement.

Olecranon bursitis is bothersome to the patient but often does not cause discomfort and may resolve spontaneously unless there has been bleeding into the bursa or the effusion is extremely tense. When the bursa is large and subject to trauma or is tender and inflamed, provided that infection is excluded, aspiration and a steroid injection will expedite resolution (Fig. 56-11 *C-E*). Smith and associates demonstrated the superiority

of intrabursal methylprednisolone acetate over oral naproxen or placebo at 6 months, noting faster resolution and less reaccumulation of fluid with the steroid injection. [23] The addition of a nonsteroidal drug after steroid injection did not affect the outcome. Aspiration without injection is often followed by recurrence in a few days or weeks.

Fluid may be aspirated for analysis and culture to help distinguish a noninflammatory process from an inflammatory or septic bursitis. Clear or serosanguineous (and xanthochromic) fluid is seen with a sterile bursitis, whereas cloudy fluid should suggest a septic process. The sterile bursal fluid may demonstrate 50 to 4000 leukocytes/mL, predominantly macrophages and lymphocytes, and nonbirefringent cytoplasmic inclusion bodies can be seen. Glucose, protein, and complement ($C_3$ ) concentrations in bursal fluid range from 60% to 80% of respective serum values.

It is usually easy to detect a septic bursitis by physical examination (Fig. 56-12) . The bursal area is red, hot, swollen, and painful. Fever and systemic leukocytosis are not generally sensitive or specific findings. Smith and associates reported that the surface temperature of the skin overlying the olecranon bursa may be a very sensitive and specific indicator that can differentiate septic from nonseptic cases. [24] In their study, using an electronic temperature probe attached to the skin, the mean temperature difference uniformly between the affected and contralateral elbow was 3.7 °C in cases of septic olecranon bursitis versus 0.7 °C in nonseptic bursitis. A temperature difference of 2.2 °C (sensitivity, 100%; specificity, 94%) was more helpful than bursal fluid leukocyte count, cell differential, or Gram stain for differentiating septic from nonseptic bursitis. This study suggests a possible role for the recently developed infrared thermometer that detects surface temperatures.

Initial treatment of a septic bursitis includes aspiration of all fluid, appropriate culturing, and antibiotic therapy. Corticosteroid injections should not be used for cases of clinically apparent septic bursitis. [25] Antibiotics directed against penicillin-resistant *Staphylococcus aureus*--either oral or parenteral, depending on the extent of infection--are indicated. In frank septic bursitis, especially with thickening of the bursal wall or in cases resistant to antibiotic therapy, open incision with drainage may be required, with possible subsequent excision of the bursa. Rarely, acute gout or a cholesterol crystal synovitis develops, and this may mimic a suppurative bursitis.

**Approach.**

A 2.5- to 3.9-cm, 20-ga needle is introduced at a dependent aspect of the bursal sac through a lidocaine skin wheal; Frigiderm spray may be used, or the skin may remain unanesthetized. After *all* of the fluid is aspirated, 15 to 30 mg of prednisolone tebutate or an equivalent preparation is injected. The elbow is wrapped with an elastic compression bandage for 5 to 7 days following aspiration and injection. If infected or inspissated fluid is anticipated, a 16- to 18-ga needle may be necessary for aspiration of the viscous contents.

**Wrist and Hand Region**

**Ganglion cysts of wrist or hand.**

These are cystic swellings occurring frequently on the hands, especially on the dorsal aspect of the wrist. Ganglion cysts are common and make up approximately 60% of all soft tissue tumors affecting the wrist and hand. They usually develop spontaneously in adults between 20 and 50 years old, with a female-to-male ratio of 3:1.

The etiology of ganglia remains obscure. The word *ganglion* is derived from the Greek word meaning "cystic tumor." The cystic structures are attached to or may arise from tendon sheaths or near the joint capsule, and attachment is often by a pedicle. In 1971, Andr'en and Eiken [26] demonstrated that contrast material instilled into the wrist would pass into some ganglia, especially the volar wrist ganglion. However, the material injected into the ganglion did not flow into the joint. At times, the wall of the ganglion is distinct from that of the synovial membrane.

The wall of the ganglia is smooth, fibrous, and of variable thickness. The cyst is filled with a clear, gelatinous, sticky or mucoid fluid of great density. The viscous fluid in the cyst may sometimes represent almost pure hyaluronic acid.

The types of ganglia vary with their location. The most common ganglion is the dorsal wrist variety, which arises from the scapholunate joint and constitutes approximately 65% of ganglia. The volar wrist ganglion, which arises over the distal aspect of the radius, constitutes approximately 20% to 25% of ganglia (Fig. 56-13 *A* and *B*). Flexor tendon sheath ganglia make up the remaining 10% to 15% of ganglia found on the hand and wrist.

Another type of cystic ganglia on the dorsal wrist occurs not infrequently in patients with rheumatoid arthritis, but is easily recognized because of its soft, irregular appearance in association with other evidence of rheumatoid extensor synovitis.

Most ganglia are of no great clinical significance, and most do not require treatment. Spontaneous regression is common. However, if the appearance of the cyst is disturbing to the patient or if the ganglion is painful or tender (from soft tissue or nerve compression or bone erosion), then simple aspiration with or without injection of a corticosteroid suspension is usually an effective approach. Up to 3 aspirations may be required before the technique is termed a failure. A recent report of treatment by aspiration alone showed that 69% of 116 patients required only a single aspiration. [27] Two or 3 aspirations were required for 19%, and only 12% ultimately needed surgical excision. Certainly, aspiration with or without steroid injection is associated with considerable cost savings when compared with surgery. The "old" treatment of attempting to rupture the ganglion with a heavy book is not advised due to the potential for local injury. In the event of failure of nonsurgical treatment, surgical excision my be indicated. Reported recurrence rates after surgical removal range between 6% and 50%.

**Approach.**

Following instillation of 1% lidocaine for local anesthesia, a 2.5-cm, relatively large-bore needle (17 to 18 ga) is inserted into the center of the ganglion. The cyst is aspirated, with an attempt to achieve maximum evacuation of the contents. Usually 1 to 2 mL of mucinous fluid can be aspirated. Milking the contents of the cyst toward the aspiration needle can maximize the volume that is removed. When a steroid is administered, 10 to 15 mg of prednisolone tebutate (or equivalent) are injected. The use of steroids instilled into the cyst is a common procedure that has been proven to significantly augment the success of simple aspiration. [28] Following aspiration, a splint is usually not required, and activity need not be restricted. There is no proven role for routine nonsteroidal anti-inflammatory drug (NSAID) or oral steroid therapy.

**de Quervain disease.**

This relatively common disease is a stenosing tenosynovitis of the short extensor (extensor pollicis brevis) and long abductor tendon (abductor pollicis longus) of the thumb. Although occasionally associated with rheumatoid arthritis, the disorder more often occurs following repetitive use of the wrists, especially with a wringing motion. The syndrome has been called "washerwoman's sprain" but has been seen after repetitive hammering, and often no specific etiology is apparent. Tenderness and, occasionally, palpable crepitation is elicited just distal to the radial styloid process where both tendons come together in an osseofibrous tunnel. Patients usually present with wrist pain and often mistakenly attribute the discomfort to some distant, albeit irrelevant, trauma. The condition may be confused with first carpometacarpal arthrosis (osteoarthritis of the thumb). Radiographs will be normal in de Quervain disease but may be appropriate to rule out other pathologic abnormality.

A useful specific clinical finding that indicates de Quervain disease is the Finkelstein test (Fig. 56-14 *A*). This test is conducted by abducting the patient's thumb into the palm of the hand and folding the fingers over the thumb. Forcible ulnar deviation at the wrist is then carried out, with severe pain at the site of the affected tendon sheaths indicating a positive test. When examining for the Finkelstein test, one should also evaluate the tender area for palpable crepitus (see Fig. 56-14 *B*). If axial traction or compression (the "grind test") and rotation of the thumb produce pain, the condition is most likely due to degenerative changes of the carpometacarpal joint of the thumb rather than from de Quervain disease. It should be noted that gonococcal tenosynovitis of the wrist may mimic de Quervain disease, and one should inquire about other symptoms (e.g., sore throat, penile or vaginal discharge, or fever) and carefully look for the characteristic rash of this sexually transmitted disease (see Fig. 56-14 *C*).

Mild cases may be treated with NSAIDs and a splint, but less than a third will show any improvement. [29] Increasing evidence supports the effectiveness of local corticosteroid injections as the therapeutic preference for de Quervain tenosynovitis. In 1 report, 90% of 56 cases were effectively managed either with a single injection (58%) or with repeat injections (33%), with only 10% requiring surgical treatment. [30] These data support a previous report describing an 83% rate of successful response to injections in 63 wrists

with de Quervain tenosynovitis. [31] The striking favorable response of local injection suggests that surgery to release the tendon sheaths is seldom needed.

**Approach.**

A 2.2-cm, 25-ga needle is introduced at the most tender point (just distal to the radial styloid) through a skin wheal, and 10 to 20 mg of prednisolone suspension mixed with 4 to 5 mL of lidocaine 1% are deposited adjacent and parallel to the tendon sheath (peritendinous infiltration) (Fig. 56-15 (Figure Not Available) *A* and *B*). One should be generous with the injection volume, since a common reason for failure is the inability to get medication into both tendon sheaths. This may be partially overcome by increasing the volume of steroid-lidocaine injected in a more diffuse area. The injection may be repeated 1 or 2 times at 7- to 14-day intervals, if needed. A lightweight thumb or wrist splint for wrist support and protection may be used at night for several weeks after the injection, but routine splinting following injection is not required. [32] Oral NSAIDs may be prescribed as analgesics but will likely not effect a cure by themselves. There is no proven role for oral corticosteroids.

**Carpal tunnel syndrome.**

Carpal tunnel syndrome is a common nerve entrapment caused by median nerve compression. It is characterized by pain at the wrist that sometimes radiates upward into the forearm and is associated with tingling and paresthesias of the palmar side of the index and middle fingers and the radial half of the ring finger. Typically, the patient wakes during the night with burning or aching pain, numbness, and tingling and must shake the hand outside the bedclothes to obtain relief and restore sensation. Occasionally the discomfort is extremely severe, causing the patient to seek emergency aid. Clinical signs supporting the diagnosis include a positive Tinel sign, in which one reproduces the tingling and paresthesias by tapping (with a reflex hammer) over the median nerve at the volar crease of the wrist. [33] In addition, one can perform wrist flexion maneuvers (the Phalen sign) in an effort to provoke the symptoms in the median nerve distribution. [34] Severe muscle atrophy of the thenar eminence may develop in advanced or neglected cases. Causes of carpal tunnel syndrome include rheumatoid arthritis (sometimes as the presenting manifestation), pregnancy, hypothyroidism, diabetes, and acromegaly, but in many cases the disturbance is idiopathic, without a recognizable underlying cause.

**Approach.**

When injecting the carpal canal, one should insert the needle through a skin wheal at a site just medial or ulnar to the palmaris longus tendon and proximal to the distal crease at the wrist (Fig. 56-16) (Figure Not Available) . Injecting medial to the palmaris longus is preferred because it avoids direct injection of the median nerve and superficial veins. A 2.5- to 3.9-cm, 25-ga needle is directed at an angle of approximately 60° to the skin surface, pointing toward the palm. The needle is advanced 1 to 2 cm, and 20 to 40 mg of prednisolone suspension with or without lidocaine are injected along the track and into the tissue space. Up to 2 weeks may be required for symptoms to abate significantly. A lightweight wrist splint may hasten recovery. Repeat injections may be

given, but if response is not successful or lasting after 2 or 3 injections, decompressive surgery should be considered. Nerve compression should be confirmed by nerve conduction studies before surgery. [35]

**Digital flexor tenosynovitis (trigger finger).**

A "trigger" or "snapping" finger is characterized by a stenosed tendon sheath on the palmar surface over the base of the metacarpal head. The area of stenosis leads to intermittent catching of the tendon. Locking occurs when the involved digit is in flexion and is especially troublesome when the patient awakens in the morning. This can occur in any finger but is seen most frequently in the ring and middle fingers.

*Tendinitis* and *tenosynovitis* are terms useful for describing inflammatory reactions in tendons and tendon sheaths. The tendon sheaths are long and tubular, and the walls are lined with a thin layer of synovial cells. Symptoms develop when the tendon becomes trapped and is unable to glide within the tendon sheath (Fig. 56-17) (Figure Not Available) . A nodule or fibrinous deposit may form at a site in the tendon sheath, usually over or just distal to the metacarpal head. Carpal tunnel syndrome commonly coexists with trigger finger and may be caused by tenosynovitis. The common causes include trauma, diabetes mellitus, and rheumatoid arthritis.

Marks and Gunther [36] reported the results of treatment of a consecutive series of 74 patients, in whom 108 local injections of fingers were given, yielding an overall "cure rate" of 93% after 1 or 2 injections. In another study, [37] repeated corticosteroid injections for stenosing tenosynovitis were highly effective and without adverse effects in a similar consecutive series of 93 patients (104 fingers injected). Kraemer and colleagues [38] reviewed their treatment of 253 consecutive digits affected with stenosing flexor tenosynovitis (trigger fingers), comparing the results of steroid injection with those of surgical release. Analysis disclosed no significant difference in efficacy for injection versus surgery. Surgical treatment was associated with more complications and was more costly. The cost for an in-office steroid injection was 10% of the cost for in-hospital surgical release. A single steroid injection provided complete relief of symptoms in 110 digits (67%); 13% of these patients had a recurrence of symptoms. A series of as many as 3 injections provided complete, lasting relief of symptoms in 63% of all digits treated. Group analysis of all patients over the age of 10 years disclosed that 68% of the patients whose initial treatment was by steroid injections experienced complete relief of symptoms, whereas 73% of those whose initial treatment was by surgical release experienced complete relief of symptoms. Gray and colleagues, [39] in a retrospective study, reported resolution of 93% of 173 episodes of rheumatoid digital flexor tenosynovitis and 52 episodes of idiopathic flexor tenosynovitis. Other studies report success rates of 48% to 82% with follow-up of at least 1 year.

The precise mechanism for the lasting analgesia and the beneficial therapeutic effects is not fully understood. Explanations that have been considered include production of local hyperemia, relaxation of reflex muscle spasm, generalized response from some systemic absorption of the steroid suspension, a beneficial influence on local tissue metabolism, pain relief allowing increased rest and sleep, and mechanical improvement. The increased mobility resulting from pain relief undoubtedly accelerates recovery and

restoration of function. Finally, the "power of suggestion" of the needle (the placebo effect) must be given consideration in some cases. Some observers suggest that part of the pain relief may result from stimulation and release of the patient's endorphins.

Conservative treatment, including local rest or splinting, application of moist heat, and NSAID therapy, is the usual initial therapeutic approach for symptomatic tenosynovitis. If these simple measures fail to control symptoms, then corticosteroid injection, appropriately administered to the involved tendon sheath, is indicated. The procedure would be relatively contraindicated in patients receiving anticoagulants. The potential hazard of tendon rupture has been reported infrequently. This rare complication can be prevented by careful infiltration and by avoiding introduction of the corticosteroid into the substance of the tendon. Division of the first annular pulley and digital nerve injury are well-known complications of surgical release.

**Approach.**

Preparation of the site before the injection requires meticulous adherence to aseptic technique. Local anesthesia is usually provided by spraying with a skin refrigerant such as dichlorotetrafluoroethane (Frigiderm) to numb the skin before entry of the needle. The technique for injecting flexor tenosynovitis includes locating and marking the tendon point at the involved metacarpal base and instilling 0.25 to 0.35 mL of any corticosteroid suspension subcutaneous with a 2.2-cm, 25-ga needle into the involved tendon sheath (Fig 56-18) (Figure Not Available) . The needle enters the skin at an angle of approximately 30° and is inserted into the tendon sheath, parallel to the tendon fibers. Resistance should not be encountered on injecting. Similar injections can be administered to the base of the thumb metacarpal for a "snapping" thumb. Up to 4 repeat injections may be given at 6- to 8-week intervals. If relapses are frequent or the clinical response is not satisfactory, then surgical release is indicated.

**Hip Region**

**Trochanteric bursitis.**

Trochanteric bursitis may simulate hip joint disease and sciatica. [42] The principal bursa lies between the gluteus maximus and the posterolateral prominence of the greater trochanter, although other bursae of the hip are also commonly affected. Pain may be acute but is more often subacute or chronic; often patients have tried NSAIDs without success and have been given a number of incorrect diagnoses. The chief locus of the pathologic condition is in the abductor mechanism of the hip. Pain occurs near the greater trochanter and may radiate down the lateral or posterolateral aspect of the thigh. Pain is described as deep, dull, and aching, and it often interferes with sleep.

Pain is provoked by lying on the side of the hip, stepping from curbs, and descending steps. Tenderness may be elicited over and adjacent to the greater trochanter. In contrast with true hip involvement, the Patrick *fabere* sign ( *f*lexion, *ab*duction, *e*xternal *r*otation, and *e*xtension) may be negative, and complete passive range of motion is relatively painless. Active abduction when the patient lies on the opposite side typically

intensifies the discomfort, and sharp external rotation may accentuate the symptoms. Hip radiographs may demonstrate a calcific deposit adjacent to the trochanter. [42] Trochanteric bursitis is a fairly frequent complication in rheumatoid arthritis. [43]

**Approach.**

Intrabursal injection uses the site of maximum tenderness for the entry point. A 3.9- to 5.0-cm, 20- or 21-ga needle is advanced until the needle tip reaches the trochanter. The needle is then withdrawn slightly, and the site is infiltrated fairly widely with 3 to 10 mL of lidocaine and 20 to 40 mg of prednisolone tebutate or the equivalent. The condition usually improves following 1 or 2 local injections.

**Ischiogluteal bursitis.**

"Weaver's bottom" is a painful

disorder characterized by pain over the center of the buttocks with radiation down the back of the leg. [44] This condition is rarely initially diagnosed, often being mistaken for lumbosacral strain, a herniated disk, or spinal cord tumor. When it is recognized, a skillful intrabursal injection, coupled with a few days' rest, usually relieves the extreme pain. The bursa is adjacent to the ischial tuberosity and overlies the sciatic and posterior femoral cutaneous nerves. Pain is provoked by sitting on hard surfaces, bending forward, and standing on tiptoe. Tenderness is present over the ischial tuberosity.

**Approach.**

The usual technique for injection requires that the patient lie in a prone position. A 5.0-cm, 20- to 22-ga needle is inserted through a skin wheal and is advanced cautiously in an effort to avoid the sciatic nerve, which lies at a depth of approximately 6.5 to 7.5 cm. Paresthesias occur on striking the nerve, and if this occurs, the needle should be withdrawn from the nerve. Generally, 5 to 10 mL of lidocaine and 20 to 40 mg of prednisolone suspension are introduced into the bursa.

**Knee Region**

**Prepatellar bursitis.**

"Housemaid's knee," or "nun's knee" is characterized by swelling with effusion of the superficial bursa overlying the lower pole of the patella. Passive motion is fully preserved, and pain is generally mild, except during extreme knee flexion or direct pressure. Although the disorder is usually caused by pressure from repetitive kneeling on a firm surface ("rug cutter's knee"), rarely it can develop after direct trauma, and occasionally it is a manifestation of rheumatoid arthritis. The prepatellar bursa is also a common site of septic bursitis.

**Approach.**

Aspiration often yields a surprisingly scant amount of clear, serous fluid, owing to the fact that the prepatellar bursa is a multilocular structure rather than the usual single cavity. The instillation of 1 to 2 mL of lidocaine with 15 to 20 mg of a prednisolone suspension with a 2.5-cm, 20- to 21-ga needle is usually sufficient to cause the swelling to abate. In some cases the procedure may need to be repeated several times to obtain a lasting result. The provocative activity should be discontinued.

**Suprapatellar bursitis.**

Suprapatellar bursitis usually is associated with synovitis of the knees. On occasion the bursa is largely separated from the synovial cavity, with only a very minor communication, and the swelling and effusion are chiefly confined to the suprapatellar area. This may be traumatic in origin or an associated manifestation of an inflammatory arthropathy.

**Approach.**

The procedure for aspiration and injection of the suprapatellar area is similar to that for the knee (see Chapter 57) .

**Anserine bursitis.**

"Cavalryman's disease" now mainly occurs in heavy women with disproportionately large thighs in association with osteoarthritis of the knee. The bursa is on the anteromedial side of the knee, inferior to the joint line at the site of the insertion of the conjoined tendons of the sartorius, semitendinous, and gracilis and superficial to the medial collateral ligament. The entity is characterized by a relatively abrupt onset of knee pain, with localized tenderness and a puffy sensation in the vicinity of the anserine bursa.

**Approach.**

An injection of 2 to 4 mL of lidocaine with or followed by approximately 20 to 40 mg of a corticosteroid suspension is given at the point of greatest tenderness, using an anterior or medial approach with a 2.5- to 3.9-cm, 22-ga needle (Fig. 56-19) . Prompt symptomatic relief frequently is obtained, but the duration of benefit is variable and probably correlates with the patient's weight-bearing activities.

**Ankle, Foot, and Heel Region**

**Ankle tendinitis.**

This is a relatively uncommon condition. It may result from unusual repetitive activity or, rarely, from acute trauma. Crepitant swollen tendon sheaths commonly occur in rheumatoid arthritis. The disorder is differentiated from ankle joint involvement by the lack of pain or restricted motion during passive flexion and extension of the ankle. Active flexion and extension of the toes does produce pain. Local tenderness is elicited along the flexor and extensor tendons. Injection of the tendon sheaths is useful, producing considerable relief of symptoms.

**Approach.**

A 2.5- to 3.9-cm, 22- or 25-ga needle is used. One makes a tangential entry to the enlarged tendon sheath, distending the sheath with approximately 2 to 4 mL of a mixture of corticosteroid and lidocaine and instilling 20 to 40 mg of prednisolone tebutate. It may be necessary to repeat the injection after several months.

**Bunion bursitis.**

It is common for bunion bursitis to overlie the first metatarsophalangeal joint at its medial surface on the great toe. On occasion, tense swelling occurs, and decompression is required. Aspiration with culture of the fluid should be performed.

**Approach.**

If no infection is present, the bursa is injected with 5 to 10 mg of prednisolone suspension with a 2.5-cm, 20-ga needle. Special shoes or an orthopedic correction will be needed if the swelling recurs.

**Heel pain.**

"Talalgia" may be caused by Achilles tendinitis, calcaneal bursitis, or plantar fasciitis. The bursae of clinical significance around the heel include the space between the skin and the Achilles tendon, the retrocalcaneal bursa, and the subcalcaneal bursa. Achilles tendinitis or bursitis may be traumatic in origin but is more apt to be part of a systemic disease, such as rheumatoid or gouty arthritis. Although the normal Achilles tendon is thick and strong, when affected by an inflammatory arthropathy, it is predisposed to degeneration, and since the Achilles tendon is not invested by a full synovial sheath, it is more vulnerable to intratendon instillation. Because of the potential hazard of tendon rupture after local steroid injection, it is wise to avoid infiltration of steroids into this area. [45] It is preferable to treat Achilles tendinitis with rest, splinting, and oral medication. The major injectable condition in this region is calcaneal bursitis (plantar fasciitis), which is frequently associated with painful heel spurs ("policeman's heel" or "soldier's heel"). If orthopedic shoe corrections and aids are not effective, injection of the painful heel is sometimes beneficial.

**Approach.**

At the spot of maximal tenderness, a 2.5-cm, 22- to 24-ga needle enters the plantar surface at 90°, sliding into the space at the midpoint of the calcaneus. The tip of the needle lies in the aponeurosis of the attachment to the os calcis (Fig. 56-20) (Figure Not Available) . One milliliter of lidocaine and 10 to 20 mg of prednisolone tebutate are instilled. The injection may need to be repeated once or twice at an interval of 6 to 8 weeks.

## SEPTIC BURSITIS

Occasionally, bursitis may be caused by an acute bacterial infection of the bursal fluid and surrounding soft tissue. [5] [46] Septic bursitis is most common in the olecranon and prepatellar bursae, whereas infection of other bursae is exceedingly rare. [46] The infection is probably secondary to acute trauma resulting in direct penetration of the bursa by common skin pathogens, rather than by hematogenous spread. The patient frequently reports a history of minor trauma or is engaged in an occupation associated with sustained pressure on the knees or the elbows.

Septic bursitis is not associated with septic arthritis of the underlying joint, although rarely, adjacent bone may become involved. [47] Many patients have an underlying predisposition to infection (e.g., diabetes mellitus, alcoholism, uremia, or gout). Rarely, an infection may follow a bursal injection of corticosteroids.

In most cases the diagnosis of septic bursitis is obvious. The onset of pain and swelling may be quite rapid (over 8 to 24 hours), as compared with the more gradual onset of aseptic bursitis. The bursa is tense, swollen, and very painful. Pitting edema and classic cellulitis of the peribursal soft tissue may be present. In some cases it may be difficult to distinguish septic bursitis from trauma or other acute inflammatory disorders, such as acute gout or tenosynovitis. In questionable cases it is prudent to aspirate and culture the fluid and to treat with antibiotics and oral NSAIDs. Corticosteroid injection is withheld pending negative bacteriologic findings.

The diagnosis is confirmed by culturing bacteria from the bursal aspirate. Fluid is usually easily obtained from the tense bursa and (in the case of an established infection) is cloudy or grossly purulent. The white blood cell count of the fluid is 50,000 to 100,000 cells/mm3 or greater, and polymorphonuclear cells usually exceed 90%. Gram-positive organisms may be seen on Gram stain. The infecting organism is usually penicillin-resistant *S. aureus*, but streptococcal organisms may be isolated. Treatment of septic bursitis includes the use of antibiotics directed against penicillinase-producing *Staphylococcus,* splinting, hot soaks, and drainage of the bursa. Drainage may be adequately performed with one or more needle aspirations, but open incision and drainage may be required. Corticosteroid injections should not be performed in infected tissue. Outpatient therapy with oral antibiotics is generally acceptable.

## CONCLUSION

Local injection therapy for painful nonarticular rheumatic disorders is a relatively simple, safe, and effective form of treatment. The patient may experience rapid relief of pain and swelling and on occasion may return to work after a single injection. The response may be long lasting, as well as very gratifying. An additional benefit of injection therapy is that it may avoid surgical intervention in such soft tissue problems as carpal tunnel syndrome, digital tenosynovitis (de Quervain disease and trigger finger), and ganglia. [30]

The local introduction of a corticosteroid suspension should be carried out with due regard for any coexisting disease, such as diabetes mellitus and peptic ulcer. It is highly unlikely that these diseases would be provoked or aggravated after a single intrasynovial injection in usual therapeutic dosage. However, systemic "spill-over" and absorption may occur, depending on the size of the dose and the solubility of the preparation injected. Transient adrenal suppression may develop but rarely lasts longer than a few days.

# Chapter 57 - Arthrocentesis

**Georges C. Benjamin**

Arthrocentesis, the puncture and aspiration of a joint, is an acknowledged, useful procedure that is easily performed in the emergency department. It has been established as both a diagnostic and a therapeutic tool for a variety of clinical situations. Many physicians are wary of joint fluid aspiration because of a lack of experience and because of the fear of introducing infection. When performed properly, however, the procedure offers a wealth of clinical information and is associated with few complications. In the emergency department it is difficult to make an accurate assessment of an acutely painful, hot, and swollen joint without performing arthrocentesis.

## INDICATIONS AND CONTRAINDICATIONS

The indications for arthrocentesis include the following [1] :

1. Diagnosis of nontraumatic joint disease by synovial fluid analysis (septic joint or crystal-induced arthritis).
2. Diagnosis of ligamentous or bony injury by confirmation of the presence of blood in the joint. Arthrocentesis may be required to differentiate a traumatic joint effusion from an inflammatory process.
3. Establishment of the existence of an intra-articular fracture by the presence of blood with fat globules in the joint.
4. Relief of the pain of an acute hemarthrosis or a tense effusion. Although a minor hemarthrosis need not be drained, arthrocentesis not only reduces pain in large effusions, it also facilitates examination of an injured joint.
5. Local instillation of medications in acute and chronic inflammatory arthritides. Instillation of lidocaine into an injured joint also makes the initial examination of a traumatic injury much easier.
6. Obtaining fluid for culture, Gram staining, immunologic

   studies, and cell count in cases of suspected joint infection.

The most important contraindication to arthrocentesis is the presence of infection in the tissue overlying the site to be punctured (e.g., an abscess or a frank cellulitis). However, inflammation with warmth, swelling, and tenderness may overlie an acutely arthritic joint, and this condition may mimic a soft tissue infection. Once convinced that cellulitis does not exist, the clinician should not hesitate to obtain the necessary diagnostic joint fluid. A relative contraindication to joint puncture is the presence of a bacteremia. Not all joint infections following arthrocentesis are the result of poor antiseptic technique. The hematogenous spread of bacteria into the joint, with or without hemorrhage, in a bacteremic patient also may lead to infection.

Bleeding diatheses may at times be a relative contraindication, but arthrocentesis to relieve a tense hemarthrosis in bleeding disorders, such as hemophilia, is an accepted

practice following infusion of the appropriate clotting factors. Arthrocentesis is also relatively contraindicated in a patient receiving anticoagulants or in the presence of a joint prosthesis, unless the procedure is being performed to rule out infection.

## Articular Versus Periarticular Disease

Periarticular diseases such as tendinitis, bursitis, contusion, cellulitis, or phlebitis may mimic articular disease and suggest the need for arthrocentesis. Therefore, administration of the correct therapy for acute joint disease requires that the physician first determine whether the patient's constellation of signs and symptoms derives from the joint itself and not from some other musculoskeletal or periarticular structure.

If swelling is secondary to joint effusion or inflammation, then the entire articular capsule will be inflamed and distended, and fluid can often be palpated within the joint. In the knee, this condition must be differentiated from effusion into the prepatellar bursa, where swelling distends the bursa that lies mainly over the lower portion of the patella, between it and the skin. Effusion into the joint occurs posterior to the patella, whereas bursal swelling occurs anterior to it. When there is considerable articular effusion, the capsule of the joint is distended, and an inverted U-shaped swelling of the joint occurs. This characteristic shape occurs because the dense patellar ligament prevents distention of the capsule along its inferior border. Also, with the knee extended, a large effusion causes the patella to "float" or lift away from the femoral condyles. A sharp thrust of the fingers makes the patella click against the condyles, confirming the effusion. [2] Complete extension and flexion are often impossible because of the joint tension produced by the effusion.

Joint effusion causes limited movement of the joint in all directions with active and passive motion producing pain. The pain arising from a pathologic condition involving a joint may be diffuse or clearly localized to the joint, or it may radiate. Hip pain, for example, frequently radiates into the groin or down the front of the thigh into the knee. Shoulder joint pain also commonly radiates into the elbow or the neck. Because of this, complete examination of contiguous structures is essential for adequate diagnosis.

In contrast, pain from a periarticular process is often more localized, and tenderness can be elicited only with certain specific movements or at specific points around the joint. In periarticular inflammation, one can often passively lead a joint through a range of motion with minimal discomfort, yet pain is significant when the patient attempts active motion. Crepitus may be elicited in tendinitis, or the pain may be traced along the course of a specific tendon.

## Septic Arthritis

Acute monoarticular arthritis is a common problem in emergency medicine. Although there are many causes of acute monoarticular arthritis, the one most requiring urgent diagnosis and treatment is septic arthritis. Infectious arthritis is still relatively frequent, and suspicion of a septic process in the joint is the first step in appropriate management; confirmation requires arthrocentesis and synovial fluid culture. Although repeated arthrocentesis may be needed when treating a septic joint, such therapy is

usually performed on an inpatient basis.

Infection of a joint occurs by 1 of 4 mechanisms: hematogenous spread, spread from a contiguous source of infection, direct implantation, or postoperative contamination. The joint distribution of septic arthritis is typically monoarticular with a swollen, erythematous, and painful joint. Radiographically the differential diagnosis includes limited rheumatoid arthritis, gout, synovial osteochondromatosis, and pigmented villonodular synovitis. Early diagnosis is essential to prevent complications such as growth impairment, articular destruction with ankylosis, osteomyelitis, or soft tissue extension. [3]

Because an acutely swollen joint may be indicative of a number of disease entities, a thorough history and physical examination are the cornerstones of evaluation, followed by arthrocentesis. Laboratory findings can be useful in diagnosis, as can response to therapy (e.g., response to penicillin in gonococcal arthritis is often the only criterion for diagnosis, as the organism is difficult to culture). Patients with malignancy (especially leukemia) or who are immunosuppressed or otherwise debilitated are at particular risk for a septic etiology. Infectious arthritis should be the first potential cause looked for in these patients, as well as in patients with such preexisting joint diseases as rheumatoid arthritis. A swollen joint is usually not injected with corticosteroids until the possibility of infection has been eliminated. [4]

*Neisseria gonorrheoae*, *Staphylococcus,* and *Streptococcus* are the most frequently identified etiologic agents. [3] *N. gonorrheoae* is the most common organism causing septic arthritis among adolescents and young adults. [5] Patients >40 years old and those with other medical illnesses are more likely to have *Staphylococcus* joint infection, whereas infants aged 6 months to 2 years show a higher incidence of *Haemophilus influenzae*. In neonates, staphylococci and *Escherichia coli* predominate. [6] Intravenous drug abusers commonly develop staphylococcal or *Pseudomonas* infections.

Although precise incidences for nongonococcal septic arthritis have not been established, predisposing factors have been described. These include chronic debilitating disease; prior antibiotic or immunosuppressive medications; a previous history of joint damage, such as from rheumatoid arthritis or degenerative joint disease; and prosthetic joints. [7] [8] [9] In a study by Sharp and coworkers, 22 of 113 patients (19%) with septic arthritis had other arthritides, the most common being rheumatoid arthritis. [3] Elderly patients with a debilitating

arthritis and minor skin infections seem to be the most susceptible; the infection may be overlooked and the patient's findings thought to be an exacerbation of the rheumatoid arthritis. [10]

The simultaneous occurrence of gout and septic arthritis may be more common than generally recognized. One should not allow the establishment of a diagnosis of crystal-induced disease to stop a thorough search for infection. [11]

Because *N. gonorrhoeae* is the most common organism causing septic arthritis, gonococcal arthritis deserves special mention. The incidence in men is increasing, but

the majority of disseminated gonococcal infections occur in women with asymptomatic anogenital infections. Dissemination usually occurs during menstruation or pregnancy.

A useful context in which to view gonococcal arthritis was presented by Gelfand and coworkers. [14] Three sequential stages with associated clinical subgroups have been described, although in practice fewer than 50% of patients will fit classically into 1 of the 3 stages. Patients in the first group *(hematogenous phase)* have constitutional symptoms (high fever and chills), polyarthralgias-polyarthritis (but with little effusion), tenosynovitis, and dermatitis. They then enter a second stage *(transitional phase)* with skin lesions; positive blood cultures; and occasionally, positive joint fluid cultures in an early arthritis and effusion. Those in the third stage *(joint localization phase)* do not have systemic symptoms or skin lesions. The infection settles into 1 or 2 large joints, yielding a purulent arthritis.

Whereas *N. gonorrhoeae*-infected joint fluid is usually "septic" in character, the yield of positive synovial fluid cultures has ranged from 25% to 60%. [15] It is rare to simultaneously find positive joint fluid cultures and blood cultures. [5] A positive Gram stain is immediately diagnostic of septic arthritis. Gram staining is positive in only 65% of cases of septic arthritis, and therefore a negative Gram stain does not rule out an infectious process. A white blood cell count and a synovial fluid glucose reduction may give confirmatory data. Counterimmunoelectrophoresis and latex agglutination have been evaluated for early identification of infection.

**Hemarthrosis**

Isolated nontraumatic hemarthrosis may occasionally be seen by the emergency physician. An inflammatory reaction may follow an intracapsular bleed, and the proliferative reaction and the hyperplastic synovium formed may predispose the patient to recurrent hemorrhage in that joint, especially patients with bleeding diatheses. The knee is the most commonly affected joint (whether the cause is hemophilia or oral anticoagulants), followed by the foot and the ankle. [17]

The most common cause of intra-articular hemorrhage in the setting of no trauma or minor trauma is a hereditary clotting factor deficiency, as in hemophilia. Hemarthrosis is an infrequent complication of oral anticoagulant therapy but may occur even with prothrombin times within the normal range. [18] Chronic arthritis does not appear to be a long-term complication in patients with intra-articular bleeds from oral anticoagulant therapy. Hemarthrosis may also be a complication of sickle cell anemia, pseudogout, amyloidosis, pigmented villonodular synovitis, synovial hemangioma, rheumatoid arthritis, and infection. [17]

Management of acute hemarthrosis depends on the cause. Studies by Jaffer and Schmid and Wild and Zvaifler showed that the synovitis associated with oral anticoagulant therapy improves only after the oral anticoagulant is discontinued and the prothrombin time returns to normal. Hemarthrosis following trauma is a frequent occurrence. It is most common in the knee and often denotes significant internal damage.

Distention of the joint by effusion or hemorrhage causes considerable pain and disability. If the exudate or the blood is not removed, it is partly absorbed, but part of it may undergo organization, resulting in formation of adhesions or bands in the joint. This is one argument for drainage of the joint. [2] Some believe that in an otherwise healthy joint that is subjected to a single traumatic event, even a relatively large hemarthrosis will be spontaneously resorbed without significant sequelae and therefore presents no pressing need to drain. This remains an area of controversy.

Nonetheless, a large, tense traumatic effusion is quite painful, and its presence precludes proper evaluation of an injured joint. Therapeutic arthrocentesis to drain a symptomatic traumatic effusion is a well-accepted practice. [20] The source of blood following trauma is frequently (1) a tear in a ligamentous structure, knee capsule, or synovium or (2) a fracture. Cruciate (especially anterior) ligament injury is the most common cause of significant hemarthrosis following trauma to the knee. [21] A joint effusion that develops 1 to 5 days following trauma may be secondary to a slow hemorrhage or a reinjury, but the swelling is often caused by a nonhemorrhagic irritative synovial effusion.

Occasionally one will diagnose an occult fracture by the presence of fat globules in the arthrocentesis blood. This may be appreciated when the bloody effusion is placed in a clear container (e.g., test tube or specimen jar) and held to the light. If the history of trauma is vague, arthrocentesis may be required to differentiate hemorrhage from other causes of joint effusion. Following therapeutic arthrocentesis for a hemarthrosis, it may be desirable to inject 2 to 15 mL of a local anesthetic, depending on joint size, into the joint to facilitate examination or provide temporary symptomatic relief. [20]

**Intra-articular Corticosteroid Injections**

In 1951 Hollander and coworkers first demonstrated that intra-articular corticosteroid injections were useful for symptomatic relief in patients with severe rheumatoid arthritis. [22] The use of steroids has proved to be a dependable method for providing rapid relief from pain and swelling of inflamed joints, although it is strictly local, usually temporary, and rarely curative.

Corticosteroid injections are most helpful when only a few of a patient's joints are actively inflamed. The most frequently used corticosteroids for intra-articular injection are shown in Table 57-1 (Table Not Available) . [12] Diminution of joint pain, swelling, effusion, and warmth is usually evident within 6 to 12 hours after injection.

The most serious complication of this practice is intra-articular infection. Therefore, steroids should not be injected into a joint if there is suspicion of a joint space infection. Repeated injections into 1 joint carry the risk of necrosis of juxta-articular bone with subsequent joint destruction and instability. Other complications include local soft tissue atrophy and calcification, tendon rupture, intra-articular bleeding, and transient nerve palsy. [24] Deposition of steroid crystals on the synovium may give rise to a transient,

self-limited flare-up of a synovitis. [25]

It is always important to ascertain whether local corticosteroid therapy has been used previously, not only to consider the array of clinical conditions associated with steroid use, but also because crystalline corticosteroid material can hinder proper interpretation of crystals found in synovial fluid.

## EQUIPMENT

Necessary materials for arthrocentesis include skin preparation solutions (usually povidone-iodine followed by alcohol), sterile gloves and drapes, local or topical anesthetics, a syringe, and various-sized needles (Table 57-2) . Fluid for cell count should be collected in a lavender-topped tube with ethylenediaminetetra-acetic acid (EDTA) anticoagulant; however, glucose and viscosity determinations do not require

| **TABLE 57-2** -- Emergency Department Arthrocentesis Tray |
|---|
| Gloves |
| Surgical scrub or plain green soap |
| Povidone-iodine solution |
| Alcohol sponges |
| Sterile towel (with center perforation) |
| Vapor coolant (e.g., fluoromethane solution) |
| 1% or 2% lidocaine |

| |
|---|
| Sterile saline for injection |
| Sterile gauze dressings (2 in. × 2 in.) |
| Sterile syringes (2 mL, 10 mL, 20 mL) Luer-Lok |
| Needles (18, 20, 22, and 25 ga) or over-the-needle intravenous catheters |
| Hemostat |
| Three-way stopcock (optional) |
| Plastic adhesive bandages |
| Sterile basin |
| Plain test tubes |
| Test tubes with liquid anticoagulant |
| Microscope slides and coverslips |
| Clear nail polish (to seal coverslip over material) |

anticoagulants. One should immediately examine fresh synovial fluid in its unadulterated form for crystals. Calcium oxalate and lithium heparin anticoagulants have been

reported to introduce artifactual crystals into the fluid. Joint fluid to be analyzed for crystals should be collected in a green-topped tube containing sodium heparin. If one is culturing for *N. gonorrhoeae*, the fluid should be immediately placed on proper medium and stored in a low-oxygen environment in the emergency department.

## GENERAL ARTHROCENTESIS TECHNIQUE

Joint fluid may be obtained even when there is little clinical evidence of an effusion. Although one may successfully aspirate where the joint bulges maximally, certain landmarks are important. The most crucial part of arthrocentesis is spending adequate time in defining the joint anatomy by palpating the bony landmarks as a guide. These are described in detail later in this chapter. A puncture site and an approach to the joint should be selected; tendons, major vessels, and major nerve branches should be avoided. In most instances, the approach is via the extensor surfaces of joints, because most major vessels and nerves are found in flexor surfaces. Also, the synovial pouch is usually more superficial on the extensor side of a joint.

Aseptic technique is essential to avoid infection. Only sterile instruments should be used. *Arthrocentesis should not be attempted if the skin or subcutaneous tissue overlying the joint is infected.* Sterile gloves should be worn for the remainder of the preparation. The skin is thoroughly scrubbed with a surgical scrub, such as povidone-iodine scrub, and the skin is painted with an iodinated solution, such as povidone-iodine (Betadine). This antiseptic solution should be applied 3 times, allowing it to dry for several minutes, because the bactericidal effects of iodine are both concentration and time dependent. The iodine solution is then removed with an alcohol sponge to prevent transference of iodine into the joint space with a resultant inflammatory process. Gloves should be changed after preparing the skin. Generally a sterile perforated drape is placed over the joint; alternatively, sterile cloth drapes can be used.

With appropriate local anesthesia, arthrocentesis should be a relatively painless procedure; without anesthesia, it may be quite painful and distressing to the patient. The synovial membrane itself has pain fibers associated with blood vessels, and the articular capsule and bone periosteum are richly supplied with nerve fibers and are very sensitive. The articular cartilage has no intrinsic pain fibers. It is important to have the patient relax during the procedure. Tense muscles narrow the joint space and make the procedure more difficult, requiring repeated attempts at aspiration, or result in inadequate drainage. Distraction of the joint may enhance the target area, especially in areas such as the wrist and the finger joints. Traction not only increases the chance of entering the joint, it also lessens the chance of scoring the articular cartilage with the needle.

Using a vapor coolant topically and then rapidly inserting the needle through the skin minimizes patient discomfort. Alternatively, one can infiltrate the skin down to the area of the joint capsule using a local anesthetic agent such as 1% or 2% lidocaine (Xylocaine) with a 22- or 25-ga needle before performing arthrocentesis. For extremely painful

joints, a regional nerve block before the procedure (see Chapter 32) is often greatly appreciated by the patient.

The landmarks described under Specific Procedures should be used, and care should be taken not to bounce the needle off bony structures as a means of finding the joint space, because this may damage the cartilage. [26] An 18- to 22-ga needle or IV catheter and needle set of appropriate length attached to a syringe is inserted at the desired anatomic point through the skin and subcutaneous tissue into the joint space. The largest needle that is practical is used to avoid obstructing the lumen with debris or clot. In large joints such as the knee, which can accommodate large effusions, it is suggested that one use a 30- to 55-mL syringe, because it may be difficult to change a syringe when the needle is within the joint cavity. A three-way stopcock placed between the needle and syringe is an option for draining large effusions. If the syringe must be changed during the procedure, the hub of the needle should be grasped with a hemostat and held tightly while the syringe is removed. If an IV catheter and needle set is used, the needle is removed, leaving the outer atraumatic plastic catheter in the joint space. The syringe is then attached to the catheter for aspiration. Now manipulation of the joint or catheter can occur with little threat of tissue injury.

Aspiration of synovial fluid and the easy injection and return of fluid indicate intra-articular placement of the needle tip. As a general rule, *one should completely remove all fluid or blood.* If the fluid stops flowing, this is a sign that the joint has been drained completely, the needle tip has become dislodged, or debris or clot is obstructing the needle. One should slightly advance or retract the tip of the needle, rotate the bevel, or ease up on the force of aspiration. Occasionally, reinjecting a small amount of fluid back into the joint space confirms the needle placement and may clear the needle. If fluid flows freely back into the joint and is easily reaspirated, one has probably removed all the fluid. If resistance is met, the needle has probably been jarred from the joint space and is lodged in the soft tissue. In some instances, minor position changes produced by flexion or extension of the joint may allow the fluid to flow more freely. Scraping or shearing the articular cartilage with the needle should be avoided, since this may produce permanent cartilage damage. One should enter the joint in a straight line and avoid unnecessary side-to-side motion of the syringe. After aspiration is complete, the needle is removed, and a sterile dressing is applied over the puncture site.

Synovial fluid should be sent for studies as indicated by the clinical situation. Studies usually obtained include cell count with differential, crystal analysis, Gram staining, bacterial culture and sensitivity analysis, and synovial fluid glucose measurement. Less frequently obtained studies include synovial fluid protein measurement, rheumatoid factor analysis, lupus erythematosus (LE) cell preparation, viscosity analysis, mucin clot, fibrin clot, fungal and acid-fast stains, fungal and tuberculous culture, and synovial fluid complement analysis. If the arthrocentesis is performed for the relief of a hemarthrosis, the fluid need not be sent for analysis. One should be selective in ordering tests. There is no need to order a large battery of tests routinely on all fluids.

If the volume of fluid collected is low, culture and examination of the "wet prep" under

regular and polarizing microscopy have the highest priority.

To avoid misdiagnosing borderline inflammatory fluids, missing crystals that dissolve with time, or overinterpreting the findings because of new artifactual crystals that appear over a prolonged time, prompt examination of synovial fluid specimens should be performed.

## COMPLICATIONS

Significant complications are rare with arthrocentesis and include the following:

1. *Infection.* Skin bacteria may be introduced into the joint space during needle puncture. One can, of course, limit this complication by maintaining rigorous sterile technique and avoiding inserting the needle through obviously infected skin or subcutaneous tissue. The chance of introducing infection with arthrocentesis through a noninfected area is minimal if proper attention is paid to technique. Various studies report the incidence of infection following routine arthrocentesis to be in the range of 1 per 10,000 aspirations. [28] Joint aspiration in the presence of a bacteremia has been discussed previously.
2. *Bleeding.* Bleeding with subsequent hemarthrosis is rarely a complication, except in the patient with a bleeding diathesis. If a patient has a bleeding diathesis, such as hemophilia, arthrocentesis should be delayed until clotting competence has been enhanced by the infusion of specific clotting factors. Occasionally, a small quantity of blood may be aspirated along with the synovial fluid. This happens most often when the joint is nearly emptied. A small amount of blood-tinged fluid is generally the result of nicking a small synovial blood vessel; this is usually inconsequential. A grossly bloody effusion must be investigated. [4]
3. *Allergic reaction.* Hypersensitivity to the local anesthetic that is used can usually be prevented by thorough history taking. Fainting during the procedure is not uncommon and is the result of vasovagal influences.
4. *Corticosteroid-induced complications.* See Intra-articular Corticosteroid Injections.

## SPECIFIC ARTHROCENTESIS TECHNIQUES

### First Carpometacarpal Joint (Fig. 57-1) (Figure Not Available)

**Landmarks.**

The radial aspect of the proximal end of the first metacarpal is the arthrocentesis landmark for this joint. The abductor pollicis longus tendon is located by active extension of the tendon.

**Position.**

The thumb is opposed against the little finger so that the proximal end of the first metacarpal is palpable. Traction is applied to the thumb in order to widen the joint space

between the first metacarpal and the greater multangular bone.

**Needle insertion.**

A 22-ga needle is inserted at a point proximal to the prominence at the base of the first metacarpal, on the palmar side of the abductor pollicis longus tendon.

**Comments.**

Degenerative arthritis commonly affects this joint. Arthrocentesis is of moderate difficulty. The anatomic "snuff box" (located more proximally and on the dorsal side of the abductor pollicis longus tendon) should be avoided, because it contains the radial artery and superficial radial nerve. A more dorsal approach may also be used.

## Interphalangeal and Metacarpophalangeal Joints (Fig. 57-2) (Figure Not Available)

**Landmarks.**

The landmarks are on the dorsal surface--the prominence at the proximal end of the proximal phalanx for metacarpophalangeal joints and the prominence at the proximal end of the middle or distal phalanx for interphalangeal joints. The extensor tendon runs down the midline.

**Position.**

The fingers are flexed to approximately 15° to 20° and *traction is applied.*

**Needle insertion.**

A 22-ga needle is inserted into the joint space dorsally, just medial or lateral to the central slip of the extensor tendon.

**Comments.**

Synovitis causes these joints to bulge dorsally. Normally, it is unusual to obtain fluid in the absence of a significant pathologic condition.

## Radiocarpal Joint (Wrist) (Fig. 57-3) (Figure Not Available)

**Landmarks.**

The dorsal radial tubercle (Lister tubercle) is an elevation found in the center of the dorsal aspect of the distal end of the radius. The extensor pollicis longus tendon runs in a groove on the radial side of the tubercle. The tendon can be palpated by active

extension of the wrist and thumb.

**Position.**

The wrist should be positioned in approximately 20° to 30° of flexion and accompanying *ulnar* deviation. *Traction is applied to the hand*.

**Needle insertion.**

A 22-ga needle is inserted dorsally, just distal to the dorsal tubercle and on the ulnar side of the extensor pollicis longus tendon. The anatomic snuff box, located more radially, should be avoided.

## Radiohumeral Joint (Elbow) (Fig. 57-4) (Figure Not Available)

**Landmarks.**

The lateral epicondyle of the humerus and the head of the radius are the arthrocentesis landmarks for the radiohumeral joint. With the elbow extended, the depression between the radial head and the lateral epicondyle of the humerus is palpated.

**Position.**

With the palpating finger still touching the radial head, the elbow is flexed to 90°. The forearm is pronated, and the palm is placed down flat on a table.

**Needle insertion.**

A 22-ga needle is inserted from the lateral aspect just distal to the lateral epicondyle and is directed medially.

**Comments.**

Effusions in the elbow joint may bulge and be readily palpated. Often the effusion appears inferior to the lateral epicondyle. The bulge can then be aspirated from a posterior approach on the lateral side. A medial approach should not be used, because the ulnar nerve and the superior ulnar collateral artery may be damaged. This is a common joint for gout or septic arthritis. A small hemarthrosis need not be aspirated.

## Glenohumeral Joint (Shoulder), Anterior Approach (Fig. 57-5) (Figure Not Available)

**Landmarks.**

The coracoid process medially and the proximal humerus laterally are palpated

anteriorly.

**Position.**

The patient should sit upright with the arm at the side and his or her hand in the lap.

**Needle insertion.**

A 20-ga needle is inserted at a point inferior and lateral to the coracoid process and is directed posteriorly toward the glenoid rim.

**Comments.**

Arthrocentesis of this joint is of moderate difficulty. Other approaches have been suggested but are less well accepted.

## Knee Joint, Anteromedial Approach (Fig. 57-6 (Figure Not Available) *A*)

**Landmarks.**

The medial surface of the patella at the middle or superior portion of the patella is the landmark for the knee joint.

**Position.**

The knee is fully extended as far as possible. Relaxation of the quadriceps muscle greatly facilitates needle placement. The foot is kept perpendicular to the floor.

**Needle insertion.**

An 18-ga needle or catheter and needle set is inserted at the midpoint or superior portion of the patella approximately 1 cm medial to the anteromedial patellar edge. The needle is directed between the posterior surface of the patella and the intercondylar femoral notch. The patella may be grasped with the hand and elevated (see Fig. 57-6 (Figure Not Available) *B*).

**Comments.**

If the patient is tense, contraction of the quadriceps will greatly hinder entering the joint. However, the knee is probably the easiest joint to enter. Removal of a tense hemarthrosis will relieve pain and facilitate examination for ligamentous injury. If fluid stops flowing, one should *squeeze the soft tissue area of the suprapatellar region to "milk" the suprapatellar pouch of fluid*. The knee can easily accommodate 50 to 70 mL of fluid, and the clinician should therefore be prepared to change syringes during the procedure. The knee is a common site for septic arthritis (especially gonococcal) and

various inflammatory or degenerative diseases. An anterolateral approach can be accomplished in a similar manner if the patella is approached laterally.

## Tibiotalar Joint (Ankle) (Fig. 57-7) (Figure Not Available)

**Landmarks.**

The medial malleolar sulcus is bordered medially by the medial malleolus and laterally by the anterior tibial tendon. The tendon can be easily identified by active dorsiflexion of the foot.

**Position.**

With the patient lying supine on the table, the foot is plantar flexed.

**Needle insertion.**

A 20-ga needle is inserted at a point just medial to the anterior tibial tendon and directed into the hollow at the anterior edge of the medial malleolus. The needle has to be inserted 2 to 3 cm to penetrate the joint space.

**Comments.**

If the joint bulges medially, one may use an approach that is more medial than anterior, entering at a point just anterior to the medial malleolus. The needle may have to be advanced 2 to 4 cm with this approach.

## Metatarsophalangeal and Interphalangeal Joints (Fig. 57-8) (Figure Not Available)

**Landmarks.**

For the first digit, landmarks are the distal metatarsal head and the proximal base of the first phalanx. For the other toes, the landmarks are the prominences at the proximal interphalangeal and distal interphalangeal joints. The extensor tendon of the great toe can be located by active extension of the toe.

**Position.**

With the patient supine on the table, the toes should be flexed 15° to 20°. *Traction is then applied.*

**Needle insertion.**

A 22-ga needle is inserted on the dorsal surface at a point just medial or lateral to the

central slip of the extensor tendon.

## SYNOVIAL FLUID INTERPRETATION

Synovial fluid examination is essential for the diagnosis of septic arthritis, gout, and pseudogout. Inflammatory joint disease of previously unknown etiology can often be diagnosed precisely by synovial fluid studies. Joint fluid is a dialysate of plasma that contains protein and hyaluronic acid. Normal fluid is clear enough to allow newsprint to be read through it, and it will not clot. Normal fluid is straw colored and flows freely, with the consistency of motor oil. Normal fluid produces a good mucin clot and gives a positive "string sign" (see next section). The uric acid level of joint fluid approaches that of serum, and the glucose concentration is normally at least 80% that of serum. A normal joint contains only a few milliliters of fluid. Clarity of fluid reflects the leukocyte count. High leukocyte counts result in opacity, the degree of which generally correlates with the degree of elevated synovial fluid leukocytes.

### String Sign

Viscosity correlates with the concentration of hyaluronate in the synovial fluid. Any inflammation degrades hyaluronate, characteristically resulting in low-viscosity synovial fluids. The string sign is a simple test for assessing viscosity. The

practitioner measures the length of the "string" formed by a falling drop extruded from a syringe of synovial fluid. Normal joint fluid produces a string of 5 to 10 cm (Fig. 57-9) . If viscosity is reduced, as in inflammatory conditions, the synovial fluid forms a shorter string or falls in drops.

### Mucin Clot Test

The mucin clot test also corresponds to viscosity and inflammation. Therefore, the greater the inflammatory response, the poorer the mucin clot and the lower the viscosity. The mucin clot test is useful to define the degree of polymerization of hyaluronate. Mucin clots are produced by mixing 1 part joint fluid with 4 parts 2% acetic acid. A good clot indicates a high degree of polymerization and correlates with normal high viscosity. In inflammatory synovial fluid, such as that seen in osteoarthritis and rheumatoid arthritis-related effusions, the mucin clot is poor. [29]

### Cell Count and Glucose

A leukocytosis consisting predominantly of neutrophils is usually seen with inflammatory arthritides; a white blood cell count >50,000/mm3 (i.e., >50,000/muL) is highly suggestive of a septic joint. Joint fluid glucose usually decreases as inflammation increases, but a proper interpretation requires a simultaneous blood glucose evaluation. A joint fluid-to-serum glucose ratio of <50% suggests a septic joint. Shmerling and coworkers [30] have found a white blood cell count of >2000/mm3 to be 84% sensitive and 84% specific for *all* inflammatory arthritides. Of their septic arthritis patients, 37% had a total white blood cell count <50,000/ mm3 . However, 89% of their patients with a

total white blood cell count >50,000/mm3 had a septic joint.

## Serology

The detection of succinic acid is helpful in identifying patients with septic arthritis who have received antibiotic treatment before arthrocentesis. [31] Gas-liquid chromatography, a rapid and sensitive method for the detection of short-chain fatty acids, may complement the currently available methods used to diagnose septic arthritis of synovial fluid. [32] Counterimmunoelectrophoresis and latex agglutination also are useful and are available in some centers on an emergency basis. Other immunologic markers such as complement, rheumatoid factor, and antinuclear antibodies have little diagnostic value in the acute setting but may be useful when compared to serum levels.

## Fluid Processing

Proper collection of the joint fluid is essential for examination and testing. Tests for viscosity, serology, and chemistries are done on fluid collected in a red-topped (clot) tube, whereas cytology samples are collected in tubes with an anticoagulant (purple top). One should always transfer the fluid for crystal examination into a tube with liquid heparin (green top), because undissolved heparin crystals from powdered anticoagulant tubes can be seen on microscopy. Early transfer of synovial fluid to this green-topped tube is essential to prevent clotting. Culture requirements for transport and processing should be accessed before the procedure to ensure appropriate processing or plating of specimens. A useful data form and summary of specimens developed by Alexander Trott, MD, of the University of Cincinnati is shown in Figure 57-10 (Figure Not Available) .

## Polarizing Microscope

No synovial fluid analysis is complete until the fluid has been examined under a polarizing light microscope for crystals. Microscopic analysis requires a compensated polarized light microscope with 10×, and 100× (oil immersion) objectives; a phase condenser and an oil immersion phase objective are highly recommended (Fig. 57-11) . The polarizing microscope used for crystal identification differs from the ordinary light microscope because it contains two identical polarizing prisms or filters. [33] One filter, called the *polarizer,* is positioned below the condenser. The other filter is called the *analyzer* and is inserted at some point above the objective. In some instruments these filters are mounted to permit horizontal rotation of the polarizer and analyzer. The mechanical stage of the polarizing microscope is mounted on a circular stage that rotates on a vertical axis coinciding with the center of the field; this allows the objective lens to be centered, keeping the specimen in the field of view. A removable first-order red compensator is located between the objective and the analyzer. All lenses and objectives are of "strain-free" (nonpolarizing) glass. Although these microscopes can be expensive, relatively inexpensive ones can be used and are quite adequate.

One can also obtain polarizing filters for insertion in a regular light microscope. One filter is placed between the light source and condenser; another is placed above the objective or in the eyepiece. The filters are rotated until a black field is obtained. This produces

the white birefringence that shows crystals more easily than ordinary light but cannot separate positive and negative birefringence.

## Polarization Physics

The polarizer allows passage of light in only 1 specific orientation. The analyzer acts as a crossed filter, removing all light in the light path unless the material being examined rotates the beam from the polarizer into the plane of the analyzer. The compensator functions by imparting color of a certain wavelength (red at about 550 nm). Birefringent materials change the wavelength to blue or yellow, depending on the direction (negative or positive) of refringence.

## Preparation of the "Wet Prep"

To prepare the "wet prep" for polarized microscopy, a drop of fluid is put on a clean slide, and a small coverslip is placed over the fluid. Sedimented or centrifuged "button" material is best, although any part of the specimen that ensures the drop contains cells, cellular debris, or unidentified material likely to contain crystals may be used. Liquid heparin should be the anticoagulant used, because undissolved powdered (crystalline) anticoagulants may appear as crystals under the microscope. Clear nail polish can be used to seal the coverslip over a specimen used for crystal analysis. This procedure preserves the slide and arrests fluid motion. The specimen is examined as noted subsequently.

## Viewing Techniques

Long periods of scanning can cause fatigue, frustration, and inaccurate observations. This can be prevented by appropriate viewing techniques. The most effective initial approach to a slide is scanning under low power (10✕) with compensated polarized light. One should vary the light intensity and adjust the condenser iris for better vision. The field should not be too light, because this will cause the crystals to be "washed out." With the naked eye, the test material or a bubble is placed on the stage in the center of the light path. If a small amount of specimen is present, this may be difficult to do. One approach is to place an ink dot on the underside of the slide and focus on this first, and then focus on the test material. If focusing to the correct depth is still difficult, one can focus on the edge of the coverslip. Frequently, trapped air bubbles, streaming cells, areas of increased cell concentration, or precipitated nail polish that is refractile along this edge will be of assistance with focusing.

Once focused on the slide's upper surface, scanning is performed under low power, looking for a cluster of cells and fibrils. Areas of clumping are scrutinized for regularly shaped material in or about the cells by rotating the microscope stage 90° back and forth while fine-focusing slightly up and down. The crystal length may be up to one tenth the diameter of a white blood cell. Thinking small will help identify crystals promptly.

To use the polarizer for crystal identification, one must use the compensator and adjust the light source and mirror to maximum light intensity. Both the polarizer and analyzer

are then engaged by pulling to the far right and adjusting both to 90°. The slide to be examined is placed on the stage and focused as noted earlier. At this point the background should be dark and the crystals bright white. If the background is not dark, the polarizer and the analyzer are adjusted as before. Crystals (preferably ones within leukocytes) are centered on the crosshairs. The compensator is engaged with lettering up. At this point the background should be red. Note that the X and Z axis are marked on the compensator. The stage is rotated so that the long axis of the crystal is parallel to the Z axis (the axis of slow vibration of the compensator). The color of the crystal is noted; the stage is rotated 90°, and the change in color of the crystal is again noted.

Although high dry magnification (40×) may confirm crystal identification, a complete examination should include evaluation under oil immersion (100× to 12.5× eyepiece or a total magnification of 100× to 1250× will achieve an optimal two-point discrimination of 0.5 to 1.0 mm). Often a fluid that initially appears to have only a few crystals or none at all is found to have a large population of small crystals or a second, different crystal population.

Thorough searching of many cellular clumps with a variation of light and condenser settings may be needed to find the thin, flat crystals of intracellular calcium pyrophosphate. A phase oil immersion objective (100×) is helpful in this situation. Once one crystal is identified, others are sought to confirm the findings. One should estimate the number of crystals of each type that are present as "few," "moderate," or "many" and what percentage are intracellular and extracellular. A systematic scan is necessary for this until one is satisfied that a fair estimate is possible. These data are helpful and relate to the acuteness of the arthritic process.

| TABLE 57-3 -- Synovial Fluid Interpretation | | | | |
|---|---|---|---|---|
| Diagnosis | Appearance | WBCs/mm3 | Polymorphonuclear Leukocytes | Glucose: % Blood Level |
| Normal | Clear | <200 | <25 | 95-100 |
| Degenerative joint disease | Clear | <4000 | <25 | 95-100 |

| | | | | |
|---|---|---|---|---|
| Traumatic arthritis | Straw-colored, bloody,xanthochromic,occasionally with fat droplets | <4000 | <25 | 95-100 |
| Acute gout | Turbid | 2000-50,000 | >75 | 80-100 |
| Pseudogout | Turbid | 2000-50,000 | >75 | 80-100 |
| Septic arthritis | Purulent/turbid | 5000->50,000 | >75 | <50 |
| Rheumatoid arthritis/ seronegative arthritis (Reiter disease, psoriatic arthritis, ankylosing spondylitis, inflammatory bowel disease) | Turbid | 2000-50,000 | 50-75 | 75 |

W BC, White blood cell.

* Negative birefringence means that crystals appear yellow when lying parallel to the axis of the slow vibration of light of the first-order red compensator. With the same orientation to the compensator, positive birefringence crystals appear blue. When the crystals lie perpendicular to the axis, the opposite is true--that is, negative birefringence crystals are blue, and positive ones are yellow. A polarizing microscope is necessary for this distinction to be made.

May be coexisting infection.

Occasionally, a joint fluid specimen has so few cells and such sparse extracellular material that centrifugation of the fluid and microscopic examination of the button are necessary. When these are done, one must record that the results were obtained from a concentrated specimen to avoid confusion.

**Microscopic Analysis**

If the long axis of the crystals is blue when parallel to the Z axis and yellow when perpendicular to it, it is calcium pyrophosphate and is termed *positively birefringent.* If the long axis of the crystal is yellow when parallel to the Z axis and blue when perpendicular to it, it is monosodium urate and is termed *negatively birefringent.* Urate crystals are 2 to 10 mum and are usually needle shaped. Calcium pyrophosphate crystals range from 10 mum down to tiny crystals that have to be examined with the oil objective; they appear as rods, rhomboids, plates, or needle-like forms and are weakly birefringent. Cholesterol crystals are sometimes seen and are large, very bright, square, or rectangular plates with broken corners.

Items found in synovial fluid that can be confused with sodium urate (Fig. 57-12) (Figure Not Available) or calcium pyrophosphate crystals (Fig. 57-13) (Figure Not Available) include collagen fibrils, cartilage fragments (Fig. 57-14) (Figure Not Available) , cholesterol crystals (Fig. 57-15) (Figure Not Available) , metallic fragments from prosthetic arthroplasty, and corticosteroid esters. [32] One may also identify fat globules (Fig. 57-16 (Figure Not Available) *A* and *B*). Note that rare cases of uric acid spherulites in gouty synovia have been reported.  The spherulites are birefringent and do not take up fat stains.

Table 57-3 summarizes synovial fluid features for the joint diseases commonly encountered and studies commonly performed in the emergency department.

## CONCLUSION

When performed correctly, arthrocentesis is a relatively safe procedure that is used to obtain valuable diagnostic information as well as to provide therapy for acute joint disease. The key to success is strict adherence to sterile technique, observance of anatomic landmarks, and proper preparation of the synovial fluid for examination.

# Chapter 58 - Compartment Syndrome Evaluation

**Lawrence B. Stack**

Compartment syndrome is a condition of increased pressure within a limited space that results in compromised tissue perfusion and ultimate dysfunction of neural and muscular structures contained within that space. [1] This is manifest clinically by pain (usually out of proportion to the injury), worsening of the pain with passive stretch of the involved muscles, paresthesias in the distribution of the involved nerves, paresis or paralysis of the involved muscles, and palpable tenseness of the involved compartment. It should be stressed that *severe pain may be the only sign or symptom in patients with compartment syndrome.* [1] Compartment syndrome requires prompt recognition and treatment for optimal outcome. One theory that explains how increasing tissue pressure compromises local blood flow is the "arteriovenous gradient theory," [2] which proposes that increased tissue pressure decreases the arteriovenous gradient and, hence, blood flow. [1] When blood flow is reduced to a level at which metabolic demands of the tissues are not met, dysfunction of those tissues occurs and a compartment syndrome ensues.

Although compartment syndrome is relatively uncommon, recognition of this process has increased with greater physician awareness of its clinical features. The lower leg and forearm are the most frequent locations of compartment syndrome [1] ; however, gluteal, hand, foot, upper arm, thigh, and back compartment syndromes are being reported with increasing frequency. Causes of compartment syndrome have been categorized into those that decrease compartment volume, increase compartment content, or create externally applied pressure. [1] If left untreated, compartment syndrome may result in permanent neurologic-muscular dysfunction and extremity deformity or lead to shock or renal failure if myonecrosis is extensive. [3]

The differential diagnosis of compartment syndrome includes neuropraxia, arterial injury, cellulitis, osteomyelitis, tenosynovitis, synovitis, and thrombophlebitis. Nerve and arterial injury may coexist with compartment syndrome. Differentiating compartment syndrome from these disease processes may be difficult even for the experienced clinician. Although compartment syndrome is a clinical diagnosis, objective measurement of compartment tissue pressure may help confirm the diagnosis and determine if operative treatment is required. This chapter discusses the indications, complications, and interpretation of compartment pressure monitoring. The equipment and techniques required to perform compartment pressure monitoring are described.

## BACKGROUND

Post-ischemic myoneural dysfunction and contractures were first described in the 1870s by Von Volkmann. [4] In 1935, Henderson and associates developed an open-needle technique

for measuring muscle "tonus." [5] Their method consisted of the 3-way connection of a syringe, a manometer, and a needle placed into the muscle itself. In the 1960s, the technique was applied to muscle compartment pressure measurement. In 1975,

Whitesides and colleagues refined the technique and described its ability to accurately reflect muscle compartment pressures. [6] They also related elevated pressures to a need for fasciotomy to relieve a compartment syndrome.

Other investigators were less confident in the reproducibility of the intermittent readings. In 1976, Matsen and coworkers modified the technique to include a constant infusion pump that allowed for prolonged, continuous monitoring. [7] Mubarak and associates objected to the modification made by Matsen and coworkers, because the continuous infusion method injected more fluid into a compartment already compromised with increased pressure. [8] Modifying a technique developed by Scholander and colleagues [9] for fluid monitoring in plants, Mubarak and associates showed that a "wick" catheter accurately reflected compartment pressures in humans. [7] [10] This method proved to be as reproducible as the infusion technique. Objections subsequently arose to the degeneration of the biodegradable wick as a source of error and to the potential for retaining the wick on removal of the catheter. Rorabeck and colleagues developed a "slit" catheter to replace the wick. [11] Accuracy and reproducibility of the wick and the slit catheter techniques have been similar. In the mid-1980s, a solid-state transducer intracompartmental (S.T.I.C.) catheter was developed. This device, produced by Millar Instruments (Houston), is a catheter with a built-in pressure transducer that does not require saline-filled tubing to transmit the compartment pressure to an external transducer. [14] Arterial line transducer monitoring systems have been used for acute monitoring of compartment pressure, although no study has been conducted to describe the accuracy of this technique. The Stryker intracompartmental pressure monitoring system (Kalamazoo, Mich) is a solid-state handheld device that is currently available for temporary or continuous compartment pressure monitoring.

## PATHOPHYSIOLOGY OF COMPARTMENT SYNDROME

Pressure studies clearly show a linear relationship between increased tissue pressure and decreased blood flow. [1] These data argue against a critical "closure" pressure of compartment vessels or a microvascular occlusion mechanism. The most acceptable theory for the reduced perfusion relates local blood flow to the arteriovenous pressure gradient: the greater the gradient, the greater the blood flow. ] In this model, veins are easily collapsed, and therefore venous pressure can be no lower than tissue pressure. As tissue pressure increases, venous pressure increases. Without a concomitant increase in arterial pressure, the arteriovenous gradient, and hence local blood flow, decreases. When local blood flow fails to meet metabolic needs, ischemia causes tissue dysfunction, and a compartment syndrome follows. This theory accounts for observed exacerbations of compartment syndromes by decreased arterial pressure such as that occurring with systemic hypotension or elevation of a limb. These relationships have been explored and upheld in several tissue pressure studies.  In an elegant evaluation by Heppenstall and colleagues, nuclear magnetic resonance analysis of muscle cell phosphorus metabolism confirmed the relationship between arteriovenous gradient (perfusion pressure) and cellular ischemia. [19]

Early in the syndrome, the dysfunction is reversible if conditions are changed. Histologic studies have shown that as ischemia continues, degeneration and necrosis occur. [20] As cell death occurs, edema may further exacerbate the pressure problem, and the entire

process may become worse. If the situation has progressed to this point, some permanent dysfunction is inevitable. Early attention to ischemic symptoms, coupled with pressure measurements, helps identify this syndrome and leads to early therapy and potential avoidance of these sequelae.

## CLINICAL PRESENTATION

Even experienced personnel may find it difficult to evaluate a potential compartment syndrome, because the time of onset is extremely variable. Matsen and Clawson have found the onset of symptoms to range from 2 hours to 6 days after the insult. [21] The peak time seems to be 15 to 30 hours (Fig. 58-1) .

The limiting envelope required to produce a compartment syndrome may include fascia, skin, casts, external dressings, or even epimysium alone. [1] General categories for the many sources of increased pressure within these envelopes include decreased compartment volume, increased compartment contents, and externally applied pressure. Table 58-1 (Table Not Available) lists the reported causes in each category. Because of the nature of the limiting envelopes and the acknowledged causes, compartment syndromes are most commonly seen in the extremities. The lower leg is at high risk because of its propensity for injury and the existence of several low-volume compartments. It is interesting to note that lumbar paraspinal compartment syndrome may represent an unusual presentation of low back pain. [22]

The essential elements that constitute the diagnosis of compartment syndrome are revealed in its definition: a condition of increased pressure within a limited space that results in compromised tissue perfusion and ultimate dysfunction of neural and muscular structures contained within that space. Clinical history is the first step in the evaluation of an injury or condition that may produce a compartment syndrome, and it may reveal the etiology (see Table 58-1) (Table Not Available) . Clinical examination confirms the diagnosis when there is clear evidence of increased tissue pressure, inadequate tissue perfusion, and loss of tissue function. The diagnosis is less certain when one or more of these factors is absent. [1] Signs and symptoms resulting from locally decreased tissue perfusion are pain and neurologic dysfunction. Evidence of dysfunction forms the basis for the physical diagnosis.

Often the first symptom described by the patient is pain greater than expected for a given clinical situation. Pain often increases with the passive stretching of muscles in the involved compartment. The muscles also may be weak in comparison to normal. Hypesthesia may be present in the distribution of nerves and tenseness may be palpable in the involved compartment. Decreased vibratory sensation may be one of the earliest sensory findings. [23] These findings usually progress during a period of observation. As a rule, *the presence or absence of arterial pulsation is not an accurate indicator of increased tissue pressure*; pulses may be present in a severely compromised extremity compartment. [24] When pulses are obliterated distally, irreversible damage has often occurred. Table 58-2 lists symptoms and signs of the respective compartments when compartment syndrome is suspected.

Patients who have an altered mental status and younger, uncooperative patients may

make the interpretation of neuromuscular signs difficult. In addition, casts or bulky dressings may make careful examination impossible. Attributing the signs and symptoms to other pathologic entities is also a problem (Table 58-3) (Table Not Available) . Primary nerve and muscle injuries can produce similar findings, but the deficit should be maximal

| **TABLE 58-2** -- Compartment Syndromes and Associated Physical Signs | | | | |
|---|---|---|---|---|
| **Compartment** | **Sensory Loss** | **Muscles Weakened** | **Painful Passive Motion** | **Tenseness Location** |
| **Forearm** | | | | |
| Dorsal | -- | Digital extensors | Digital flexion | Dorsal forearm |
| Volar | Ulnar/median nerves | Digital flexors | Digital extension | Volar forearm |
| **Hand** | | | | |
| Interosseus | | Interosseus | Abduct/adduct (metacarpophalangeal joints) | Dorsum hand between metacarpals |
| **Leg** | | | | |
| Anterior | Deep peroneal nerve | Toe extensors Tibialis anterior | Toe flexion | Anterior aspect leg |

| | | | | |
|---|---|---|---|---|
| Superficial posterior | -- | Soleus and gastrocnemius | Foot dorsiflexion | Calf |
| Deep posterior | Posterior tibial nerve | Toe flexors Tibialis posterior | Toe extension | Distal medial leg, between Achilles tendon and tibia |
| *Gluteal* | (Rarely sciatic) | Gluteals, piriformis, or tensor fascia lata | Hip flexion | Buttock |
| *Upper arm* | | | | |
| Flexor | Ulnar/median nerves | Biceps and distal flexors | Elbow extension | Anterior upper arm |
| Extensor | Radial nerves | Triceps and forearm extensors | Elbow flexion | Posterior upper arm |
| *Foot* | Digital nerves | Foot intrinsics | Toe flexion/extension | Dorsal/plantar foot |
| *Lumbar* | -- | Erector spinae | Lumbar flexion | Paraspinous |

initially and should not progress. Arterial injuries and subsequent ischemia may produce pain and dysfunction, although neurologic changes may be less pronounced unless secondary edema produces a compartment syndrome. Thrombophlebitis [25] and cellulitis [21] must also be considered in the differential diagnosis. Pain out of proportion to obvious injury may raise the issue of drug-seeking behavior, but a focused evaluation

for potential compartment syndrome should precede such a diagnosis.

## INDICATIONS FOR INVASIVE (COMPARTMENT PRESSURE) MONITORING

The earliest manifestation of acute compartment syndrome is an elevated tissue pressure. Signs and symptoms occur after tissue pressure has been elevated beyond a critical time period. Measurement of this pressure allows earlier diagnosis and treatment. In some patients, the diagnosis of compartment syndrome is clinically obvious, and one can proceed directly to fasciotomy. However, when clinical findings are equivocal or difficult to interpret, tissue pressure measurement may help guide treatment (Fig. 58-2) (Figure Not Available) . Three groups of patients for whom clinical findings are difficult to interpret, and who may benefit from compartment pressure measurement, are unresponsive patients, uncooperative patients (such as children or intoxicated patients), and patients with peripheral nerve deficits attributable to other causes (e.g., tibial fracture with peroneal nerve injury).

## PATIENT PREPARATION

Patient preparation includes obtaining informed consent when appropriate, positioning the patient and extremity for optimal results, performing skin preparation at the site of needle introduction, local anesthesia, and conscious sedation when necessary. Because compartment pressure monitoring is an invasive procedure with potential complications and may require conscious sedation and possibly an operative procedure, informed consent should be obtained from the patient or guardian, using standard hospital forms. The patient's clinical status and lack of availability of family may prohibit formal written consent in some circumstances.

Patient and extremity position for compartment pressure measurement depends on the extremity being studied, the compartment being studied, and presence of coexisting injury. Patients should be comfortable, usually in the supine or prone position, and the compartment to be measured should be at the same level as the heart. The patient and extremity should be positioned to allow the needle to be introduced perpendicular to the muscular compartment being measured. Any obstructions to the needle entry point into the skin and all structures that may put pressure on the compartment and falsely elevate the pressure should be removed. That is, the extremity of the compartment being measured should be positioned so that no external pressure is applied to that compartment. This may require that the extremity be held slightly above the stretcher by an assistant.

Once the location for needle placement has been identified, preparation of the skin should be conducted as for any sterile procedure. Care should be taken to avoid needle placement in areas in which the overlying skin is possibly infected, to avoid introduction of bacteria into deep tissues. If an overlying cast is present, it should be bivalved, and if necessary, a window overlying the desired area of needle penetration should be cut from the cast. The skin should be anesthetized with a small amount of local anesthetic, with care taken to avoid injection into muscle or fascia. Excessive, deep infiltration may

falsely elevate the compartment pressure reading.

Conscious sedation should be considered in any patient who may be uncooperative and unable to hold the extremity still during the procedure. Any struggling or movement that requires restraint of the extremity may falsely elevate the compartment pressure. Short-acting narcotics or benzodiazepines are ideal because of their brief duration of action and the availability of specific antagonists (see Chapter 35) . Caution should be taken because these agents may blunt the pain and paresthesias that are paramount in the diagnosis of compartment syndrome. The benefits of accurate pressure measurement must be weighed against obliterating these clinical findings for the duration of sedation.

## TISSUE PRESSURE MONITORING

The methods described in this chapter focus on the acute compartment pressure measurement with equipment commonly found in the emergency department (ED) or intensive care setting. These include the mercury manometer system, [18] arterial line system, and the Stryker 295 intracompartmental pressure monitor system (Fig. 58-3) (Figure Not Available) . All techniques are based on the premise that pressure is equal in an open system. The described methods provide rapid compartment pressure measurement with acceptable accuracy. Although the mercury manometer system is the least expensive and most readily available, it is also the least accurate and its results are the least reproducible. [1]

Devices commercially available for placement into the compartment for pressure measurement include the simple 18-ga needle, 18-ga spinal needle for deep compartments, the side-port needle (Stryker Instruments, Kalamazoo, Mich), wick catheter, slit catheter, and the solid-state transducer intracompartmental catheter (S.T.I.C.). The latter 3 devices are generally not available to most EDs and are discussed only briefly. All pressure measurement systems described above can be used with the simple 18-ga needle, 18-ga spinal needle, and the side-port needle. The side-port needle and slit catheter have comparable accuracy when used for compartment pressure measurement. [26]

### Mercury Manometer System

#### Equipment

> 2 18-ga needles (or spinal needles)
>
> 2 plastic extension tubes
>
> 1 20-mL syringe
>
> 1 3-way stopcock
>
> 1 vial of sterile normal saline

1 mercury manometer

1. The patient and extremity are prepared according to the guidelines above.
2. The syringe, tubing, extension tubing, and needle are assembled as shown in Fig. 58-4 (Figure Not Available) *A*.
3. The needle is inserted into a vented vial of sterile saline. A column of saline is aspirated into the tubing halfway to the stopcock; care should be taken to avoid bubble formation. The 3-way stopcock is turned to close off this tube to avoid losing saline during movement of the needle into the patient.
4. The needle is inserted into the muscle of the desired compartment (see Needle Placement for details).
5. The second extension tubing is attached to the monitor and to the third port of the 3-way stopcock. The stopcock is turned so that the syringe is open to both extension tubes (Fig. 58-4 (Figure Not Available) *B*). This closed system has equal pressure in both extension tubes.
6. The pressure in the system is increased gradually by *slowly* depressing the syringe plunger while simultaneously watching the column of saline. The mercury manometer will rise as the pressure in the system rises. When the pressure in the system exceeds the tissue pressure, saline is injected into the compartment, causing the saline column to move. The manometer is read at the time at which the saline moves. This reading corresponds to the tissue pressure in millimeters of mercury.
7. A second reading should be obtained by completely removing the needle and repeating steps 4 to 6. A third

   measurement may be necessary to get 2 readings in agreement. The needle should be checked between readings for tissue plugs and blood clots.

**Procedural Pearls**

The most common error with this system is depressing the syringe plunger too quickly. Only when the saline is slowly injected into the compartment will the mercury column (which has greater inertia) accurately reflect the compartment pressure. Another source of error with this system is obstruction of the needle with a plug of tissue if the syringe plunger is pulled back. Finally, aneroid manometers are not calibrated at lower pressure ranges and should not be substituted for the more accurate mercury manometers.

## Arterial Line System

**Equipment**

Intracompartment needle

High-pressure tubing

Pressure transducer with cable

Pressure monitor/module

Sterile saline

Transducer stand that allows variable height

2 3-way stopcocks

1 20-mL syringe

**Setup and Procedure**

1. The patient and extremity are prepared according to the guidelines above.
2. The transducer cable is connected to the pressure monitor/module.
3. The stopcocks, transducer, transducer cable, syringe, high-pressure tubing, and needle are assembled as in Figure 58-5 (Figure Not Available) .
4. The syringe is filled with 15 mL of sterile saline and placed on 1 stopcock. The stopcocks are turned to allow filling of the transducer, high-pressure tubing, and needle. The stopcock to the high-pressure tubing is then closed.
5. The top stopcock is opened to air and the transducer placed at the height of the compartment to be measured. The transducer is calibrated to zero and the top stopcock closed.
6. The lower stopcock to the high-pressure tubing is opened and the needle is inserted into the muscle of the desired compartment (see Needle Placement for details). Appropriate placement is determined by digitally compressing the compartment or passively moving the muscles within the compartment. The compartment is allowed to equilibrate for several seconds after this maneuver, and the *mean* pressure is measured.
7. A second reading should be obtained by completely removing the needle and repeating steps 4 through 6. A third measurement may be necessary to get 2 readings in agreement. The needle should be checked between readings for tissue plugs and blood clots.

## Stryker 295 Intracompartmental Pressure Monitor System

**Equipment**

Handheld pressure monitor

Stryker 295 quick-pressure monitor set (disposable pouch)

1 prefilled syringe with saline

1 side-port needle

1 diaphragm chamber

**Setup and Procedure**

1. The 295 quick-pressure monitor set (disposable) is opened, and contents are removed, maintaining appropriate system sterility.
2. The needle is placed firmly on the tapered chamber stem (Fig. 58-6 (Figure Not Available) *A*).
3. The blue cap on the prefilled syringe is removed and screwed onto the remaining chamber stem (Fig. 58-6 (Figure Not Available) *A*). Care must be maintained to not contaminate the fluid pathway.
4. The cover of the monitor is opened. The chamber is placed in the device well (black surface down) and pushed gently until it seats (Fig. 58-6 (Figure Not Available) *B*).
5. The cover is snapped closed-- *NOT FORCED*. The latch must have "snapped" in place.
6. The clear end cap is pulled off the syringe and the plunger rod is attached to the syringe.
7. The needle is held at approximately 45° up from horizontal while fluid is slowly forced through the disposable system to purge it of air. *Caution:* Saline *MUST NOT* roll down the needle into the transducer well.
8. The unit is turned on and should read between 0 and 9 mm Hg.
9. The intended angle of insertion of the needle into the skin is approximated while the "zero" button is pressed. The display should read "00" (Fig. 58-6 (Figure Not Available) *C*). *Note:* The display must read "00" before continuing. If it does not, troubleshooting using that section of the maintenance manual should be undertaken.
10. The needle is inserted into the compartment (see Needle Placement for details). Less than 0.3 mL of saline should be slowly injected into the compartment for equilibration with interstitial fluids.
11. The pressure is read after the display reaches equilibrium.
12. *For additional measurements:* The unit is turned off and steps 8 through 11 are repeated. The unit must be recalibrated to zero before each measurement.

## NEEDLE PLACEMENT TECHNIQUES

### General Principles

Once the decision to perform compartment pressure measurement has been made, the approach for needle placement and confirmation of correct placement must be considered. The requirements for a useful approach include: (1) reliable placement in the compartment to be measured; (2) avoidance of important neurovascular structures; (3) simplicity and reproducibility; and (4) minimal discomfort to the patient. [27] Most compartments are superficial and are easily accessible. Only the deep posterior compartment of the lower leg and the gluteal compartment may require deeper needle placement (spinal needle). Most approaches allow the needle to enter perpendicular to the skin. Figures 58-7 through 58-18, in conjunction with the text, provide landmarks for

proper needle placement and passage to ensure proper compartment placement and avoidance of neurovascular structures.

## Lower Leg

The lower leg traditionally has 4 compartments [1] : anterior, lateral, deep posterior, and superficial posterior (Fig. 58-7) . However, it has been theorized that the tibialis posterior muscle is contained within a separate osseofascial muscle compartment, [28] creating a fifth compartment of the lower leg. This chapter considers the tibialis posterior as part of the deep posterior compartment. The lower leg, especially the anterior compartment, is predisposed to compartment syndrome because of its high vulnerability to injury and the relatively limited compartment compliance. The anterior compartment was the most frequent site of compartment syndrome in one patient population. [28]

The easiest cross-sectional level for needle placement for all 4 compartments is approximately 3 cm on either side of a transverse line drawn at the junction of the proximal and middle thirds of the lower leg (Figs. 58-8 (Figure Not Available) to 58-11). When measuring the leg compartment pressures, the patient should be placed in the supine position with the leg at heart level (however, when measuring the superficial posterior compartment, the patient should be prone). Patient preparation should be performed as previously described.

### Anterior Compartment

With the patient supine, the anterior border of the tibia is palpated at the level of the junction of the proximal and middle thirds of the lower leg (see Fig. 58-8 (Figure Not Available) *A*). The needle entry point is 1 cm *lateral* to the anterior border of the tibia. The needle should be directed perpendicular to the skin to a depth of 1 to 3 cm (see Fig. 58-8 (Figure Not Available) *B*). Proper needle placement can be confirmed by seeing a rise in pressure during (1) digital compression of the anterior compartment just proximal or distal to the needle insertion site, (2) plantar flexion of the foot, and (3) dorsiflexion of the foot. These maneuvers should produce a several-fold rise in pressure on the monitor.

### Deep Posterior Compartment

With the patient supine, the leg should be slightly elevated off the stretcher if the clinical situation permits. The medial border of the tibia is palpated at the level of the junction of the proximal and middle thirds of the lower leg. The needle entry point is just posterior to the medial border of the tibia (see Fig. 58-9 (Figure Not Available) *A*). The posterior border of the fibula should be palpated on the lateral aspect of the leg at the same level. The needle path should be perpendicular to the skin and directed toward the palpated posterior border of the fibula to a depth of 2 to 4 cm, depending on the amount of subcutaneous fat (see Fig. 58-9 (Figure Not Available) *B*). Proper needle placement can be confirmed by seeing a rise in pressure during (1) toe extension and (2) ankle eversion.

**Lateral Compartment**

With the patient supine, the leg should be slightly elevated off the stretcher if the clinical situation permits. The posterior border of the fibula is palpated at the level of the junction of the proximal and middle thirds of the lower leg. The needle entry point is just anterior to the posterior border of the fibula (see Fig. 58-10 (Figure Not Available) *A*). The needle path should be perpendicular to the skin and directed toward the fibula to a depth of 1 to 1.5 cm (see Fig. 58-10 (Figure Not Available) *B*). If the needle contacts bone, the needle should be retracted 0.5 cm. Proper needle placement can be confirmed by seeing a rise in pressure during (1) digital compression of the lateral compartment just inferior or superior to the needle entrance site and (2) inversion of the foot and ankle.

**Superficial Posterior Compartment**

With the patient prone and the leg at heart level, a transverse line at the level of the junction of the proximal and middle thirds of the lower leg is identified. The needle entry point is at this level and 3 to 5 cm on either side of a vertical line drawn down the middle of the calf (see Fig. 58-11 (Figure Not Available) *A*). The needle path should be perpendicular to the skin and directed toward the center of the lower leg to a depth of 2 to 4 cm (see Fig. 58-11 (Figure Not Available) *B*). Proper needle placement can be confirmed by seeing a rise in pressure during (1) digital compression of the superficial posterior compartment just inferior or superior to the needle entrance site and (2) foot dorsiflexion.

## Forearm

The forearm traditionally has 2 compartments, [1] volar and dorsal, which are divided by the interosseus membrane. However, some authors place the extensor carpi radialis longus, extensor carpi radialis brevis, and brachioradialis muscles into their own compartment, called the "mobile wad." [29] The latter group of muscles is considered a separate compartment in this chapter. The forearm compartments are predisposed to compartment syndrome, especially the volar compartment, because of their use during vigorous exercise, accessibility for drug injection, and vulnerability to burns. [1]

The junction of the proximal and middle thirds of the forearm is the cross-sectional level for needle insertion (Fig. 58-12) (Figure Not Available) . [27] When measuring forearm compartment pressures, the patient should be placed in the supine position with the arm at heart level. Patient preparation should be performed as described previously.

**Volar Compartment**

The forearm should be held in supination. The palmaris longus tendon is identified and its course traced proximal to the level of the junction of the proximal and middle thirds of the forearm. The posterior border of the ulna is palpated. The needle entry point is just medial to the palmaris longus (Fig. 58-13 (Figure Not Available) *A*). The needle path

should be perpendicular to the skin and directed toward the palpated posterior border of the ulna to a depth of 1 to 2 cm (g. 58-13 *B*). Proper needle placement can be confirmed by seeing a rise in pressure during (1) digital compression of the volar compartment just proximal or distal to the needle entry point and (2) extension of the fingers or wrist.

**Dorsal Compartment**

The forearm should be held in supination with the elbow flexed, allowing the dorsum of the forearm to face downward. The posterior aspect of the ulna is palpated at the level of the junction of the proximal and middle thirds of the forearm. The needle entry point is 1 to 2 cm lateral to the posterior aspect of the ulna (Fig. 58-14 (Figure Not Available) *A*). The needle should be directed perpendicular to the skin to a depth of 1 to 2 cm (Fig. 58-14 (Figure Not Available) *B*). Proper needle placement can be confirmed by seeing a rise in pressure during (1) digital compression of the dorsal compartment just proximal or distal to the needle entry point and (2) flexion of the fingers or wrist.

**Mobile Wad**

The forearm should be held in supination. The most lateral portion of the forearm is identified at the level of the junction of the proximal and middle thirds. The needle entry point is the muscle tissue lateral to the radius. The needle should be directed perpendicular to the skin (Fig. 58-15 (Figure Not Available) *A*) and inserted to a depth of 1 to 1.5 cm (Fig. 58-15 (Figure Not Available) *B*). Proper needle placement can be confirmed by seeing a rise in pressure during (1) digital compression of the mobile wad just proximal and distal to the needle entry point and (2) ulnar deviation of the wrist.

## Gluteal Musculature

The gluteal musculature is enclosed by an enveloping fascia that splits into 2 layers and encases the muscle bellies of the tensor fascia lata anteriorly and gluteus maximus posteriorly. This fascia divides the musculature into 3 distinct compartments: maximus, tensor, and medius/minimus (Fig. 58-16) (Figure Not Available) . The sciatic nerve is deep to the fascia but lies between the pelvis-external rotator complex and the gluteus maximus, making it susceptible to injury in compartment syndrome of the gluteus maximus. Most reported cases of gluteal compartment syndrome result from prolonged immobilization and local compression in association with drug or alcohol intoxication. [30] Gluteal compartment syndrome is rare, making the diagnosis difficult, as local tenderness is often attributed to contusion or hematoma. Rhabdomyolysis should be considered in patients with gluteal compartment syndrome because of the large muscle mass that may be injured.

**Gluteal Compartments**

The patient should be placed in the prone position with the buttocks at heart level. Patient preparation should be performed as described previously. Cutaneous landmarks for the 3 compartments are not consistent from patient to patient. In all cases of suspected gluteal compartment syndrome, the needle insertion point can be at the point

of maximal tenderness. [30] An 18-ga spinal needle should be used. The needle should be directed perpendicularly to the skin toward the point of maximum tenderness to a depth of 4 to 8 cm. Proper needle placement is confirmed by seeing a rise in pressure during digital compression of the gluteal musculature.

## Foot

Compartment syndrome of the foot is rare, but it is being reported with increasing frequency as physicians become more aware of the disease process. The 4 compartments of the foot as seen from a coronal section at the base of the first metatarsal are the central, interosseous, medial, and lateral compartments (Fig. 58-17) (Figure Not Available) . The foot is vulnerable to isolated extremity injury by virtue of its location. Most compartment syndromes in the foot are the result of crushing forces.

When measuring compartment pressures in the foot, the patient should be placed in a supine position with the foot at the level of the heart. [31] Patient preparation should be performed as described previously.

### Medial Compartment

The medial compartment contains the abductor hallucis and flexor hallucis brevis muscles and is bounded medially and inferiorly by the extension of the plantar aponeurosis, laterally by an intermuscular septum, and dorsally by the first metatarsal (Fig. 58-18 (Figure Not Available) *A*). The medial aspect of the base of the first metatarsal is palpated. The needle entry point is the medial aspect of the foot just inferior to the base of the first metatarsal and into the abductor hallucis muscle. [32] The needle is advanced to a depth of 1 to 1.5 cm. Proper needle placement is confirmed by seeing a rise in pressure during digital compression of the medial compartment of the foot.

### Central Compartment

The central compartment contains the flexor digitorum brevis, the quadratus plantae, the lumbricals, and the abductor hallucis muscles. Its boundaries are the plantar aponeurosis inferiorly, the osseofascial tarsometatarsal structures dorsally, and the intermuscular septa medially and laterally. The medial aspect of the base of the first metatarsal is palpated. The needle entry point is the medial aspect of the foot just inferior to the base of the first metatarsal and through the abductor hallucis muscle. The needle is advanced to a depth of 3 cm. [32] Proper needle placement is confirmed by seeing a rise in pressure during digital compression of the central compartment of the foot.

### Lateral Compartment

The lateral compartment contains the abductor, flexor, and opponens muscles of the fifth toe. The boundaries are the fifth metatarsal dorsally, the plantar aponeurosis inferiorly and laterally, and an intermuscular septum medially. [33] The base of the fifth

metatarsal is palpated. The needle entry point is just inferior to the base of the fifth metatarsal (Fig. 58-18 (Figure Not Available) *B*). The needle should be directed parallel to the plantar aspect of the foot to a depth of 1 to 1.5 cm. Proper needle placement is confirmed by seeing a rise in pressure during digital compression of the lateral compartment of the foot.

**Interosseous Compartment**

The interosseous compartment contains the 7 interossei muscles and is bounded by the metatarsals and the interosseous fascia. The dorsum of the bases of the metatarsals are palpated and the second and fourth web spaces are identified. The first web space is avoided to prevent inadvertent puncture of the dorsalis pedis or deep peroneal nerve. [32] The needle entry point is the dorsum of the second and fourth web spaces at the metatarsal bases (Fig. 58-18 (Figure Not Available) *C*). The needle is directed perpendicularly to the skin to a depth of 1 cm. [32] Proper needle placement is confirmed by seeing a rise in pressure during digital compression of the interosseous compartment adjacent to the needle entry point.

## COMPLICATIONS

All of the procedures described have a similar risk of infection, both local and systemic; exact risk figures are unavailable. Strict adherence to aseptic technique, careful sterilization of catheters, and use of sterile, disposable components whenever possible help to minimize this risk.

All monitoring procedures cause some pain. The pain associated with the actual insertion of needles and catheters may be reduced by local anesthesia. Caution is advised to avoid injections into the compartment, which might result in inaccurate readings or further increases in tissue pressure. Once inserted and secured, the needle or catheter should produce only minimal discomfort. However, the injection of saline into an already tense compartment may produce further discomfort. Reassurance and systemic analgesia may be required to gain the cooperation necessary for accurate readings. Technically, the injection of fluid may actually exacerbate a compartment syndrome. Whitesides and colleagues found an increase in compartment pressure of 1 mm Hg for each milliliter of saline infused into human anterior leg compartments. [6] It is difficult to assess the relevance of this problem, but recognition of the potential for its occurrence is important.

## INTERPRETATION

When properly performed, each method has an acceptable accuracy in the clinical setting. Investigators report standard deviations from 2 to 6 mm Hg with any of the techniques.   It is generally agreed that the mercury manometer system is the least accurate. The arterial line system used with a simple or side-port needle provides a high degree of accuracy for simple, episodic readings. The Stryker intracompartmental pressure monitor system provides consistent, accurate readings for episodic and

extended monitoring situations.

Reports of normal human compartment pressures vary in the literature. In comparing several techniques, Shakespeare and associates found an average pressure of 8.5 mm Hg. [12] Other investigators have found similar pressures with a range from 0 to 16 mm Hg. Shakespeare and

associates found higher pressures in individuals who were physically fit.

Hargens and coworkers found dog muscle capillary pressures to be 20 to 30 mm Hg. [18] Whitesides and associates showed a significant decrease in tissue perfusion when compartment pressures rose to 10 to 30 mm Hg below diastolic blood pressure. [6] Mubarak and colleagues showed that tissue pressures of 30 mm Hg closely corresponded to the onset of pain and paresthesias. [10] Therefore, a tissue pressure of 25 to 30 mm Hg is abnormal but does not necessarily precipitate a compartment syndrome in the absence of other factors.

Some variability seems to exist among patients for tolerance of increased pressures. Matsen found that no patients with pressures of less than 45 mm Hg had symptoms of compartment syndrome, whereas all patients with pressures greater than 60 mm Hg had symptoms. [1] Heppenstall and colleagues used phosphorus metabolism to study perfusion pressures and anaerobic metabolism in skeletal muscle. Cellular injury was confirmed by electron microscopy. Their results confirm that cellular perfusion is dependent on the pressure differential (DeltaP). This value is obtained by subtracting the compartment tissue pressure from the mean arterial blood pressure. Anaerobic metabolism became significant in normal muscle when the DeltaP was 30 mm Hg or less. More important, in traumatized muscle (in which metabolic demands are greater), the critical DeltaP was 40 mm Hg. [19] Factors other than compartment pressure alone may become important. Situations in which the mean arterial pressure is lowered (e.g., hypovolemia) may compromise a patient's ability to tolerate even mildly elevated compartment pressures. Zweifach and associates showed that there is significant muscle damage with tissue pressure at 20 mm Hg in the presence of a systemic blood pressure at 65 mm Hg. [17] Duration of increased pressure is also important. Matsen found that 12 hours of increased pressure reliably produces deficits. [1] One series showed that none of the patients undergoing fasciotomy for compartment syndrome before the passage of 12 hours had residual deficits. In another series by Matsen, 3 of 18 patients who had compartment decompression *before* 12 hours vs 22 of 24 patients who had decompression *after* 12 hours had residual deficits.

Compartment pressures must be interpreted within the context of the clinical picture. Inaccurate measurements are far worse than no measurement at all. Falsely elevated pressures may be a result of needles placed into tendons or fascia, plugged catheters, or faulty electronic systems. Falsely low readings may result from bubbles in the lines or transducer, plugged catheters, or faulty electronic systems. One must carefully troubleshoot the system before making a decision to treat a presumed compartment syndrome.

Actual treatment consists of improving the perfusion pressure gradient. Support of

arterial pressures in hypotensive patients can prevent ischemia in marginal compartments. If external pressure is at fault, removal of pneumatic antishock garments, casts, or dressings may be therapeutic. Interstitial edema may be a factor in increased tissue pressure as well. Significant reduction of tissue pressure, resulting in an improved perfusion gradient, has been observed after the use of systemic diuretics [34] and intracompartment hyaluronidase. If noninvasive therapies fail, fasciotomy, which involves opening the skin and muscle fascia at key points overlying the involved compartments, should be performed. The escape of enclosed muscles causes a decrease in compartment pressure, thereby improving blood flow to the tissues. This procedure is left to personnel who are experienced in the problem and who will subsequently manage the patient. Details of fasciotomy technique may be found in surgical or orthopedic texts. Escharotomy for release of circumferential burn injury may be required on an emergent basis (see Chapter 41) .

## CONCLUSION

A compartment syndrome, with its myriad potential causes, is a challenging problem for all physicians. If untreated, the sequelae are devastating. Appropriate management requires rapid assessment and treatment. In the early stages of the syndrome, clinical examination findings may be equivocal. Pressure readings can be an objective aid to prompt recognition of this problem. All of the techniques outlined in this chapter are clinically acceptable. Whether using an arterial line system or a mercury manometer system, placement of a needle into the compartment for a pressure reading appears to be the technique best suited to episodic ED evaluation of acute compartment syndromes.

For more prolonged in-hospital monitoring or research, a technique with reliable serial measurement capability is needed. For these applications, the wick or slit catheter techniques (or the continuous-infusion technique, discussed elsewhere [1] ) may be preferable. The complications from all of these procedures are negligible in comparison to delayed recognition of a compartment syndrome.

# Chapter 59 - Urologic Procedures

**Robert E. Schneider**

This chapter addresses urologic conditions that either are initially or eventually associated with an emergency procedure or may need to be performed in the absence of a urologic surgeon.

Paraphimosis and priapism are both urologic emergencies. Treatment options must be instituted as soon as they are encountered. Phimosis by itself is not an emergency unless it results in complete obstruction of the preputial opening (rare) or is transposed into paraphimosis.

Testicular torsion is an emergency that can be difficult to diagnose under the best of clinical conditions. Surgical exploration, rather than radionuclide scanning or color Doppler ultrasonography, is the diagnostic and therapeutic procedure of choice. This chapter addresses bedside maneuvers including testicular detorsion for this entity in the setting of scrotal pain.

Access to and the subsequent evaluation of bladder urine is clinically important to every practicing physician. Various approaches to urine sampling, including the techniques and complications of male and female urethral catheterization in various clinical situations, are addressed.

Finally, a discussion of radiographic imaging of the genitourinary system is provided, with an emphasis on assessing lower urinary tract injury. Although the timing of genitourinary radiologic examinations within the workup of the critically ill multiple trauma patient must be individualized, general guidelines are provided.

## PHIMOSIS AND DORSAL SLIT

### Background

Phimosis has been recognized since ancient times. Models of phimotic foreskin have been found near the altars of Hygeia and Aesendopius in ancient Greece. Orikosius (A.D. 325-403) was the first to describe the dorsal slit as definitive treatment for phimosis.

Following any injury or inflammatory event, the foreskin (prepuce) reacts by forming scar tissue. The normally soft pliable foreskin can develop sufficient distal scarring to make routine retraction of the tissue over the glans penis difficult or impossible. This is especially true if the end of the foreskin is injured, such as in zipper injuries, toilet seat trauma, or other crush injuries (known as the Tristram Shandy syndrome, after the well-known literary character who had a window sash fall on his penis while he was urinating out the window). A chronically irritated and infected foreskin often occurs in diabetic patients. [1] Rarely, a tight phimosis and accompanying poor hygiene may lead

to abscesses of the foreskin, which can result in further contracture.

Asymptomatic phimosis does not ordinarily require any treatment. It may prevent or make easy urethral catheterization more difficult. In such situations, the phimotic opening may need to be dilated or crushed and formally incised (dorsal slit), using light sedation and local anesthesia to allow access to the urethral meatus. This minor operative procedure can be performed in the emergency department (ED) by any practicing physician.

## Indications and Contraindications

Dorsal slit of the foreskin is performed in any emergent situation either to gain access to the urethral meatus for urethral catheterization or as definitive treatment following simple foreskin reduction or phimotic ring incision and foreskin reduction in a patient with paraphimosis. Elective circumcision rather than dorsal slit of the foreskin is the definitive procedure of choice in nonemergent situations.

## Procedure

The equipment needed to perform dorsal slit of the foreskin is listed in Table 59-1 . After cleansing and draping the penis with sterile towels, one infiltrates 1% plain lidocaine *without* epinephrine into the dorsal midline of the foreskin just beneath the superficial fascia throughout the course of the proposed incision, starting proximally at the level of the coronal sulcus and proceeding distally to the tip of the foreskin (Fig. 59-1) . After 3 to 5 minutes, the foreskin is grasped with

| TABLE 59-1 -- Equipment Needed to Perform Dorsal Slit for the Emergency Treatment of Phimosis | |
| --- | --- |
| 1% lidocaine (Xylocaine) without epinephrine | 1 straight Crile clamp |
| | 1 straight scissors |
| 5-mL syringe | 1 needle holder |
| 27-ga needle | 4-0 absorbable suture |

toothed forceps to test for anesthesia. The operator must be certain that the inner surface of the foreskin is also anesthetized. If this area is not numb, a dorsal nerve block or "ring block" at the base of the penis should be used (Fig. 59-2) (Figure Not Available) . [2] [3]

After achieving both adequate local anesthesia and light sedation, the operator takes a straight hemostat and carefully advances both jaws of the hemostat proximally to the area of the coronal sulcus between the inner layer of the foreskin and the smooth glans penis, carefully separating any existing preputial adhesions. Care must be taken that the meatus and urethra are visualized at all times so they are not inadvertently injured during this maneuver. Once release of adhesions is complete, the hemostat is then opened, and one jaw of the hemostat is placed in the recently developed plane between the glans penis and the superior overlying inner layer of foreskin; the hemostat is advanced to the level of the coronal sulcus and then closed, effectively crushing the interposed anesthetized foreskin (Fig. 59-3) . The closed hemostat is left in place for 3 to 5 minutes, after which it is removed, and the resultant serrated, crushed foreskin is cut longitudinally with straight scissors throughout the extent

of the crushed tissue. Normally, the incised, anatomically approximated skin edges bleed and ooze. Not infrequently, these skin edges may separate (Fig. 59-4 A and B). Two absorbable chromic or Vicryl running hemostatic sutures may be placed, each beginning proximally at the apex of the dorsal slit and carried distally, reapproximating the 2 leaves of foreskin.

After successful dorsal slit of the foreskin, the prepuce is easily retracted for cleansing of the glans penis or exposure of the urethral meatus. Postprocedural conscientious foreskin reduction to its normal anatomic position must be assured after any distal penile procedure, to avoid iatrogenic paraphimosis.

Ideally, a definitive elective circumcision is recommended following a dorsal slit. Some patients complain about the appearance of their incised foreskin ("dog ears") and the relative inconvenience during urination, whereas others are pleased they no longer have their phimosis and refuse further treatment (Fig. 59-4 C and D).

### Complications

Injury to the urethral meatus and the glans penis may occur if the hemostat or straight scissors are blindly and unknowingly introduced into the urethra. Bleeding may occur if the hemostat has not adequately crushed the foreskin or the scissor incision is made lateral to the serrated crushed tissue. The latter 2 problems are easily resolved with the previously described running hemostatic suture.

## PARAPHIMOSIS AND FORESKIN REDUCTION

### Background

Paraphimosis is a urologic emergency. By definition, it is the inability to reduce the proximally positioned foreskin over the glans penis back to its normal anatomic position. Paraphimosis is most often associated with phimosis. In the obtunded or demented patient, subjective pain may not be perceived or communicated. Today, the most common cause of paraphimosis is iatrogenic: the catheterist or examining health care provider forgets to reduce the foreskin after penile examination or urethral instrumentation. Paraphimosis can be quite subtle and may either be unrecognized or misdiagnosed as an allergic reaction, penile trauma, or an infection, by those unfamiliar with the condition (Fig. 59-5 *A*).

The coexisting phimotic ring initially interferes with venous and lymphatic drainage, precipitating foreskin swelling. Over time, the degree of swelling prevents manual reduction of the retracted foreskin. When left untreated, eventual arterial embarrassment leads to tissue anoxia, skin ulceration, and, ultimately, to infection and/or penile gangrene. [4]

Emergent manual or surgical reduction of the edematous foreskin is mandatory to restore proper circulation, relieve discomfort, and permit resolution of potential serious sequelae: skin ulceration and gangrene. It must be done as soon as a paraphimosis is recognized. Once the foreskin is successfully reduced, dorsal slit as previously described is advised in those cases of potential patient noncompliance until definitive circumcision can be performed.

### Indications and Contraindications

Emergent reduction of a paraphimotic foreskin is indicated whenever the condition exists. There are no contraindications.

### Procedure

The equipment needed for reduction of paraphimosis is listed in Table 59-2 .

#### Manual Reduction

A nonirritating topical anesthetic lubricant is applied to the inner surface of the foreskin (not to the shaft of the penis)

| **TABLE 59-2** -- Equipment Needed for Reduction of Paraphimosis |
| --- |
| **For Nonoperative Emergency Reduction of Paraphimosis** |

| |
|---|
| 1% lidocaine (Xylocaine) jelly |
| Crushed ice |
| Size 8 latex surgical glove |
| Babcock clamps (6 to 8) |
| **For Operative Reduction of Paraphimosis** |
| Sterile preparation solution |
| 1% lidocaine (Xylocaine) without epinephrine |
| 5-mL syringe |
| 27-ga needle |
| No. 15 surgical knife with handle |
| Needle holder |
| 4-0 absorbable suture |

and the glans to reduce friction and decrease the discomfort of the procedure. A

previously described penile block may be performed if required. Light sedation is an excellent adjunct. The foreskin is then manually compressed for several minutes to reduce as much edema fluid as possible (Fig. 59-5 *B*). Snugly wrapping the distal penis in a 5-cm piece of Elastoplast (or elastic bandage) for 10 minutes has also been described. [6] Injection of hyaluronidase [7] has been reported to reduce edema, but seems unnecessary. If the patient is catheterized, *the catheter generally should be removed and replaced later*. If the swelling is minimal, reduction can at times be completed with the catheter in place.

The index and long fingers of both hands are placed in apposition proximal to the phimotic ring. Both thumbs are aligned on the urethral meatus. Constant force is applied as the thumbs try to invert the glans penis proximally while the index and long fingers attempt to reduce the phimotic ring distally over the glans penis into its normal anatomic position (Fig. 59-5 *C*). Successful reduction results in the appearance of an uncircumcised penis with a phimotic foreskin. Alternatively, the thumb may be used to push the glans through the foreskin that has been encircled by the entire palm--in a maneuver similar to taking off a rubber glove (Fig. 59-5 *D* and *E*). The key to success in both of these maneuvers is the application of slow, steady pressure.

### Assisted Manual Reduction ("Iced-Glove" Method or Babcock Clamp Method)

If the constricting phimotic ring cannot be brought down over the glans easily, additional measures may be used. In the "iced-glove" method, [8] cold compression is used to reduce foreskin swelling and to induce vasoconstriction in the glans penis. A large latex glove is half filled with crushed ice and water, and the cuff end is securely tied. The thumb of the glove is invaginated by the operator and then is drawn over the lubricated paraphimotic penis. The thumb of the glove is held securely in place over the penis for 5 to 10 minutes. The combination of cooling and compression usually decreases the edema sufficiently to permit manual reduction of the foreskin. If the constricting ring cannot be brought down over the glans after this maneuver, it may be necessary to use Babcock clamps. From 6 to 8 Babcock clamps ( *not* Allis clamps, which are serrated and intolerably painful for the patient) are used to grasp the phimotic ring circumferentially. [9] The clamps are then slowly pulled distally over the glans. With gentle, slow traction, the phimotic ring can hopefully be reduced over the glans penis (Fig. 59-6) (Figure Not Available) .

### Phimotic Ring Incision

This procedure is indicated when other methods fail or when skin ulceration, infection, or gangrene is present. [10] [11] [12] Although it is usually recommended that this procedure be performed in the operating room, it may be undertaken in the emergency department using local anesthesia and light sedation.

The penis is cleansed and draped with sterile towels. Using a 1% solution of lidocaine *without epinephrine*, one infiltrates the phimotic ring on the dorsum or 12 o'clock position (when facing the patient), making sure to infiltrate proximally and distally as well as into the constricting ring (Fig. 59-7 *A*).

The skin, the edematous subcutaneous tissue, and the constricting ring are then slowly incised perpendicular to the phimotic ring. You must incise directly on the ring so as not to injure the penile shaft. When the constricting ring is completely incised, it will spring open, and the foreskin edges will relax laterally and produce a diamond-shaped defect (Fig. 59-7 *B*). At this point, successful foreskin reduction can be accomplished quite easily and an immediate dorsal slit incision carried out by extension of the wound, if recurring paraphimosis or patient compliance is a concern. Definitive circumcision, as always, is elective, and should be delayed 7-10 days until edema, inflammation, and ulceration have resolved.

### Complications

Penile shaft laceration or simple tearing of compromised penile skin may occur during manual or surgical paraphimotic reduction. Simple suturing will resolve most injuries.

## PRIAPISM AND PENILE ASPIRATION

Priapism is manifested by a persistent, painful, penile erection, unrelated to sexual stimulation and not relieved by ejaculation. Priapism is a urologic emergency that is associated with a high incidence of impotence, regardless of treatment. It is characterized clinically by a soft glans penis and spongy urethra in the presence of 2 painfully erect penile bodies or corpora cavernosa. Although reported in most age groups, the condition is most common between the ages of 30 and 50 years. In the past, priapism was most often encountered as a complication of a number of medical (e.g., hematologic, neoplastic, and drug-related) conditions (Table 59-3) (Table Not Available) .

Today the majority of cases are idiopathic or iatrogenic, resulting from the current practice of using vasoactive substances (e.g., papaverine and phentolamine) to induce penile erections in impotent men. These vasoactive drugs promote engorgement of the corpora cavernosa, which may result in priapism. Prostaglandin $E_1$ (PGE$_1$ ) is currently the only FDA-approved drug for impotence. PGE$_1$ produces increased penile blood flow by enhancing smooth muscle relaxation, and the incidence of priapism with this medication is quite low. Penile rigidity due to a nondeflating penile prosthesis (pseudopriapism) or malignant replacement of the corpora in patients with bladder or prostate cancer should not be confused with true priapism.

Increased arterial inflow of blood into the corpora cavernosa secondary to dilation of the cavernosal arteries with relaxation of the cavernosal tissue and secondary compression of the emissary veins leads to engorgement of both corpora cavernosa during an erection. When the cavernosal pressure approaches the arterial pressure, blood flow is markedly reduced. Ischemia results after several hours of continuous erection, leading to intracavernosal acidosis and sludging of blood with subsequent thrombosis of the cavernosal arteries, fibrosis of the corporal tissue, and irreversible impotence.

Detumescence is mediated primarily by adrenergic nerves that produce

vasoconstriction, resulting in a decreased inflow of blood to the penis. Priapism may be due to either increased or unregulated inflow or failure of venous outflow. By far, the most common cause is failure of the blood to leave the corporal bodies, as is the case with drug-induced priapism and intracavernosal injections. Although an increased inflow of blood is an uncommon mechanism of priapism, the condition is most commonly associated with a trauma-induced fistula between the cavernosal artery and the corpus cavernosum.

**Indications and Contraindications for Priapism Procedures**

The emergency physician should attempt to identify reversible causes for the patient's priapism and, in conjunction with a urologist, initiate specific corrective therapy as soon as possible. Almost 66% of cases of priapism in children are due to sickle cell disease, and such cases may respond to noninvasive standard anti-sickling measures (including exchange transfusion).

Regardless of the etiology, empiric terbutaline, 0.25-0.5 mg given subcutaneously (SQ), is recommended for every patient presenting with priapism as soon as the diagnosis is made; this treatment may be repeated, when needed, in 15 to 20 minutes. If a patient with previous priapism calls from home and has the appropriate supplies, he should be instructed to inject himself with terbutaline (as above) or take a 5 mg terbutaline tablet by mouth, if available, before coming to the ED. [16] The pharmacotherapeutic action of terbutaline for priapism is not well elucidated. Oral pseudoephedrine (60 to 120 mg) also has been suggested as a noninvasive initial therapy for priapism secondary to intracavernosal agents, but its efficacy has not been well studied. [17] Table 59-4 lists the reversible causes of priapism with their respective therapies.

In the majority of patients, no reversible cause will be disclosed. In about 50% of patients, detumescence will not occur with SQ terbutaline, thus necessitating corporal aspiration alone or corporal aspiration and irrigation with an alpha-agonist (epinephrine or phenylephrine [Neo-Synephrine]) to promote detumescence. Conservative measures, such as sedation and analgesia, oral estrogens, ice-water enemas, transurethral diathermy, spinal/epidural/general anesthesia, and local anesthetic injections have not proven to be of value and should not be used in lieu of definitive intracavernosal therapy. [17]

Should initial medical and corporal aspiration and irrigation fail, Lue and associates recommend that further therapy be guided by cavernosal blood gas and pressure measurements. [18] If one elects to use blood gas analysis of aspirated corporal blood to help guide therapy, the initial results will reflect the degree of ischemia present in the priapismic penis. Sequential blood gas analyses that fail to show an improvement in the initial acidosis (pH remaining at 7.10) despite corporal aspiration and/or irrigation suggests that a more aggressive definitive corpus cavernosum-spongiosum shunt may be indicated, and urgent urologic intervention should be sought. In addition, the aspirating or irrigating corporal needle may also be used for measurement of intracorporal pressures similar to the approach outlined for muscular compartmental pressures (see Chapter 58) .

The mainstay of therapy for advanced priapism is aspiration of the corpora cavernosa combined with saline irrigation, often coupled with the intracavernosal injection of alpha-adrenergic agents. When urologic consultation is unavailable or delayed, the emergency physician must be familiar with diagnostic and therapeutic corporal aspiration. Because prolonged priapism increases the risk of subsequent erectile dysfunction, an aggressive management strategy is advised. Impotence rates of up to 35% to 60% have been reported when priapism persists for 5 to 10 days, respectively. [17] If present for >24 hours, priapism often does not respond to aspiration alone. In such cases, irrigation should always be performed.

## Equipment

The equipment needed for aspiration and irrigation of the corpus cavernosum is listed in Table 59-5 .

## Procedure

The patient is placed in the supine position (Fig. 59-8 *A- D*). Local anesthesia is recommended for this procedure. An injection of 1% plain lidocaine placed at the base of the penis for a dorsal penile nerve block or placement of a circumferential penile block can be done (see Fig. 59-2) (Figure Not Available) . The penis is sterilely prepared and draped. When standing to the right of the patient, the clinician grasps the shaft of the penis with the left hand using the thumb and index finger. An engorged corpus cavernosum is palpated laterally and a 21- to 19-ga butterfly needle is inserted into one of the corpora cavernosa (at 2 or 10 o'clock). [19] The site of needle placement is one of personal preference, and locations from the base to the distal shaft of the penis have been suggested. *The glans should not be used as a puncture site.* Deep penetration is to be avoided to minimize the risk of injury to the cavernosal artery during this procedure.

An initial 20 to 30 mL of corporal blood is aspirated while the operator milks the corpus with the left hand. This initial aspiration is continued until the original egress of dark blood ceases and bright red arterial blood is obtained. Because multiple anastomoses exist between the 2 corpora cavernosa, bilateral aspiration is not required.

If detumescence is achieved after initial aspiration, no further treatment is required. Simple aspiration is most likely to be successful if the priapism has been present for <24 hours. If retumescence occurs, then repeated aspiration, followed by an equal exchange of an alpha-agonist solution for aspirated corporal blood, should be carried out (i.e., 20 to 30 mL of a phenylephrine/normal saline solution [10 mg of phenylephrine in 500 mL of normal saline] in exchange for 20 to 30 mL of aspirated corporal blood). Some clinicians add 2500 to 5000 units of heparin to the solution, but the value of heparin is unproven. The use of beta-agonists for

**TABLE 59-4** -- Treatment of Priapism Based on Etiology

A. Terbutaline 0.25 to 0.5 mg SQ in the deltoid muscle area or thigh for all patients with priapism (may be repeated in 15-20 min); alternatively 5 mg terbutaline PO may be used (1 dose)

B. Reversible causes

  1. Sickle cell anemia

    a. Packed RBC transfusion

    b. Hyperbaric oxygenation (investigational)

  2. Iatrogenic injection of $PGE_1$ , papaverine or phentolamine for impotence

    a. Corporal aspiration of 30-60 mL of blood, followed by observation

     1) Detumescence: no further treatment

     2) Persistent erection: inject an equal volume of alpha-agonist (i.e., phenylephrine, 10 mg in 500 mL of normal saline)

  3. Leukemic infiltration

    a. Specific chemotherapy

| |
|---|
| 4. Medication (phenothiazines, trazodone) |
| a. Corporal aspiration, observation |
| b. alpha-Agonist instillation, observation |
| c. Heparin irrigation of corporal bodies, observation |
| d. Corpus cavernosum-spongiosum shunt |
| C. Nonreversible causes |
| 1. Idiopathic |
| 2. High spinal cord lesion |

corporal injection or irrigation also has been reported, and the advantage of pure alpha-agonists over beta-agonists is a subject of debate. O'Brien and associates recommend a 1:100,000 solution of epinephrine (1 mg/100 mL of saline), irrigating with 2 to 3 mL at a time to a maximum dose of 0.1 mg (10 mL). [19] Alternatively, 1 mg of epinephrine can be added to 1 L of saline, with irrigation performed using 20 to 30 mL aliquots. A norepinephrine solution also may be used (Table 59-5) . Some clinicians wrap the penis snugly in an elastic wrap with the needle securely in place following aspiration to discourage retumescence and to decrease hematoma formation of the penile shaft.

**Complications**

Although hematoma and infection can occur with properly performed aspiration, these complications are infrequent. Both phenylephrine and epinephrine can be absorbed systemically, with the potential for toxic effects. [18] Therefore, the intracavernosal use of vasoactive agents is contraindicated in patients with conditions sensitive to these agents (e.g., severe hypertension, dysrhythmias, monoamine oxidase inhibitor use). Blood

pressure and cardiac rhythm should be closely monitored throughout the procedure. Supplemental O$_2$ should be considered in any patient with underlying respiratory or cardiac disease. Because impotence is a well-recognized complication following priapism, regardless of the cause or the promptness of therapeutic intervention, the patient must be advised both verbally and in writing of this potential complication.

# ADJUNCTIVE TESTING IN

ACUTE SCROTAL PAIN

Establishing a diagnosis in the patient with acute scrotal pain creates significant physician anxiety. The condition most

| **TABLE 59-5** -- Equipment Needed for Aspiration of Corpus Cavernosum for Priapism |
| --- |
| 27-ga needle (for penile block) |
| 12-mL syringe (for local anesthetic) |
| 1% lidocaine without epinephrine (for penile block) |
| Sterile drapes |
| Gauze sponges |
| Povidone-iodine (or alternative) preparation solution |
| 19-ga butterfly needles (for aspiration) |

| Two 30-mL syringes (for aspiration and injection) |
| --- |
| Sterile basin for aspirated blood |
| Blood gas syringe with cap |
| Injection fluid (1 of the following vasoactive agents * is diluted with 500 mL of normal saline and up to 20 to 30 mL is administered; 5000 units of heparin added to solution is optional; see text) |
| Phenylephrine, 10 mg/500 mL of saline |
| Norepinephrine, 1 mg/500 mL of saline |
| Epinephrine, 0.5 mg/500 mL of saline |

* Systemic absorption of vasoactive agents may occur with adverse cardiovascular effects.

easily confused with torsion of the testicle is acute epididymitis. The prompt diagnosis of testicular torsion and differentiation of this condition from epididymitis can be quite difficult, but it is obviously crucial to the patient's care. The treatment of acute epididymitis requires appropriate antimicrobial and supportive therapy. The treatment of testicular torsion requires emergent operative intervention, not adjunctive testing! [20]

Torsion of the testicle is the most frequent cause of acute testicular pain in celibate men younger than 20 years. One pediatric series of patients presenting with a painful scrotum reported a 6% incidence of epididymitis vs a 42% incidence of testicular torsion and a 32% incidence of torsion of the appendix testis and epididymis. [21] It is interesting to note that pyuria was seen in 10% of the patients with testicular torsion and in 80% of the children with epididymitis. In men 20 years or older, testicular torsion is less common, but it must always be considered in the differential diagnosis as it may occur well into the seventh decade of life. [22]

In contrast, epididymitis is an *uncommon* condition in young celibate boys unless a congenital lower urinary tract abnormality is present that promotes urinary tract infection

with subsequent epididymitis.

As with all difficult diagnostic dilemmas, the patient's history is of paramount importance in establishing the correct diagnosis. The evaluation of acute scrotal pain is no different. In considering epididymitis, detailed and often probing questions must be asked regarding the patient's work habits and his sexual experiences. The pathophysiology of epididymitis emanates from the generation of increased pressure in the prostatic urethra as a result of either heavy lifting (e.g., in roofers or construction workers), detrusor external sphincter dyssynergia (e.g., in patients with neurogenic bladder dysfunction), or iatrogenic promotion of a Valsalva maneuver for whatever reason. These mechanisms force sterile or infected urine out through the ejaculatory ducts into the vas deferens (urethrovasal reflux) then retrograde down the vas into the tail of the epididymis (globus minor). This may initially cause nonspecific lower quadrant abdominal pain or inguinal canal pain (vasitis) before it causes scrotal or testicular pain, and vasitis should be included in the initial differential diagnosis in any male presenting with lower abdominal pain.

Sexually transmitted diseases with subsequent epididymitis are quite common in sexually active men younger than 40 years, usually associated with chlamydial or gonococcal urethritis. Signs and symptoms of infection may be minimal when the patient is first examined. [23] The presence of genitourinary infection, a history of previous sexually transmitted diseases, or a history of multiple sexual partners and the absence of penile protection during intercourse may all be important clues to the diagnosis of epididymitis. In older men, epididymitis is usually due to urinary tract infection secondary to outlet obstruction or urethral stricture disease. In such cases, enteric gram-negative bacilli and *Pseudomonas* species are the predominant organisms. If one cannot be certain after taking a careful history and examining the patient that epididymitis or torsion of the appendix testis or epididymis is the correct diagnosis, then urologic consultation to exclude testicular torsion is required. A clinical diagnosis of epididymitis should be based on the focal finding of epididymal induration and tenderness (the vas deferens also may be involved) in the proper patient population. The site of tenderness is best discerned when examining the patient in the relaxed supine position. Although testicular tenderness can develop with advanced epididymo-orchitis, the time course is generally gradual and on an order of days. However, marked testicular tenderness in a patient with advanced epididymo-orchitis may reflect secondary testicular ischemia and also warrants urologic consultation. [24]

The "gold standard" for the diagnosis of testicular torsion is scrotal exploration. [25] However, some clinicians incorporate radionuclide testicular scanning or color Doppler ultrasound examination in their diagnostic workup. [26] Unfortunately, these studies are time-, technician-, and reader-dependent examinations in a clinical situation in which *TIME IS TESTICLE*. The quintessential point in testicular torsion is that there are no markers, signs, or symptoms that will distinguish incomplete from complete testicular ischemia. One minute there may be total absence of blood flow to the involved testis, the next minute sufficient spontaneous detorsion may have occurred to provide temporary testicular perfusion. This is the dilemma. Scrotal exploration, detorsion, and orchiopexy or orchiectomy of the involved testis and orchiopexy of the contralateral

testis, is a relatively benign, straightforward operative procedure.

Remember that intravaginal testicular torsion is a congenital bilateral abnormality. The ischemic testis must be detorsed and pexed with nonabsorbable (e.g., nylon, polypropylene), rather than absorbable (e.g., chromic, Vicryl), suture. The torsed testis that is pexed with absorbable suture remains at risk for subsequent postoperative torsion. And last, orchiopexy of the nonischemic contralateral testis is mandatory to ensure prevention of future torsion.

Once the diagnosis of testicular torsion is suspected, a call should be placed immediately to notify a urologist of the suspected diagnosis, the perceived need for surgical exploration, and the fact that you will be attempting testicular detorsion while awaiting patient transport to the operating room. At some point before the patient leaves the ED, meticulous charting to document time, suspected diagnosis, notification of the urologist, and any manipulation of the affected

testis must be done. All efforts are then focused on attempting testicular detorsion.

## Doppler Assessment of Testicular Perfusion

### Indications and Contraindications

The most important nonoperative adjunctive procedure in suspected testicular torsion is testicular *detorsion*. This can be carried out with or without the aid of a Doppler stethoscope. Using the Doppler stethoscope alone to make a diagnosis of testicular torsion or successful detorsion is *not* recommended. Nonetheless, the Doppler device may be used to support one's clinical assessment in cases strongly suggestive of epididymitis or to monitor the response to detorsion efforts.

### Equipment

In circumstances in which one wishes to use the Doppler, the directional Doppler operating on a 10-MHz transducer is recommended. See Chapter 70 for further discussion of Doppler ultrasound physics. A pencil transducer is the most appropriate (e.g., the Model 806 directional 10-MHz Doppler, Park Electronics Laboratory, Beaverton, Ore). The Doppler response may be transmitted over a loudspeaker, copied on a measuring device, or transmitted through a stethoscope to the physician. The higher the frequency of the transducer, the narrower the beam and the less the distance of transmission through the tissues. In this respect, a 10-MHz transducer is preferred over a lower-frequency transducer for examination of the testicle.

### Technique

Because scrotal tenderness often precludes adequate examination, anesthesia may be needed. A cord block using 1% lidocaine may be applied at the external ring (see discussion of this block under Testicular Detorsion below). The area of maximum testicular swelling is determined. An aqueous transmission gel is then placed over the

scrotum. Holding the testicle in one hand and the Doppler probe in the other, one displaces as much of the scrotal wall as possible between the skin and the underlying testicle. The Doppler probe should be placed in the center of the testicle, pointing slightly caudally so that pulsations in the cord are not detected (Fig. 59-9) . (Firm probe pressure "focuses" the ultrasound waves deep to the scrotum into the testis.) The pulsation in the tender, ipsilateral testicle is then compared with that in the contralateral testicle. Decreased or absent flow to the ipsilateral testicle is most surely a result of torsion. Increased flow to the ipsilateral testicle may be a result of epididymitis, inflamed scrotal tissue, a false signal from either the cord or the examiner's fingers, or a false comparison with a contralateral partial torsion of the testis. [27]

The *funicular compression test* should be performed to confirm that flow signals are related to perfusion to the testicle. If the increased signal lessens on compression of the patient's spermatic cord, then the signal is most probably coming from the patient's testicle and not from inflamed scrotal tissues. If there is no change in the signal on adequate cord compression, the increased flow may be originating in inflamed scrotal tissue, and torsion should still be suspected.

### Complications

Absolute proficiency with the Doppler stethoscope is difficult to achieve, and examination findings are subject to misinter pretation. The greatest risk comes in those cases that represent a later stage of testicular torsion. Although the testes may not be necrotic, a secondary inflammatory response may develop as a result of the underlying ischemia. Any perceived Doppler signal in the hyperemic surrounding scrotum may be misinterpreted by an inexperienced examiner as epididymitis rather than late torsion with an associated inflammatory response, the result of which may be inappropriate treatment. Obviously, the affected testis would remain ischemic, but more important, the contralateral testis would remain at risk for later torsion.

Failure to perform the funicular compression test may lead to the mistaking of scrotal blood flow for testicular blood flow. The Doppler signal should fade and then promptly return as the spermatic cord is first compressed and then released. With an incomplete torsion, one may also hear an attenuated arterial pulse in the testicle. For these reasons, it should be evident that one cannot rely entirely on the Doppler ultrasound examination. [28]

## Manual Detorsion and Spermatic Cord Anesthesia

Manual detorsion is performed in the following manner. Advise the patient that the procedure will be uncomfortable and painful and offer light sedation if it seems appropriate. The rationale for not using spermatic cord anesthesia with attempted detorsion is that the anesthesia takes away an important subjective end point (i.e., relief of the patient's pain after manipulation of the testis). However, many authors do advocate spermatic cord anesthesia prior to detorsion, and if anesthesia of the spermatic cord is elected, it can be done in the following manner.

**Spermatic Cord Anesthesia**

Local anesthesia of the spermatic cord using 1% plain lidocaine is usually done at the external inguinal ring. [29] The skin is first prepared with an iodophor solution. The cord can usually be grasped between the thumb and index finger, and 10 mL of 1% plain lidocaine can be directly injected into the cord. If the cord is swollen, as it often is in testicular torsion, or if the testicle is lying very high in the hemiscrotum as a result of spermatic cord torsion (so as to preclude

grasping), the cord may be palpated at the pubic tubercle as it passes over the pubis and the lidocaine injected at this landmark. Lee and colleagues [30] were able to perform manual detorsion with local spermatic cord anesthesia in 70% of their adult cases of torsion. Kresling and associates [31] had success in 15 of 16 patients and noted a fair amount of associated cremasteric muscle spasm, which must also be relieved. In their experience, torsion usually resulted from an initial lateral-to-medial rotation (Fig. 59-10) , with an occasional caudal-to-cranial component.

**Manual Detorsion**

The goal of manual detorsion is to reestablish or increase blood flow to a previously ischemic testis. This should be done in conjunction with preparation of the operating suite. *It should never delay operative intervention*.

Before initiating detorsion, one must ensure that the patient is as comfortable as possible in a reclining or supine position. Lithotomy position gives the examiner the most access to the patient's genitalia and prevents the patient from retreating during the procedure. If light analgesia/sedation are selected they should be implemented at this time.

Manual detorsion begins with the physician standing comfortably at the side of the bed or stretcher, preferably on the patient's right side if the physician is right handed, or vice versa. Detorsion is begun just as one would open a book (i.e., an initial 180° detorsion of the patient's right testis is done in a counterclockwise fashion. The patient's left testis is detorsed 180° in a clockwise fashion (see Fig. 59-10) . Pain relief and an increase in the Doppler signal are the objective end points. If one rotation relieves some but not all of the pain, continue with another rotation. If the initial detorsion is mechanically difficult (which it will be if you are detorsing in the wrong direction) or makes the pain worse, detorse the testis in the opposite direction and observe your result. The objective success or failure of any testicular manipulation can be substantiated by an increase in Doppler signal and the patient's relief of pain.

With successful detorsion, the testicle returns to its normal anatomic position. Resolution of induration and swelling of the spermatic cord, testis, and epididymis will depend on the degree and duration of ischemia. Thus, the more severe the torsion and the longer it has been present, the longer it will take for the edema and induration to resolve. With significant ischemia, the entire epididymis often becomes enlarged like a

link sausage (uncommon in epididymitis except in severe cases or those that are initially misdiagnosed or seen late in their clinical course), and the testis becomes quite firm, simulating a testicular tumor. In the author's experience, both of these reversible changes usually resolve over 3 to 4 hours. Occasionally the testis will torse in the opposite direction (medial to lateral) or have multiple twists. This may become apparent as the clinician assesses the results of the detorsion procedure by palpation, relief of edema, and return or increase of the Doppler signal. Even though manual detorsion will save an ischemic testicle, *it should not be substituted for definitive scrotal exploration.*

# URETHRAL CATHETERIZATION

## Alternatives to Catheterization

The merits of alternative approaches to urine specimen collection over patient catheterization are dependent on the patient's age and clinical setting.  In children, the collection technique reported by Amir and coworkers consists of placing the young child on his or her back. [32] In very young children, collection and analysis of spontaneously voided urine specimens following penile cleansing or suprapubic "finger tap" showed identical urine

culture results to those specimens obtained by suprapubic aspiration. [32] The applicability of this to a busy ED is questionable. Often, it is possible to collect a spontaneously voided midstream specimen in a child if ED personnel are prepared in advance. The problem is that the voiding events tend to occur as the child is undergoing venipuncture, spinal tap, or attempted urethral catheterization. And more important, if the first specimen is missed or inadequate, how long are we willing to wait for another chance? Urethral catheterization is quick, definitive, and routinely used.

In adult men without anatomic lesions, first-voided specimens can define the presence or absence of culture-proven bacteriuria. [33] This certainly represents a user-friendly approach to urine collection in a busy ED and needs to be carefully considered.

In adult women, collection of clean-catch midstream specimens has been found to be as reliable bacteriologically as catheterized specimens. [34] A few caveats are worth mentioning: patients must sit backward on the toilet when collecting the specimen (i.e., facing the wall, which theoretically promotes labial spreading). Of more concern is the fact that these studies excluded patients with vaginitis, urologic abnormalities, pregnancy, and vaginal bleeding. These are the clinical circumstances for which urine is commonly examined in young women visiting the ED. In this "at risk" population, catheterized urine specimens are preferred.

Urethral catheterization seems a simple task--insertion of one tube into a larger tube. Nonetheless, many difficulties may arise. Patients often remember catheterization as either painful or uneventful and reflective of the operator's expertise, confidence, and gentleness.

Patients are often apprehensive about catheterization. If the physician shows concern

regarding position and exposure, the patient will be reassured. A moment should be spent in making sure a patient is positioned comfortably and appropriately for the procedure. Although adequate exposure may be obtained from a frog-legged position, the use of a table with stirrups (lithotomy position) is ideal, especially for female catheterization.

Anticipation and preparation of all materials necessary for urethral catheterization beforehand is reassuring to the patient. It is frustrating for the health care provider and upsetting to the patient when he or she is told "not to move or touch anything" while a search is made for additional equipment. Most catheterizations are performed using a standard catheterization tray. Often, these trays contain more equipment than is truly needed. This necessitates opening the tray and establishing a sterile field at the bedside, selecting those items that will be needed, and discarding the rest of the equipment. Once the penis or labia has been touched in preparation for the procedure, the touching hand is contaminated and ideally should not be handling any of the sterile equipment. When a standard catheterization tray is not used or is not available, catheterists should go through the anticipated procedure mentally to secure all of the appropriate equipment before actually starting the procedure.

## Indications and Contraindications

Urinary catheterization and instrumentation are rarely a primary cause of urinary infection in otherwise healthy patients who urinate normally and carry small amounts of post-void residual urine. As with any procedure, catheterization needs to be limited to those clinical situations in which the benefits outweigh the risks. The following are considered to be indications for urethral catheterization:

1. Acute urinary retention.
2. Urethral or prostatic obstruction leading to compromised renal function.
3. Urine output monitoring in any critically ill or injured patient.
4. Collection of a sterile urine specimen for diagnostic purposes.
5. Intermittent bladder catheterization in patients with neurogenic bladder dysfunction.

Urologic study of the lower urinary tract.Urethral catheterization should be avoided when other less invasive procedures will be as informative. The only absolute contraindication to urethral catheterization is the trauma patient with suspected urethral injury as evidenced by blood at the urethral meatus, an abnormal-feeling or high-riding prostate on rectal examination, or penile, scrotal,

---

**TABLE 59-6** -- Sterile Equipment Required for Urethral and Suprapubic Catheterization

| Urethral Catheterization |
|---|
| Foley catheter of appropriate size |
| Water-soluble lubricant for catheter |
| 10-mL syringe of sterile water for Foley balloon |
| Sterile drainage bag with tubing |
| Sterile drapes |
| Sterile gloves |
| Povidone-iodine |
| Sterile gauze pads or cotton balls |
| Sterile specimen cup with lid |
| Cloth, paper, or plastic tape (to secure catheter to trunk or leg) |
| Benzoin (for increasing tape adherence) |

| |
|---|
| Forceps |
| **Suprapubic Catheterization** |
| Foley catheter of appropriate size * |
| Sterile gloves |
| Sterile drapes |
| Sterile drainage bag with tubing |
| Sterile specimen cup with lid |
| Cloth, paper, or plastic tape (to secure catheter to trunk or leg) |
| Benzoin (for increasing tape adherence) |
| **Materials in Cook "Peel-Away" Catheter Set** |
| 10-mL syringe |
| 25-ga needle |

| |
|---|
| 22-ga needle |
| 1% plain lidocaine |
| Povidone-iodine swab-sticks |
| Fenestrated drape |
| Gauze sponges |
| Disposable scalpel |
| Suture with needle |
| Introducer needle |
| Curved J-tipped wire guide |
| Peel-away sheath |
| Introducer |

* Select the sheath size (in catheter set) based on the Foley catheter size. Generally, the sheath is 1 to 2 Fr sizes larger than the catheter (e.g., use an 18 Fr peel-away sheath with a 16 Fr Foley).

| TABLE 59-7 -- Pediatric Urethral Catheter Size | |
| --- | --- |
| **Age Group** | **Pediatric Catheter Size** * |
| Infants | 5-8 Fr feeding tube |
| 1-3 yr | 8 Fr feeding tube |
| 4-6 yr | 10 Fr feeding tube |
| 7-12 yr | 10-12 Fr red rubber catheter (Robinson) |
| >12 yr | 14 Fr red rubber catheter (Robinson) |

* 6 Fr is the smallest balloon catheter and is quite flimsy; would recommend 8 Fr in most cases; 12 Fr is the smallest coude catheter.

or perineal hematoma. These findings dictate the need for retrograde urethrography to define the integrity of the urethra prior to any attempted urethral catheterization.

## Equipment

The equipment listed in Table 59-6 is included in most standard catheterization trays and must be at hand before attempting urethral catheterization. The catheterist should check the list of contents *before* opening the tray, as some trays do not include certain items. For most routine adult in-and-out catheterizations, a 14 Fr red rubber catheter or Foley balloon catheter is adequate. In infants or neonates, a 2 or 5 Fr feeding tube taped in place produces the least amount of urethral trauma. In older boys, a 5 to 12 Fr red rubber catheter or Foley balloon catheter may be used. Table 59-7 lists appropriate-sized catheters and feeding tubes for all ages. A 14-18 Fr coude catheter should be considered after unsuccessful passage of a straight Foley balloon catheter or in any male patient with known enlargement of the prostatic median lobe. If a coude catheter is not available, a larger 18 to 22 Fr Foley balloon catheter can be tried. In a male patient with a urethral stricture in whom attempts at catheterization with a straight Foley or coude catheter have failed, passage of filiforms and followers is the next logical

step. This requires special expertise, but it can be learned quickly. If immediate bladder access is required in this circumstance or in any emergency, suprapubic placement of a peel-away sheath and Foley balloon catheter using the Seldinger technique will be needed. [36]

## Anatomic Considerations

### Female Catheterization

The female urethra is a short (approximately 4 cm), straight tube, usually of wide caliber, lying on top of the vagina. It must be approached between double labia, and the urethral meatus is occasionally hidden and not obvious (in contradistinction to that of most males, except those with hypospadias). If the female patient nervously adducts her legs, successful catheterization is most uncertain.

The female urethral meatus is an anteroposterior slit with rather prominent margins that is situated directly superior to the opening of the vagina and approximately 2.5 cm inferior to the glans clitoris (Fig. 59-11) (Figure Not Available) . [37] It is the first of 3 orifices encountered when examining any female genitalia cephalad to caudad in the lithotomy position. The urethral meatus may be especially difficult to find in the very young infant and the older postmenopausal woman. Anticipation of this and knowledge of the anatomic variances will help to modify any patient discomfort associated with needless catheter tip probing, which is an unsettling experience for both the patient and the catheterist.

Occasionally, the urethral meatus recedes superiorly into the vagina and is not immediately visible, because of either prior surgical procedures or atrophic postmenopausal changes. Anticipation of such cases will allow the examiner to gently advance an index finger into the vagina in the superior midline. The urethral meatus can usually be palpated and often visualized as a soft mound surrounded by a firmer ring of supporting periurethral tissue. Rarely, the meatus will have receded so far superiorly that it cannot be visualized at all, and catheterization must be carried out by palpation alone. From the meatus (if the patient assumes a supine position), the urethra proceeds straight back to slightly downward as it advances into the bladder just behind the symphysis pubis (Fig. 59-12 *A*).

In women with a *urethrocele or cystourethrocele,* in whom the urethra or the bladder falls into the vagina, the "normal" urethral course may be more significantly posterior (Fig. 59-12 *B* and *C*). The normal anatomic relationships in these situations may be recreated by spreading the index and long fingers and placing them along the superior vaginal wall and gently applying upward support (Fig. 59-12 *D* and *E*). This reconstitutes the normal anatomic relationships and permits straight, rapid urethral catheterization.

Because the female urethra is so short, only half the total length of the catheter has to be inserted before it is safe to inflate the Foley balloon.

**Male Catheterization**

Because the urethral meatus is usually evident in most males, it may seem a simple matter to insert a urethral catheter. [38] Yet catheterization can be quite difficult. The normal male urethra is approximately 20 cm long from the external urethral meatus to the bladder neck (Fig. 59-13) (Figure Not Available) . The posterior prostatic urethra is approximately 3.5 cm long, and the contiguous external sphincter or urogenital diaphragm that encompasses the membranous urethra is 4 cm from the bladder neck. In males, any catheter must be fully inserted to the balloon-inflating side-arm channel before it is safe to inflate the balloon (Fig. 59-14) . At the first egress of urine from the catheter, the balloon is just passing through the membranous urethra. The catheter still has 3 cm to go before clearing the bladder neck. Inflation of the Foley balloon at any point other than full insertion of the catheter may result in iatrogenic urethral injury.

The male urethra is relatively fixed at the level of the urogenital diaphragm and symphysis pubis; traction downward on the penis kinks and promotes urethral folding at the level of the penile suspensory ligament (Fig. 59-15 (Figure Not Available) *A*). This creates a level of spurious obstruction. For this reason, the penis should always be held taut and upright during any urethral instrumentation including catheterization (see Fig. 59-15 (Figure Not Available) *B*). The catheter then needs to make only a single curve rather than a complex S curve as it traverses into the bladder.

## General Procedure

As stated previously, urethral catheterization must be done using sterile technique. Both male and female patients have special urethral meatal considerations. In the male, absolute total control of the penile foreskin is paramount to ensuring success. This is true for any male patient, but it is especially important in uncircumcised patients. Prior to establishing a sterile field, the foreskin should be retracted to its fullest extent proximal to the glans penis (Fig. 59-16 *A*). A standard 4 × 4 gauze pad is unfolded, then refolded in its longest dimension, and carefully wrapped around the retracted foreskin at the level of the coronal sulcus (Fig. 59-16 *B*). This will prevent the tendency for normal anatomic foreskin reduction during catheterization and provide a continuously dry and sterile field. The folded 4 × 4 gauze surrounding the foreskin is secured between the nondominant long and ring fingers when beginning the procedure and is not released until the procedure is completed (Fig. 59-17) . This position leaves the nondominant index finger and thumb available for manipulating the catheter. Following catheterization, removal of the 4 × 4 pad and reduction of the penile foreskin to its normal anatomic position will prevent the development of iatrogenic paraphimosis.

In the female patient, the catheterist's nondominant thumb and index finger are used to separate the labia and create exposure of the urethral meatus. Once meatal visualization is achieved, the nondominant hand should not be removed (see Fig. 59-11 (Figure Not Available) *B*). This underscores the importance of proper equipment preparation. Access to antiseptic solution, viscous lubricating jelly, the appropriate catheter, and the urinary collection device should be prepared in advance to permit

single-handed sterile use.

Following exposure of the urethral meatus in the female and the glans penis and urethral meatus in the male, an antiseptic solution (e.g., povidone-iodine) soaked into cotton balls or oversized cotton-tipped applicators is used to cleanse the exposed meatus and surrounding tissues. This is best done by hand but can also be done using the plastic forceps in the catheterization tray. The cleansing circular motion should begin on the urethral meatus and proceed outward, intentionally moving any debris toward the periphery and thus creating a sterile field.

An appropriately sized catheter (10 Fr is adequate for small children, whereas 14 to 16 Fr is commonly used in adults) that has been lubricated with viscous rather than inspissated lubricating jelly is gently passed by hand, or with the aid of a hemostat or plastic forceps, into the urethra and upward into the bladder. Injection of the male urethra with 5 mL of 2% viscous lidocaine (Anestacon) or a similar anesthetic lubricant administered through a syringe can be helpful for urethral distention and topical anesthesia. Regardless, the patient should be advised of mild urethral discomfort and the potential urge to void. During slow, gentle passage of the catheter, one should be aware of the anatomic considerations discussed previously. A catheter that inadvertently enters the vagina should be discarded.

After passing the catheter "to the hilt" in all male patients, the balloon should be slowly inflated with 10 mL of air or tap water. Sterile water or saline is not required. Most 5 mL balloons will easily accommodate up to 30 to 50 mL of air or water without bursting. Obvious resistance or patient discomfort on balloon inflation should signal potential erroneous urethral positioning and mandates reevaluation. If this occurs, the Foley balloon should be immediately deflated and the catheter repositioned or withdrawn slightly, then passed "to the hilt" again prior to balloon reinflation. If this is unsuccessful a second time, catheter removal and urethral evaluation for a potential obstructive problem or false passage using retrograde urethrography is recommended (see Retrograde Urethrogram below).

Following successful catheter passage and Foley balloon inflation, the catheter should be slowly withdrawn until the approximation of the balloon with the bladder neck precludes further withdrawal. The catheter is then connected to either a sterile leg bag or closed-system bedside drainage bag. If the patient will be released with an indwelling Foley catheter, it can be initially connected to a leg bag, which is then comfortably fastened to the lower thigh and upper calf. The patient and family must be instructed regarding proper care of the catheter and drainage device. In most other cases, the catheter may be secured either to the thigh or the lower abdomen (preferred with males) with adhesive tape or simply placed under the knee and left to drain dependently into the bedside drainage bag.

The use of antibiotics before simple catheterization is not warranted. However, patients with known valvular heart disease, suspected urinary tract bacteremia, chronic urinary tract infection, urethral stricture disease, or outlet obstruction associated with infection are considered to be at risk for procedure-induced bacteremia. In these patients, any urinary instrumentation, especially urethral dilation, requires early prophylactic

parenteral gram-negative antibiotic coverage before any instrumentation is begun. These patients must also be released with appropriate antibiotic coverage and be scheduled for early urologic follow-up within 1 to 2 days. [39]

## Difficulties in Male Catheterization

### Phimosis

Physiologic adhesions between the foreskin (prepuce) and glans penis with associated inability to retract the foreskin are normal in children. This condition must be distinguished from phimosis, which is the inability to retract the foreskin proximally over the glans penis due to recurrent inflammation or trauma, which results in gradual scarring of the preputial opening. [40] It is not essential to retract the foreskin in uncircumcised boys at the time of catheterization if the urethral meatus can be visualized. At birth, the foreskin is fully retractable in only 4% of boys. Sufficient foreskin retraction to visualize the urethral meatus is possible in only half of newborn boys. Although the foreskin can be retracted completely in only 20% of 6-month-old boys, 90% of 3-year-old boys have a fully retractable prepuce. By 17 years of age, the foreskin should be physiologically separated and fully retractable in all males unless they have experienced secondary infections or repeated preputial trauma. [41]

The foreskin, especially in diabetics, is susceptible to recurrent infections and inflammation. A scarred, contracted, difficult-to-retract preputial opening may result, leading to phimosis. Phimosis precludes optimal hygiene of the glans penis and coronal sulcus, resulting in an increased risk of bacterial infection, transmission of sexually transmitted diseases (especially human immunodeficiency virus), and the development of penile malignancies. Occasionally the phimotic opening becomes so tight that the meatus cannot be visualized or palpated, even for nonsterile catheterization. If the patient requires catheterization, it will be necessary either to perform a dorsal slit of the foreskin to expose

the glans and urethral meatus sufficiently for cleansing and catheterization or to dilate the phimotic opening sufficiently to identify the urethral meatus and blindly pass the catheter. These procedures are discussed at the beginning of the chapter.

### Edema of the Foreskin

Patients with penile trauma, paraphimosis, anasarca, or significant lymphatic obstruction from irradiation or cancer may have marked edema of the foreskin, subsequently burying the urethral meatus and glans penis in several centimeters of boggy foreskin (Fig. 59-18) . Because the latter group of patients often require careful fluid monitoring, they may need an indwelling catheter. The physician's first responsibility is to disclose the proper etiology for the foreskin edema. This will require enough foreskin manipulation to identify the glans penis, coronal sulcus, and urethral meatus, as well as the relationship of the prepuce and preputial opening to these structures, to ensure the absence of foreign body strangulation or paraphimosis (Fig. 59-19) .

Two separate methods of visualizing the glans penis and urethral meatus are available to the physician. [42] The simplest method is to manually compress the swollen foreskin by hand or between opposing cold packs in an attempt to reduce the edema fluid (see Fig. 59-5 *C* and *D*). Snugly wrapping the distal penis with Elastoplast for 10 minutes may also be helpful. In most mild cases, manual compression is often successful, and no further maneuvers are required. In more severe cases, the foreskin may be 6 cm thick in diameter. In such cases, the least traumatic but unfortunately most cumbersome way to visualize the glans penis and urethral meatus is to use a pediatric-sized vaginal speculum. The outer surfaces of the speculum are lightly lubricated, and the speculum is inserted into the opening of the edematous foreskin. The operator then opens the speculum gently and visualizes the glans penis, similar to viewing the cervix. Commonly, the 2 leaves of the speculum will slip out of the foreskin, making adequate visualization quite difficult. Subsequent meatal cleansing and catheterization performed with plastic instruments is cumbersome and is not recommended. It is much easier to palpate the urethral meatus with the index finger of the nondominant hand, then use the same index finger as a guide to blindly insert the Foley or coude catheter into the urethra and upward into the bladder.

**Meatal Stenosis**

The urethral meatus may be either congenitally or secondarily narrowed by scarring, resulting in meatal stenosis. The stenosis may prevent admission of a normal-sized catheter. If the meatus admits a small-caliber tube (i.e., 5 Fr pediatric feeding tube or larger), this may be all that is required for the short term. It should be remembered that the inner diameter of the feeding tube or catheter is the important diameter for urinary drainage. A smaller, single-lumen tube may provide better drainage than a larger, double-lumen Foley balloon catheter.

If a larger-caliber catheter or a Foley balloon catheter is required, meatal dilation or meatotomy may be necessary. Meatal dilation is accomplished by repeatedly inserting larger meatal dilators to a certain end point. This procedure is painful and should be performed with topical meatal anesthesia in conjunction with light IV sedation.

On occasion, a ventral meatotomy may be required in men in whom repeated instrumentation or long-term catheterization has resulted in severe meatal stenosis. Using a 25- or 27-ga needle, infiltration of the ventral midline of the glans from the coronal sulcus to the distal tip of the meatus is performed with 1% plain lidocaine. A straight hemostatic clamp is then gently applied to the anesthetized area, with one jaw inside the meatus and the other on the anesthetized ventral midline of the glans. After the hemostat has been closed and left in place for 3 to 5 minutes, it is opened and removed. The crushed tissue is then cut with straight scissors. Some physicians place a running hemostatic 4-0 chromic suture through the apex of the meatal incision and carry it out distally on both sides of the incision to prevent restenosis of the meatus. A 16 or 18 Fr Foley catheter left indwelling for 3 to 4 days may serve the same purpose.

**Urethral Stricture**

Urethral obstruction encountered in the anterior or bulbous urethra during catheterization is usually the result of urethral stricture disease. [43] Urethral strictures develop as a result of trauma, infection (especially sexually transmitted diseases), lower urinary tract instrumentation, or long-term indwelling catheter drainage. Strictures may remain asymptomatic for a period of time, but eventually most become symptomatic and cause urethral voiding symptoms, hematuria, or bloody urethral discharge that may or may not be associated with infection. *Manual force should not be used to negotiate or to dilate urethral strictures*. Force merely promotes a vicious cycle of false passages, bleeding, and eventual increased scarring, which makes catheterization more difficult. Inability to negotiate a urethral stricture with a simple straight Foley catheter or coude catheter leads to consideration of urethral dilation using filiforms and followers.

*Filiforms* are very narrow, flexible, solid catheters, usually not exceeding 4 Fr in caliber. They are not dilators. Their sole function is to locate and successfully negotiate a strictured urethral segment. Each filiform has a straight or pigtailed (curved) distal end and a proximal female-threaded coupling into which the distal male end of a follower may be threaded. Following generous topical anesthesia with 2% lidocaine (Anestacon), the previously prepared penis is grasped between the nondominant long and ring fingers and stretched upward. The filiform is slowly passed down the urethra in an attempt to negotiate the strictured iris or narrowed segment of involved urethra (Fig. 59-20 *A*). This is all done by "feel" and experience and may require several attempts. It is always best to advance the filiform with the gentlest of pressure.

Filiforms should never be forced through the urethra. Any encountered resistance represents the potential edge of the strictured urethra or fold of urethral mucosa. Undue force applied to the filiform at this point may result in perforation of the urethral mucosa and creation of a false passage along the urethral body or inferiorly into the perineum. If the filiform meets resistance, it should be partially withdrawn, rotated 90° to 180°, then gently reinserted. If resistance is met again at the same location, the first filiform may be left in place (to fill up that particular obstructing site) and a second filiform advanced alongside it. A third and a fourth filiform may be necessary before one of them successfully navigates the narrowed aperture of the stricture and advances upward through the normal proximal urethra and into the bladder (Fig. 59-20 *B-D*). The sine qua non of success is the effortless passage of the filiform through the strictured area without spontaneous discharge of the filiform when it is released. Any amount of filiform return should alert the catheterist that the filiform has not negotiated the stricture and requires replacement. Pigtail filiforms (with a corkscrew-shaped tip) (Fig. 59-20 *E*), rather than straight-tipped filiforms, are often easier to advance over an abrupt urethral edge and through the smallest of strictured openings. Once through the stricture, the filiform is advanced until the threaded coupling is near the urethral meatus (Fig. 59-20 *F*).

Because filiforms are very pliable, they must be grasped securely. This is ensured by stabilizing the retracted foreskin and penis just proximal to the glans between the long and ring fingers of the nondominant hand as described previously (see Fig. 59-17) and holding the filiform between the nondominant index finger and thumb. This allows controlled attachment and subsequent detachment of graduated followers as serial

dilation is performed *G*).

A follower of the smallest caliber (usually 8 Fr) should always be selected first; it should be copiously lubricated and threaded onto the filiform. When it has been threaded completely (no threads showing), it is gently advanced without pressure through the upwardly stretched penis and urethra into the bladder (see *G*). Stretching the penis upward discourages telescoping of the urethra and subsequent kinking of the filiform and follower (see Fig. 59-15 (Figure Not Available) *A* and *B*). The follower is advanced into the bladder until there is spontaneous egress of urine from the follower, guaranteeing successful passage. Note that the side drainage holes of the 8 and 10 Fr follower are quite small and frequently become occluded with lubricating jelly. This may prevent spontaneous urinary drainage from the follower even though it has successfully negotiated the stricture and passed into the bladder. In this circumstance, easy passage of the 8 Fr follower into the bladder and the lack of gross blood or bloody drainage at the filiform-follower coupling when changing follower sizes are indicative of successful passage and dilation rather than of the creation of a false passage.

Absolute certainty of location of the 8 or 10 Fr follower can be established at any time by irrigating the follower with sterile saline, much like irrigating a Foley catheter. The irrigating fluid will dislodge any obstructing lubricating jelly from the side holes of the follower and will allow egress of urine from the follower. The entire dilating procedure is repeated with sequentially larger followers until one size larger than the proposed retention catheter is successfully introduced. Following completed dilation, the coupled filiform and follower are removed intact, the urethra is relubricated with topical lidocaine (Anestacon) anesthesia, and the previously selected 14 to 16 Fr balloon Foley or coude catheter is passed to its fullest extent into the bladder. After return of urine from the Foley ensures proper placement, the Foley balloon is inflated and the catheter is withdrawn and left to drain as described previously.

Occasionally, a urethral stricture is so dense and irregular that a filiform and 8 Fr follower may pass successfully, but the density and length of the stricture prevent further dilation. In this circumstance, the indwelling filiform and follower may be taped to the penile shaft for 1 to 2 days to provide adequate bladder drainage and probable softening of the stricture.

The following procedure is used to *tape the follower to the penis*. After wiping excess lubricant from the follower and from the penile glans and shaft, a small amount of tincture of benzoin is applied to the follower and to the unbroken skin of the penile shaft ( *not to the glans*). After the benzoin has dried, strips of paper tape 1.25 cm in width are placed longitudinally down the penile shaft without overlapping. The distal ends of each strip of tape are wrapped around the follower much like taping a nasogastric tube to the nose. These longitudinal strips will keep the follower in place. *It is important not to circumferentially wrap these strips around the penis*. If the tape were applied circumferentially, the tape might constrict venous and lymphatic return sufficiently to produce a relative paraphimosis.

Urethral dilation with filiforms and followers should be neither bloody nor excessively uncomfortable for the patient. If the procedure is bloody or uncomfortable or if no urine

is returned despite advancement of the follower for at least 24 cm, the physician should consider that the filiform may not be in the urethra, but instead has created a false passage. In such a situation, retrograde urethrography will define the urethral anatomy. In cases in which urethral instrumentation is unsuccessful for whatever reason, it may be necessary to place a suprapubic peel-away sheath cystostomy tube rather than persist with unsuccessful urethral dilation.

**Spasm of the External Urethral Sphincter**

The male patient may voluntarily or involuntarily contract the urogenital diaphragm (external sphincter), the striated urethral sphincter at the apex of the prostate. (This is especially true of trauma patients and men with neurogenic bladder dysfunction and pelvic floor spasms.) This produces spurious urethral resistance at approximately 16 cm from the meatus. Because increased abdominal pressure or voluntary perineal contraction causes reflex contraction of the external sphincter, the patient in these situations should be encouraged to lie supine and take slow, deep breaths, consciously trying to relax the perineum and rectum. Plantar flexion of the toes and ankles also aids in relaxation of the pelvic floor. Because the external sphincter is composed of striated muscle and fatigues within a few minutes, gentle but steady pressure exerted on the syringe or the catheter while the above maneuvers are undertaken usually results in successful catheterization. If these maneuvers do not result in successful passage of the catheter, the catheterist may be encountering an anatomic abnormality that will require definitive retrograde urethrography before instrumentation.

**High Bladder Neck**

Occasionally, a patient may have an enlarged intravesical portion of the prostate with a secondary high-riding bladder neck. The tip of the standard Foley catheter may encounter this intravesical portion of the prostate and may not readily pass above it into the bladder. Resistance is usually encountered after the catheter has been passed 16 to 20 cm into the urethra. Slow instillation or injection of 20 to 30 mL of sterile lubricating jelly into the urethra may allow the catheter to slip over the prostate and into the bladder. If this fails, a coude catheter may be inserted. This catheter has a bend in the tip, and one will almost always be able to maneuver it gently into the bladder (Fig. 59-21 *A* and *B*). Passage of the coude catheter often may be enhanced by having an assistant exert digital compression and flattening of the elongated prostate using a gloved finger in the patient's rectum (Fig. 59-21 *C*).

Use of a catheter guide or stylet is *best reserved for the urologist,* as such techniques encourage creation of a urethral false passage (Fig. 59-21 *D*). If the coude catheter cannot be advanced on its own or with the added help of an assistant, a scarred, fixed bladder neck contracture, usually the result of transurethral surgery, may be present. Such a contracture is often very difficult to blindly negotiate. An attempt with filiforms and followers may be warranted. Direct cystoscopic visualization and incision of the bladder neck obstruction by a urologist is usually required.

**Catheterization in the Patient with Pelvic Trauma**

The patient with pelvic trauma or a straddle injury presents special problems in urinary management. The patient may be in shock from associated injuries. Accurate minute-to-minute monitoring of urinary output requiring bladder catheterization may be required in the initial resuscitation. Furthermore, radiographic evaluation to define the extent of lower urinary tract injury may require a retrograde cystogram, which necessitates urethral catheterization. Prior to urethral catheterization in all trauma patients, the penis should be examined for evidence of blood at the urethral meatus, the absolute marker of urethral injury. [35] Blood at the urethral meatus, a "high-riding" or abnormal feeling prostate on rectal examination, and penile, scrotal, or perineal ecchymoses are all hallmarks of potential urethral injury and represent contraindications to empiric urethral catheterization without an antecedent normal retrograde urethrogram. [35]

The hazard of injudicious urethral catheterization in the pelvic trauma patient is the potential worsening of an already existing, less serious urethral injury that is often associated with such trauma. Pelvic fractures most often impart injury to the prostatomembranous urethra just above the urogenital diaphragm. The shearing pelvic fracture fragments transect the urethra and puboprostatic ligaments, thereby displacing the prostate superiorly from its normal anatomic attachment to the posterior surface of the pubic bone. A partial or complete urethral injury may occur. An appropriately recognized and treated partial urethral disruption may heal with little or no scarring. However, a complete urethral disruption requires surgical repair and usually results in some degree of postoperative urethral stricture and, occasionally, urinary incontinence and impotence, all of which portend significant morbidity. The danger of injudicious urethral catheterization in this situation is the potential conversion of a partial injury into a complete urethral injury, with its associated complications. Retrograde urethrography is the diagnostic procedure of choice in any suspected urethral injury (see Retrograde Urethrography).

In the event that contrast material flows easily from the urethra into the bladder without extravasation, complete urethral integrity is ensured, and an attempt to pass a 14 or 16 Fr Foley catheter should be made. If *any* urethral resistance is encountered other than that normally expected at the voluntary external sphincter (urogenital diaphragm) in a conscious, anxious patient, the catheterization should be immediately aborted and a urologist consulted. If passage is successful, the catheter balloon should be inflated and the catheter withdrawn until it approximates the bladder neck. At this point, it is left to dependent drainage, and very careful attention is paid to the initial bladder effluent. Any colored urine other than clear or yellow is considered gross hematuria and mandates evaluation of the bladder and upper urinary tract to disclose the source. If the retrograde urethrogram shows urethral extravasation *AND* passage of contrast into the bladder, a partial urethral injury has been identified. One gentle attempt at passage of a 12 to 14 Fr Foley or coude catheter may be attempted, depending on the experience and confidence of the catheterist. Once again, any degree of resistance dictates termination of the procedure and urologic consultation.

In the patient without external sphincter spasm in whom the retrograde urethrogram shows evidence of urethral extravasation without any contrast filling the bladder, a

complete urethral injury has been identified, and immediate urologic consultation is indicated for placement of a suprapubic catheter and subsequent surgical repair of the complete urethral injury. One caveat: if >50 to 60 mL of contrast is used at any time for urethrography and more than gentle pressure is exerted during urethral contrast instillation, it is possible to create objective penile venous intravasation of contrast, which is benign but may simulate urethral extravasation and produce a spurious examination result similar to urethral injury. The distinguishing feature with intravasation is that any subsequent film (i.e., post-void film) will disclose immediate clearing of the iatrogenic penile venogram, whereas urethral extravasation will persist indefinitely.

Suprapubic placement of a peel-away sheath and Foley catheter as an alternative to urethral catheterization for the trauma patient is covered elsewhere in this chapter.

**Complications of Urethral Catheterization**

Although urethral catheterization performed by skilled personnel in appropriate circumstances has an acceptable complication rate, untoward sequelae of catheterization are not unusual.

The frequency of bacteriuria after a single catheterization in a healthy outpatient population is probably <1%. [39] However, in hospitalized, elderly, debilitated, or postpartum patients, the rate may be considerably higher. Urinary catheterization is the leading cause of nosocomial urinary tract infections. The mortality in patients with nosocomial urinary tract infection is approximately 3 times that in patients not acquiring infection. [44] Of patients catheterized with a closed system for 2 to 7 days, 8% to 10% will have significant bacteriuria once the catheter is removed. [45] Patients with catheters indwelling >10 days almost always acquire an infection. Infection from the urethra and the bladder may disseminate to cause epididymitis, pyelonephritis, and bacteremia. Although the use of a povidone-iodine lubricating gel has been shown to reduce the inoculation of bacteria into the bladder at the time of catheterization, [46] further study will be needed to determine if this antiseptic lubricant actually reduces infectious sequelae.

With long-term catheterization, bacteriuria is inevitable. Although episodes of high temperature (38.8 °C) due to urinary tract infection in patients with long-term catheterizations are rare (2 per 1000 patient-days), these episodes can be associated with bacteremia and death. [47] Use of a condom catheter, adult diapers, and intermittent self-catheterization [48] represent noninvasive alternatives to long-term Foley catheterization in nonambulatory incontinent men and women. Other rare complications of long-term indwelling urethral catheterization include bladder stones, recurring bladder spasm, periurethral abscesses, bladder perforation, [49] and urethral erosion. [50]

In addition, complications may occur during the act of catheterization. False passages may be established in any area of the urethra when force is exerted on the catheter. In an uncircumcised patient, negligence in reducing the retracted foreskin to its normal anatomic position after urethral catheterization or instrumentation may lead to painful paraphimosis and associated complications.

Leaving a catheter indwelling too long or using a larger catheter than is needed

promotes poor drainage of the periurethral glands, urethritis, and periurethral abscesses, all of which may lead to urethral stricture disease. Likewise, concretions may form around the catheter balloon and lead to the formation of bladder stones, many of which require operative removal.

The use of silicone rather than latex catheters for postoperative urinary drainage in adult males undergoing cardiac surgery has been shown to reduce the incidence of subsequent urethral stricture formation. [51] Although patients with indwelling latex catheters were catheterized <48 hours, a 2% incidence of urethral stricture was still noted at 1 year and a 5% incidence at 2 years. None of the patients with indwelling silicone catheters developed a stricture.

Hematuria has long been considered to be common immediately following even atraumatic catheterization. Although

Sklar and colleagues found a small increase in urinary red blood cell (RBC) count with catheterization, only 1 in 47 patients had an increase of >4 RBCs per high-power field (HPF) attributable to the procedure. [52] They suggest that >4 RBCs per HPF following catheterization is unlikely to be due to the procedure and is, in fact, evidence of preexisting hematuria, which must be explained.

Undesirably retained urethral catheters are an uncommon but frustrating problem. Catheters may be retained because of balloons that do not deflate (see following section) or very rarely because of a knot that has spontaneously developed in the catheter (very rare). Catheter knotting has been associated with the insertion of a highly flexible catheter far into the bladder. [53] A guide wire passed up the catheter may be successful to manipulate some knots free, but urethral dilation with progressively larger catheters adjacent to the retained catheter may be needed to permit urethral passage of the knot.

## REMOVING THE NON-DEFLATING CATHETER

The self-retaining Foley balloon-type catheter obviates the need for cumbersome taping or suturing of the catheter to keep it in place. Occasionally, however, an indwelling catheter balloon does not deflate. Needless to say, this problem has challenged and frustrated many physicians and has produced a number of solutions. The usual cause of the nondeflating catheter balloon is the malfunction of the flap-type valve in the balloon lumen of the catheter, which normally allows fluid to enter the balloon of the catheter but prevents passive egress (Fig. 59-22) (Figure Not Available) . [54] The ideal solution is one that resolves the problem--deflating the balloon--without creating another problem (i.e., unnecessary bladder irritation or balloon fragmentation). Of the methods recommended to decompress non-deflating catheter balloons, the only technique that approaches the ideal directly attacks this flap valve deformity. Other methods of deflation are effective but require more creativity and dexterity on the part of the catheterist.

**Techniques**

One method of balloon deflation consists of simply overstretching the balloon with air or water to the point of rupture. Up to 200 mL of fluid can be injected before a 5-mL balloon will rupture. [54] [55] Adding volume to the empty bladder may not be a problem. Unfortunately, this technique may produce unacceptably painful bladder distention for the patient whose catheter is blocked and whose bladder is either secondarily contracted due to chronic infection or neurogenic bladder dysfunction or distended to the point of maximum filling. An even more compelling reason not to use this technique is the disconcerting frequency of balloon fragmentation and subsequent foreign-body bladder stone formation. In an experimental study of 100 catheters (50 of which were overdistended with water and 50 of which were overdistended with air), all 100 catheter balloons ruptured into fragments. [54] Cystoscopic inspection of the bladder and removal of any fragments will be required to prevent bladder stone formation if this method of balloon deflation is selected.

A second method of balloon deflation involves injecting an erosive substance into the balloon port. This causes the balloon to deflate after part of the balloon wall has been eroded. Organic compounds that attack the latex polymers are often used. Ether, acetone, mineral oil, and even petrolatum ointment have been used. In general, the more volatile the substance, the more rapidly it ruptures the balloon. Rupture of the balloon may be partly a result of the rapid expansion that some of these volatile substances--especially ether--undergo at body temperature. Ether was reported to rupture 58 of 60 catheter balloons within 2 minutes of injection into the balloon port. Unfortunately, in 56 of the catheters, a free fragment of the balloon was created. Mineral oil, which works more slowly, was associated with fragment production in 95 of 100 catheters tested. [54] When released into the bladder, organic substances often produce a symptomatic chemical cystitis. Use of these substances is discouraged.

A third method of deflating the balloon is to puncture it with a needle. With gentle traction, the balloon is located in the urethra or drawn against the bladder neck and is punctured with a thin 25- or 27-ga spinal needle. This needle may be directed suprapubically (transvesically), transvaginally, transperineally, or transrectally. The procedure may be done either blindly [57] or with the aid of ultrasound. In women, a spinal needle may be gently introduced transurethrally alongside the catheter. Fragmentation during puncture can occur, but it is much rarer than in the 2 techniques described previously.

The easiest way to deflate a non-deflating balloon is to attack the inflate-deflate channel that normally prevents the passive egress of inflating fluid. Patients may be sent to the ED in the late evening or early morning hours after their catheters have been progressively shortened by ingenious health care providers during the day. Cutting the catheter may result in rapid deflation if the valve-flap defect happens to be present in the part of the catheter that is cut off, an uncommon occurrence. A shorter catheter with a more proximal valve-flap defect can often be left for 24 hours with ongoing slow balloon deflation, but this maneuver leaves the problem of managing an unconnected catheter and an incontinent patient. Devising a waterproof and aseptic method of

collecting urine from the shortened Foley catheter may require use of a ureteral catheter drainage bag or other ingenious approaches.

When presented with this situation, it is often best to insert a thin, rigid wire into the balloon-port lumen in an effort to deflate the valve-flap defect sufficiently and promote the escape of fluid from the balloon. A stainless steel wire

suture of 3-0 or 4-0 gauge is the thinnest suitable material. The wire stylet from an angiographic catheter, guide wires from ureteral catheters, and very small, well-lubricated ureteral catheters themselves have all been reported to be successful. When a ureteral catheter guide wire was used in one series, 34 of 39 balloons were deflated without fragmentation. In the 5 unsuccessful cases, needle puncture of the balloon was required and was successful. [57]

One approach is to use a stepwise series of maneuvers. If the balloon does not deflate, remove the syringe adapter plug from the balloon-inflating channel. This rules out a malfunction of the adapter. If the balloon water does not escape, next insert an angiographic catheter stylet into the balloon-inflating channel and rotate it. Usually, the water from the balloon flows out along the wire. If it does not, place the catheter on traction and attempt to locate the balloon by palpation either perineally, transvaginally, or transrectally. If this is successful, a 25- to 27-ga spinal needle under local anesthesia is used to blindly puncture the balloon and then remove the catheter. If localization is unsuccessful, multiple blind passes with the 27-ga needle can be attempted; this is usually successful in decompressing the balloon and removing the catheter. [58] If the patient requires a permanent indwelling catheter one may be replaced immediately. Concomitant inadvertent needle punctures of the rectum are usually of no clinical significance.

Once a malfunctioning balloon has been deflated, it is mandatory to carefully inspect the balloon itself for missing fragments. If a piece of the balloon is missing, it is necessary to arrange for subsequent cystoscopy to look for and remove the fragment. Unfortunately, pretesting Foley catheter balloons by trial inflation and deflation before insertion does not eliminate the potential for a non-deflating Foley catheter balloon.

## SUPRAPUBIC ASPIRATION OF THE BLADDER

One of the problems of interpreting voided urine samples is that the urine from the bladder passes through a progressively more contaminated urethral conduit. In the female, the perineum is a culture medium where bacteria are seemingly eager to be swept along into the sterile collection cup and onto the agar plate. To avoid the dilemma of interpretation, physicians have devised maneuvers to minimize the presence of contaminating organisms. Male patients are instructed to retract the foreskin, cleanse the meatus, discard the first portion of urine, and catch the midstream part of the voided specimen. Female patients are asked to perform even more difficult maneuvers to avoid bacterial contamination: sit backward on the commode facing the wall, hold the labia apart with one hand, cleanse the periurethral skin blindly with the other, then reach for the cup, initiate voiding, and catch the midstream urine--all while holding the labia apart and maintaining the precarious position on the commode. Some experts [59] have

women void in the lithotomy position after an assistant retracts the labia, cleanses the perineum, and then catches the midstream urine.

In standard transurethral bladder catheterization, even under ideal circumstances, the procedure is often uncomfortable. The catheter must traverse the distal contaminated urethra and may infrequently introduce contaminating bacteria into the specimen and into the bladder of the patient, resulting in infection, primarily in patients who don't empty their bladder with normal voiding.

Suprapubic aspiration of the bladder, first reported as a method of collecting urine for bacteriologic study in 1956, [60] offers the physician a relatively simple means of obtaining uncontaminated bladder urine. Urethral contamination is successfully avoided, and positive results always represent true bacteriuria. The one caveat is that the bladder must be full to avoid multiple painful needle sticks, a clinical situation that may be difficult to discern in a sick child.

**Indications**

In the neonate or the young child, suprapubic aspiration or urethral catheterization can provide the physician with a sample that is reliable for bacteriologic interpretation. Although disconcerting to some parents (they may wish to leave the room or look away during the procedure), suprapubic aspiration is not a dangerous procedure, and the sensitivity of urinalysis of this urine for bacteriuria approaches 100%. However, for children 2 years or older, urine can generally be more easily collected by urethral catheterization.

For adult patients, the indications for suprapubic aspiration are more limited, because these patients usually can cooperate with the physician. Men with condom catheters or phimosis, however, may require suprapubic aspiration to minimize urethral contamination. Aspirated cultures, rather than catheterized specimens, may help rule out contamination in patients with asymptomatic bacteriuria on routine urine collection. In infections caused by organisms that in other circumstances are often discounted as contaminants (e.g., *Staphylococcus epidermidis* or *Candida albicans*), suprapubic aspiration or a catheterized specimen is required to confirm the presence of such pathogens.

In patients in whom the possibility of infravesical infection is a concern (e.g., patients with chronic infections of the urethra or the periurethral glands), suprapubic aspiration may help localize a bladder from a urethral source.

**Procedure**

The physician must first locate the bladder. A full, palpable, or percussible bladder should be readily apparent, but this can be difficult to discern in all but the thinnest patients. If there is any question about the location or the amount of bladder urine, a quick ultrasound examination is informative. The point of entry in the skin should be 1 to 2 cm above the superior edge of the symphysis pubis. The syringe and needle are

passed perpendicular to the abdominal wall toward the bladder, usually a 10° to 20° angle from the true vertical, somewhat *cephalad* in *children* (Fig. 59-23) and somewhat *caudad* in *adults* (Fig. 59-24) . Note that the bladder of a newborn is an abdominal organ and that it will be missed if the needle is inserted too close to the pubis or is angled toward the feet.

The child is placed supine and is restrained with the legs in a frog-legged position. Once the prepared skin has been draped and the point of entry has been chosen, a skin wheal of local anesthesia is raised to reduce discomfort. When the skin has been anesthetized, a longer, larger-caliber needle (usually 22 ga, 3.75 to 8.75 cm in length) is advanced in the midline through the skin and quickly into the bladder.

This author prefers to advance the needle attached to a syringe, with active aspiration during advancement. As soon as the bladder is entered, urine appears in the syringe. A short needle is adequate for virtually all pediatric patients. After the urine is collected, the syringe and needle are withdrawn. Microscopic hematuria always follows the procedure but gross hematuria is uncommon. A bandage may be placed over the puncture site. If urine is not obtained, the needle is not removed but withdrawn to a subcutaneous position and redirected at a different angle. Often a child may spontaneously start to void following any type of invasive stimulus (e.g., bladder irritation by a probing needle, venipuncture, or lumbar puncture). Hence, preparation to collect a spontaneously voided specimen is recommended, should that option arise. This should be anticipated before beginning blood or spinal fluid collection during the bacteremic workup of the febrile neonate.

In most patients, an acceptable urine sample can be obtained with the first needle pass. If the needle points too caudad in an effort to avoid entering the peritoneum, it is possible to enter the retropubic space, skimming the bladder muscle and never penetrating the bladder mucosa.

### Complications

Stamey has performed several thousand aspirations without complications. [59] Bacteremia does not result from this procedure. [63] Bowel penetration has occurred in children who had distended abdomens from gastrointestinal disturbances. [64] The combination of gaseous bowel distention and relative hypovolemia may displace and flatten the relatively empty bladder against the pelvic floor. Even when the large bowel has been penetrated, patients recover uneventfully. Simple penetration of the bowel with a needle is considered an innocuous event and requires no specific treatment.

## PERCUTANEOUS SUPRAPUBIC CYSTOSTOMY

### Background

Although suprapubic cystostomy was described as early as 4 centuries ago, the safety of the procedure was first demonstrated by Garson and Peterson in 1888. The first modern method was the Campbell trocar set, described in 1951. [65] [66] Campbell used a

sharp trocar passing through a sheath. The sheath had one longitudinal portion of its wall missing to permit a balloon-type Foley catheter to be passed into the bladder. The Campbell trocar is a large-diameter instrument, accepting up to a 20 Fr catheter. Newer technologies have made its use obsolete in the ED.

The development of punch thoracostomy tube sets suggested their use as modified cystostomy tubes. This led to the invention of medium-caliber cystostomy tubes, which were easier to insert than the Campbell trocar but provided more satisfactory drainage than adaptations of IV infusion sets. Ingram's trocar catheter is perhaps the best known of these tubes. It has 3 lumina: 1 for inflating the retention balloon and the other 2 for drainage or irrigation. The Ingram catheter is available in a 12 or 16 Fr size. The Stamey suprapubic catheter is another variation of this type, but it uses a 4-wing Malecot-type retention device rather than a preferred user-friendly inflatable balloon.

Perhaps the most widely known and frequently used trocar-type cystostomy tube is the Cystocath. [70] It is available in 8 and 12 Fr sizes. The latter is more commonly used for adult patients. The Cystocath is packaged as a self-contained set supplying virtually everything needed for insertion. The device is easy to insert and may be satisfactory for relatively long periods of trouble-free use if the patient is given conscientious nursing care.

The major difficulty with cystostomy tubes of all designs has been securing them to the patient's skin. Those with retention balloons, such as the regular Foley urethral catheter or the Ingram catheter, are most secure and only need tape to secure them to the anterior abdominal wall. Virtually all other systems depend on tape or skin adhesive to hold either the tube or the appliance in place. They become an annoyance to both the patient and the care provider.

The latest and most user-friendly device for suprapubic bladder access is the Cook peel-away sheath unit. [36] It uses the Seldinger (guide wire) technique to gain bladder access and allows suprapubic placement of a Foley balloon catheter for definitive bladder drainage. This device is recommended for ED use over other suprapubic bladder access approaches and is discussed in this section.

### Indications

In general, any patient who would require a urethral catheter but in whom a catheter cannot be passed is a candidate for a suprapubic cystostomy tube. In emergency situations, the majority of these patients are men with urethral stricture or complex prostatic disease and trauma patients with urethral disruption. Depending on the experience of the catheterist, dilation can usually be performed in patients with urethral strictures using filiforms and followers. If there is any difficulty with urethral instrumentation, a suprapubic cystostomy tube is prudent and prevents further urethral injury. Complete urethral transection associated with a pelvic fracture is an absolute indication for emergent suprapubic cystostomy. Many affected patients need laparotomy because of associated injuries, and a large suprapubic catheter can be placed intraoperatively. However, if the patient does not require laparotomy, a percutaneously placed Foley catheter allows urologic surgery to be done electively after the patient's

condition has stabilized clinically.

Patients with lower genitourinary infection deserve special care before instituting any type of urethral instrumentation. The risk of inciting an episode of gram-negative bacteremia with urethral dilation must be considered, and appropriate IV gram-negative antibiotic coverage started before the patient is instrumented. Foley catheter drainage is the first choice and suprapubic drainage is an option in patients with acute prostatitis or epididymitis who require bladder drainage. Ideally, a suprapubic catheter allows both bladder drainage and unobstructed drainage of prostatic, seminal vesicle, and urethral secretions but requires an invasive procedure with its associated risks.

Neurologically disabled patients (e.g., quadriplegics or paraplegics) or patients with any type of neurogenic bladder dysfunction who have been successfully maintained on a program of intermittent self-catheterization occasionally have difficulty with urethral catheterization. In these patients,

especially those with high spinal cord lesions, suprapubic needle aspiration or suprapubic cystostomy can be a rapidly effective method of relieving autonomic hyperreflexia associated with acute bladder distention. [71] Bladder decompression in the dysreflexic, profusely perspiring, hypertensive quadriplegic in sympathetic crisis provides dramatic symptom resolution, whether by suprapubic bladder decompression or Foley catheter placement.

Suprapubic catheterization is *not* recommended as first-line treatment for the patient who is voiding poorly from lower urinary tract prostatic obstruction. Such patients, although symptomatic, are better off with intermittent self-catheterization or an indwelling Foley catheter if they are in retention or have chronically infected urine. Young women with psychosocial or emotional neurogenic bladder dysfunction are best managed by intermittent self-catheterization. In all such cases, clinical judgment will dictate the most appropriate form of treatment and whether concomitant antibiotic therapy is required.

**Contraindications**

Because placement of a suprapubic tube involves some risk, patient selection is important. The procedure should not be performed in a patient whose bladder is not definable. Although no absolute reported minimum bladder volume has ever been established, there must be enough urine in the bladder to allow the needle to fully penetrate the bladder dome without immediately exiting through the base. There must also be enough urine in the bladder to displace the bowel away from the anterosuperior surface of the bladder and the entrance of the needle. Ultrasound may be helpful in defining bladder anatomy.

Individuals who have a history of previous lower abdominal surgery, intraperitoneal surgery, or irradiation may have developed adhesions or adherence of the bowel to the anterior bladder wall. They are potentially at greater risk for bowel injury during percutaneous suprapubic cystostomy tube placement than those without previous abdominal surgery. Blind suprapubic cystostomy tube placement in these patients

should be avoided. The absence of any of these risk factors does not totally exclude the risks of bowel or intraperitoneal injury, but it reduces them significantly.

Patients with bleeding diatheses are at greater risk for postinsertion bleeding, either into the bladder or into the retropubic space, than their normal counterparts.

## Equipment

The items of equipment needed for Cook's peel-away sheath placement are listed in Table 59-6 .

## Procedure

The following comments describe the placement of the Cook peel-away sheath. With modifications, these guidelines are adaptable for any type of suprapubic catheter placement.

### Preparing the Patient

If necessary, the lower abdomen is shaved. Povidone-iodine skin preparation or another suitable bactericide is used to cleanse the area. The extra liquid is removed, and the skin is allowed to dry. A 6-mL syringe is filled with 1% lidocaine, and a 22-ga, 7.75 cm spinal needle is attached. A skin wheal is raised in the proposed site (approximately 2 to 3 cm above the pubic symphysis), and the subcutaneous tissue and rectus abdominis muscle fascia is infiltrated at a 10° to 20° angle toward the pelvis.

The bladder is located by advancing the needle in the prescribed direction while aspirating the syringe. Urine is easily aspirated when the bladder is entered (Fig. 59-25 A).

### Placing the Tube

Once the bladder has been located, the syringe is removed from the needle and a guide wire is advanced through the needle into the bladder (Fig. 59-25 B). The needle is withdrawn, leaving only the guide wire traversing the anterior abdominal wall and positioned inside the bladder. A No. 15 scalpel blade is used to make a stab incision directly posterior to the wire through the skin, subcutaneous tissue, and superficial anterior abdominal wall fascia. The peel-away sheath and indwelling fascial dilator are then passed together over the wire into the bladder (Fig. 59-25 C). The guide wire and fascial dilator are removed, leaving only the peel-away sheath inside the bladder (Fig. 59-25 D). A preselected Foley balloon catheter is then passed through the indwelling intravesical sheath into the bladder (Fig. 59-25 E). Urine is aspirated to confirm proper placement. The Foley balloon is inflated with a minimum of 10 mL of air, water, or saline (Fig. 59-25 F). The peel-away sheath is withdrawn from the bladder and anterior abdominal wall and is literally peeled away from the catheter, leaving only the indwelling suprapubic Foley catheter (Fig. 59-25 G). The catheter is withdrawn slowly until the inflated balloon approximates the cystostomy site (Fig. 59-25 H). The catheter is

connected to a drainage bag, and the wound is dressed with 4 × 4 gauze pads to complete the procedure.

## Complications

A wide variety of complications specific to each procedure have been reported, which serve as reminders that suprapubic cystostomy is not innocuous. Occasionally, despite the best intentions, the suprapubic tube or catheter cannot be positioned or maintained successfully without untoward sequelae (Table 59-8) .

The most serious complications involve perforation of the peritoneum or the intraperitoneal contents. Any condition that might fix the anterior peritoneum so that the filled bladder cannot lift the peritoneum cephalad may result in either transperitoneal bladder puncture or possible perforation of small or large bowel. Although finding the bladder

| **TABLE 59-8** -- Reported Complications of Suprapubic Cystostomy |
|---|
| Bowel perforation |
| Intraperitoneal extravasation (without a prior history of surgery) |
| Extraperitoneal extravasation |
| Infection of space of Retzius |
| Ureteral catheterization |
| Obstruction of tubing by blood, mucus, or kinking |
| Tubing comes out |

using a small-gauge scout needle may help reduce bowel injury, even in the most apparently successful of bladder punctures, a complication may result.

The cystostomy tube or catheter that merely traverses the peritoneum may produce a mild ileus, serve as a route for peritoneal infection, or drain the bladder contents into the peritoneal cavity. The last situation would be expected if a Cystocath, rather than a peel-away sheath, were used, and one of the extra holes of the Cystocath tubing opened into the peritoneal cavity. Through-and-through bladder penetration with associated rectal, vaginal, or uterine injury has been reported, although the consistent use of small-gauge bladder locator needles and the judicious advancement of fascial dilators should reduce the incidence.

Occasionally the physician is tempted to proceed with suprapubic cystostomy when the bladder is not palpable and has not been located with a syringe and needle. Injury of adjacent organs is much more frequent in these circumstances. If physicians remind themselves that the bladder eventually refills, they will find waiting much more tolerable. If faced with an emergency, ultrasound guidance may be helpful for determining bladder size and location.

Infection may occur at the suprapubic cystostomy skin site or anywhere along the course of the catheter. [75] Use of antimicrobial ointment daily after cleaning the catheter entry site may reduce purulence around the tube. However, topical care does not prevent eventual deep space or bladder infection from the presence of a foreign body. Deeper tissue infections may result from extravasated infected urine or from a superficial infection spreading along the tube to a hematoma at the bladder or fascial level. Parenteral antibiotics may be required. Open drainage is rarely needed unless a loculated abscess has formed.

Hematuria is rarely more than a transient problem. [76] After suprapubic Foley catheter insertion, bladder irrigation may occasionally be required to clear the hematuria. Transient Toomey syringe aspiration may be needed to evacuate clots.

## EMERGENCY LOWER GENITOURINARY RADIOLOGIC PROCEDURES

Trauma to the urinary tract accounts for about 10% of all injuries seen in EDs. Although the signs of genitourinary trauma in general can be quite subtle, lower urinary tract injury can often be quickly identified and thoroughly evaluated radiographically in the ED. Radiologic imaging of the upper urinary tract is generally a less urgent matter and can usually be done in the radiology suite or, when important for emergency operative decision-making, as a single shot intravenous pyelogram (IVP) in the operating room. Hence, this section does not discuss the role or technique of IVP in detail. Note that the timing of any radiologic evaluation can be challenging to the emergency physician, especially when faced with a critically ill multiple trauma patient. The priority and extent

of such an evaluation, of course, must be determined by the trauma team of physicians involved in each resuscitation.

**Indications for Evaluation**

The urinary tract includes the kidneys, ureters, bladder, urethra, and external genitalia. Approximately 8% to 10% of *blunt* abdominal trauma is associated with injuries to the urinary tract. [77] In one large series, [78] 7% of gunshot wounds and 6% of stab wounds to the abdomen resulted in *penetrating* wounds to the kidney. For injury identification purposes, the genitourinary system is best divided into lower urinary tract (i.e., urethra and bladder), upper urinary tract (i.e., kidneys and ureter), and external genitalia (i.e., penis, scrotum, and testes or vagina, labia majora, and labia minora). Each of these subdivisions has its own markers for potential injury. These markers are addressed during the resuscitation phase of trauma care and during secondary injury survey when the abdomen, pelvis, external genitalia, vaginal vault, and rectum are systematically examined.

The markers for lower urinary tract injury are blood at the urethral meatus, abnormal position of the prostate on rectal examination (in men), and gross hematuria. [79] Perineal ecchymosis and scrotal hematoma also represent potential lower urinary tract injury, but these findings are usually seen later in the patient's course rather than acutely in the ED. Gross hematuria or microscopic hematuria (3 to 5 RBCs per HPF--spun specimen) *in conjunction with any history of shock* (systolic blood pressure 90 mm Hg) *in the field or in the ED following blunt trauma* are markers of potential upper urinary tract injury in any adult. [80] In children, there are no markers for upper tract injury; consequently, any degree of microscopic or gross hematuria indicates potential genitourinary injury and requires radiographic diagnostic evaluation. [81]

In genitourinary trauma, the lower urinary tract is always evaluated before the upper urinary tract. Retrograde urethrography and retrograde cystography are the diagnostic procedures of choice to evaluate potential injury to the lower urinary tract. These studies must be carried out in the proper sequence and in a retrograde fashion to avoid missing potential injuries. *Retrograde* refers to the technique of instilling contrast retrograde through the urethra or by gravity filling of the bladder. It must be distinguished from antegrade filling, in which IV contrast for IVP or abdominal computed tomography (CT) is excreted from the kidneys and allowed to fill the bladder passively over time.

Contrast-enhanced CT is the diagnostic examination of choice for suspected renal trauma. It provides greater resolution and sensitivity than bolus infusion IVP with nephrotomography and has the advantage of evaluating other intra-abdominal structures as well. [82] However, it is expensive, and in some hospitals it is not readily available on a 24-hour basis. A reasonable course of action under these circumstances would be to initiate the upper urinary tract investigation with bolus-infusion IVP with nephrotomography and to investigate further with contrast-enhanced CT if an ill-defined or poorly visualizing kidney is the result of the initial study. Contrast-enhanced CT should be performed initially if thoracic or intra-abdominal injuries are present or suspected, or if there is concern about renal pedicle injury.

**Gross Hematuria**

Gross hematuria is indicated by any color of urine other than clear or yellow. It is an *absolute* marker for urinary tract injury and an indication for diagnostic evaluation. The resuscitating physician must be responsible for observing the initial bladder effluent following Foley catheter insertion. Vigorous fluid resuscitation may quickly clear initial gross hematuria and eliminate the only marker for potential injury. When gross hematuria is encountered as an injury marker, the bladder and kidneys are thought of as potential sources for the hematuria. In most cases, gross hematuria in association with a pelvic fracture will implicate the bladder as the most likely source of injury. In the absence of a pelvic fracture and with a history of upper abdominal or chest trauma, the kidneys are the most likely source of the hematuria. In urologic trauma, the lower urinary tract must always be studied before the upper urinary tract (i.e., study the urethra before the bladder, study the bladder before the kidneys). The specific diagnostic studies must always be done in a retrograde fashion. This allows the responsible physician to directly control the amount of contrast used to investigate potential urethral or bladder injuries. Whenever any doubt exists about the mechanism of injury, the patient's physical examination, or the source of gross hematuria, the resuscitating physician is always advised to begin with an evaluation of the lower urinary tract before evaluating the upper urinary tract.

**Evidence of Lower Urinary Tract Injury**

In the resuscitation of any trauma patient, placement of a Foley catheter has become the standard method of monitoring urinary output. Blood at the urethral meatus, however, indicates a potential partial or complete urethral disruption and dictates the need for a retrograde urethrogram to delineate urethral integrity. This study can be done by the resuscitating physician in the ED or on the operating room table by the trauma surgeon or urologist if the patient requires immediate surgical intervention for life-threatening injuries.

The male posterior urethra, which includes the membranous and prostatic urethra, is injured more frequently than the anterior urethra. The urogenital diaphragm encloses and fixes the membranous urethra; the prostate and prostatic urethra are firmly attached to the posterior surface of the symphysis pubis by the puboprostatic ligaments. Blunt trauma and pelvic fractures, especially in the presence of a full bladder, may result in shearing forces that partially or completely avulse portions of the firmly attached posterior urethra. Usually the bladder and prostate gland are sheared from the membranous urethra, resulting in a complete urethral disruption (Fig. 59-26) . The female urethra, in contrast, is short and relatively mobile and generally escapes injury in blunt trauma. Occasionally, a significant pelvic fracture will result in a laceration or avulsion of the female urethra at the bladder neck. Direct injuries to the female urethra may also occur secondary to penetrating trauma to the vagina or perineum. These injuries often are disclosed by blood at the introitus or an abnormal vaginal examination in the female pelvic fracture patient. [83]

Contusions or lacerations of the male anterior urethra occur when the bulbous urethra is

compressed against the inferior surface of the symphysis pubis. This happens most commonly as a result of straddle injuries in males but may result from any blunt perineal trauma. Significant trauma to the penile urethra is rare without penetrating injuries or urethral instrumentation. *Anterior urethral injuries* may result in extravasation of blood or urine into the penis, scrotum, or perineum, or along the anterior abdominal wall, depending on whether or not Buck's fascia has been violated (Fig. 59-27) . [35] This is in contrast to *posterior urethral injuries*, in which blood and urine extravasate into the pelvis.

The rectal examination is highly specific in the evaluation of a posterior urethral disruption. If the prostate is not clearly defined (it should have the consistency of the examiner's thenar eminence), is high-riding rather than in its normal anatomic location, or if a pelvic hematoma can be palpated (see Fig. 59-26) , one should be suspicious of a posterior urethral injury, and a retrograde urethrogram should be performed before attempting urethral catheterization. However, a normal rectal examination, by itself, *should not be considered definitive evidence of an intact urethra* if other clinical signs raise suspicion for urethral injury. Retrograde urethrography is a quick, technically easy study to perform and should be part of every emergency physician's armamentarium.

**Pelvic Fracture**

Pelvic fractures occur commonly in patients with urethral or bladder injury. The incidence of lower tract injuries in males with pelvic fractures ranges from 7% to 25%. Conversely, approximately 80% of all posterior urethral and bladder injuries are associated with pelvic fractures. [79] Because of the severity of late complications, especially urethral strictures, which most often require difficult surgical repair, it is paramount that these injuries not be missed. Again, in any female patient with a pelvic fracture, it is most important to examine the introitus and vaginal vault for blood, which may be indicative of urethral, bladder neck, or vaginal wall lacerations. In male patients, rectal examination of the prostate to assess its position will be most helpful in assessing the posterior urethra. A pelvic fracture in association with gross hematuria is an absolute indication for retrograde cystography. In a review of 234 patients with traumatic pelvic fractures, no major lower urinary tract injuries were found in the absence of gross hematuria. [79]

## Radiographic Contrast Material

Radiographic contrast material is used to fill vessels and other structures to render them diagnostically radiopaque. To evaluate the urethra and bladder, contrast is injected or instilled into these structures in a retrograde manner. To evaluate the kidneys and ureters, a bolus of contrast material is injected into the venous system, opacifying the renal parenchyma and collecting system as it is excreted unchanged in the urine. Three types of contrast material are currently available (Table 59-9) . All contain iodine, and all are hyperosmolar with respect to blood. Conventional agents, such as Hypaque and Renografin (diatrizoate), are triiodinated water-soluble agents (ionic monoacetic monomers) that completely dissociate into anion and cation moieties on intravascular injection. Osmolality is quite high, ranging from 1200 to 2000 mOsm/kg. Many of the side effects of contrast agents have been attributed to their osmolarity. Although iodine

concentrations do determine the quality of the radiographic image, iodine itself is not thought to play a major role in the typical anaphylactoid side effects. [84]

Two new classes of contrast agents are ioxaglate (Hexabrix), an ionic monoacetic dimer, and nonionic (non-dissociating) agents, such as iopamidol (Isovue) and iohexol (Omnipaque). The newer agents have twice as many iodine atoms per particle in solution as conventional agents and therefore provide significantly higher urinary iodine concentration, offering better diagnostic imaging. The osmolality of the newer agents is markedly lower, ranging from 600 to 700 mOsm/kg. The lower osmolality and improved chemical structure may be associated with fewer adverse side effects. [85] [86] Although these new agents are promising for intravascular use, there is still some skepticism that they will truly limit major or clinically significant contrast reactions. [87] The lower-osmolarity nonionic agents have not been associated with a lower incidence of contrast-induced nephropathy. Furthermore, there is no indication for using these more expensive products in the retrograde evaluation of the injured lower urinary tract.

## Radiographic Techniques

### Kidneys, Ureters, and Bladder

The plain film, scout film, or KUB (for kidneys, ureters, and bladder) film of the abdomen, as this view is variously referenced, includes the kidneys, ureters, bladder, and full pelvis. It is essential as the initial diagnostic film because it serves as the film of reference for all subsequent films after injection or instillation of contrast material. Incidental nondiagnostic findings on initial KUB that may alert the physician to the possibility of urinary tract injury include the following:

1. Loss of one or both psoas shadows secondary to blood in the retroperitoneum.
2. Spinal curvature secondary to splinting--usually concave to the side of the injury.
3. Lower rib or transverse process fractures, both of which may be associated with upper urinary tract injury.
4. Pelvic fracture.

---

**TABLE 59-9** -- Clinical Use of Radiographic Contrast Material (RCM) for Intravenous Pyelogram (IVP) and Retrograde Studies

**Use of RCM for IVP**

| | Iodine Content (mg/mL of Solution) | Osmolality (mOsm/kg) ($H_2O$) | Average Volume for IVP |
|---|---|---|---|
| **Conventional ionic RMC** | | | |
| Renografin-60 (diatrizoate sodium) | 288 | 1511 | Adult: 100 mL over 30-60 sec * <br> Child: 1.5 mL/kg |
| Hypaque (50%) (diatrizoate sodium) | 300 | 1500 | Adult: 100 mL over 30-60 sec * <br> Child: 1.5 mL/kg |
| Conray (methyl glucamine iothalamate) | 282 | 1217 | Adult: 100 mL over 30-60 sec * <br> Child: 1.5 mL/kg |
| **New nonionic RMC** | | | |
| Isovue (iopamidol) | 300 | 616 | Adult: 50 mL over 30-60 sec <br> Child: 1-1.5 mL/kg |
| Omnipaque 300 (iohexol) | 300 | 672 | Adult: 50 mL over 30-60 sec <br> Child: 1-1.5 mL/kg |
| **Use of RCM for Retrograde Studies** | | | |

| RCM | Use | Procedure |
|---|---|---|
| Renografin-60 or Hypaque (50%) | Dilute stock solutions with saline 1:10 (10% solution) | Urethrogram: 10-15 mL of dilute solution injected slowly through urethral meatus. Children: 0.2 mL/kg<br>Cystogram: after plain film and with Foley catheter in place, fill bladder of adult with 400 mL of dilute contrast material, introduced under gravity. Children: 5 mL/kg |

* Average dose of iodine for IVP with ionic RCM: 350-400 mg/kg or 1.5 mL/kg
A dult: Low dose:
10 g
Intermediate dose:
30 g
High dose:
60 g
Do not exceed 3 mL/kg total dose.
Because the ratio of iodine atoms to dissolved particles is 1.5 with conventional ionic agents and 3.0 with the nonionic agents, less volume is required with the new agents. Average dose is 200-350 mg/kg.

The KUB must always precede the injection or instillation of contrast material, because radiopaque shadows seen on the plain film must be differentiated from extravasation on the postevacuation film.

**Retrograde Urethrogram**

Retrograde urethrography is indicated whenever there is uncertainty about the integrity of the urethra. In cases associated with pelvic fracture, the patient should remain supine throughout the entire radiographic examination. This is important to ensure stability of any possible retropubic hematoma that may result from extensive venous bleeding associated with the initial pelvic fracture. In cases of suspected urethral injury not associated with pelvic fracture, it is acceptable to obtain oblique films during the study that may complement the examination findings. Perpendicular stretching of the penis across the thigh or oblique films may be needed to ensure urethral unfolding and a high-quality urethrogram.

Although several techniques have been promoted for retrograde urethrography, one is emphasized in this section. The choice of technique is not as important as attention to detail. Solutions of either full-strength Hypaque (50%), Cystografin or Renografin-60, or the same agents diluted to a <10% solution using sterile saline as the diluent, are frequently used (see Table 59-9) . First, a plain film (KUB) of reference must be taken before injection of any contrast material. [80] The penile foreskin must be retracted and secured with a folded 4 × 4 gauze sponge. Second, the penis should be held between

the long and ring fingers of the nondominant hand to allow the thumb and index finger of the nondominant hand (see Fig. 59-17) to ensure a snug fit of the contrast-filled syringe inside the urethra (Fig. 59-28 *A*).

After sterile penile preparation, a catheter-tipped Toomey irrigating syringe or a regular 60 mL syringe with an attached Christmas-tree adapter (Fig. 59-28 *B*) is gently advanced inside the urethral meatus until a snug fit is ensured (Fig. 59-28 *C*). Third, approximately 50 to 60 mL of full- or half-strength contrast material is then injected *slowly* under constant pressure into the urethra. Prior to the injection of contrast, the penis should be stretched perpendicularly across the patient's thigh to prevent urethral folding (i.e., the double image of the proximal penile and bulbous urethra superimposed on one another) (Fig. 59-29) . Overly forceful injection of contrast material may cause intravasation of contrast material into the venous plexus of the urethra (Fig. 59-30) (Figure Not Available) . Finally, during the injection of the last 10 mL of contrast, a film (the urethrogram) is taken.  The alternative to this technique, which is discussed in most standard textbooks, is to insert a Foley catheter just inside the urethral meatus, inflate the balloon to ensure a snug fit in the fossa navicularis, and then inject contrast through the catheter (Fig. 59-31) . If not done carefully, this technique often results in the spillage and deposition of contrast outside the urethra and onto the patient and the examination table, thus yielding a spurious result.

The extravasation of contrast material from a urethral disruption usually appears as a flame-like density outside the urethral contour (Fig. 59-32 *A-C*). If any contrast material is seen within the bladder in conjunction with urethral extravasation, a partial rather than complete urethral disruption is more likely. In a complete urethral disruption, urethral extravasation will be present without evidence of contrast within the bladder. The examiner needs to be certain that the lack of bladder contrast is not secondary to voluntary contraction of the external sphincter. Occasionally, as mentioned previously, intravasated contrast material is seen in the periurethral penile venous plexus (see Fig. 59-30) (Figure Not Available) . It is of no clinical significance and should not be mistaken for urethral extravasation. As expected, penile venous intravasation (venous plexus opacification) is seen to clear spontaneously on any post-void films, as compared with urethral extravasation, which remains indefinitely.

If a Foley catheter has been successfully placed into the bladder and a partial urethral injury is suspected later, such an injury can be easily demonstrated without removing the catheter. The lubricated end of a pediatric feeding tube is placed into the penile urethra alongside the existing Foley catheter (Fig. 59-33) . A seal can be obtained by compressing the glans penis with the nondominant thumb and index finger and gently injecting contrast material via a Luer-Lock syringe with the dominant hand. In this way, extravasation can be demonstrated. It should be noted, however, that successful placement of the Foley catheter obviates the need for further treatment of a partial urethral tear in the emergency setting, because an indwelling catheter alone is appropriate initial management for this type of injury. The finding of an associated urethral injury must be conveyed to a urologist, as it will dictate the duration of definitive Foley catheter drainage.

**Retrograde Cystogram**

A retrograde cystogram is performed any time a bladder injury is suspected. It assumes the urethra is normal prior to passing the Foley catheter. A preliminary KUB is obtained that will serve as the reference film for the entire examination. Next, the bladder is filled under direct operator supervision by *gravity instillation of contrast material*. After the central piston is removed from a 60-mL catheter-tip syringe, the catheter-tipped end of the syringe is attached to the Foley catheter and held above the level of the patient's bladder. The contrast material is poured into the syringe and allowed to fill the bladder by gravity instillation to 1 of 3 end points: (1) 100 mL with evidence of gross extravasation on fluoroscopy or on plain film, (if the examiner elects to check at this point); (2) 400 mL in an adult or any child 11 years or older. In children younger than 11 years, bladder capacity, and therefore appropriate contrast volumes, are estimated based on the formula "(age in years + 2) × 30"; or (3) to the point of initiating a bladder contraction (see below), then adding an additional 50 mL by hand injection under pressure.

Anteroposterior (AP) and complementary oblique projections are obtained so long as there is no evidence of a pelvic fracture. In the presence of a pelvic fracture, all films are obtained with the patient in the supine position for the same reasons that were elucidated for retrograde urethrography. A lateral film may be informative when oblique films are not possible (Fig. 59-34) . An AP postevacuation film must be obtained in all cases following bladder drainage. This will disclose posterior perforation in selected cases, especially those associated with penetrating trauma (Fig. 59-35 *A-E*). Again, a *dilute solution of contrast material* (see Table 59-9) may be used, rather than full-strength contrast. Some authors recommend a dilute solution of contrast material (10%) because extravasation into periurethral or perivesical tissues may cause considerable inflammatory reaction at higher concentrations. The dilute solutions do not appear to compromise the quality of the study, but this must be a consideration. *Retrograde cystography done by any technique other than hand-poured gravity instillation is subject to inadequate bladder filling or connector tubing-catheter disconnection.* Both conditions will result in spurious examination results, which may adversely impact important patient management decisions.

It must be stressed that in the absence of initial gross extravasation, the bladder must be filled to 400 mL in an adult, and to an appropriate capacity in a child, and the catheter clamped with a Kelly clamp. Volumes <400 mL have been associated with false-negative findings, especially in penetrating bladder injuries. [88] At times, the patient may have difficulty cooperating with bladder filling because of a head injury or associated pain; and in the case of severe injury, the patient may have involuntary bladder contractions, causing contrast material to back up into the Toomey syringe. If this occurs, refill the bladder to the point of initiating a bladder contraction, clamp the Foley, remove the initial syringe, and replace it with a 60-mL contrast-filled syringe, unclamp the catheter, hand-inject the additional 50 mL under pressure, and reclamp the catheter. The goal is to overdistend the bladder. Once the filled-bladder films have been obtained and reviewed, the Foley catheter is unclamped and the contrast material is allowed to drain into a bedside drainage bag. The AP postevacuation film is then

obtained to visualize any posterior extravasation that may have been hidden by the distended bladder during the AP filled-bladder film. Once again, care must be taken to ensure that contrast material is not spilled onto the patient or the examination table during the procedure. Spilled contrast can lead to spurious examination results.

Extravasation from an injured bladder may be intraperitoneal, extraperitoneal, or both. Extraperitoneal extravasation is usually seen as flame-like areas of contrast material confined to the pelvis and projecting lateral to the bladder (Fig. 59-36) (Figure Not Available) . If the contrast material extravasates intraperitoneally, it tends to fill the paracolic gutters and outline intraperitoneal structures, particularly the bowel, spleen, or liver (Fig. 59-37) (Figure Not Available) . It is important to distinguish extraperitoneal from intraperitoneal injury, as the treatment options are totally different (i.e., surgical repair for all intraperitoneal injuries and for extraperitoneal injuries that extend into or primarily involve the bladder neck, especially in women). Most other extraperitoneal injuries can be managed confidently by Foley catheter drainage alone.

Retrograde cystography may be done in conjunction with contrast-enhanced abdominal CT scanning. The bladder must be filled just as if a conventional retrograde cystogram were being obtained. The catheter is clamped, and evidence for contrast ascites is sought on the CT scan (Fig. 59-38) . When this is encountered, bladder injury with extravasation must be looked for with selective images of the pelvis.

**Contrast Medium Reactions and Toxic Effects**

In the lower genitourinary tract radiologic procedures that are described in this section, the contrast material is administered within the urinary collecting and drainage system. Hence, the patient is at low risk for systemic absorption and allergic reaction. Even with IV infusion, contrast medium reactions are rare; the incidence of significant reactions (i.e., of sufficient severity to require medical intervention) with IV administration is between 1 in 1000 and 1 in 10,000 uses.

Although IV use of contrast medium is outside the scope of this text, volumes and administration information are outlined in Table 59-9 . The ED use of contrast agents is often necessary and justified despite the small possibility of untoward reactions. At times, the patient's past history may not be known, underlying renal function cannot be rapidly assessed, or alternative imaging techniques (e.g., ultrasonography) are unavailable. In such circumstances, the risks vs the benefits of emergent imaging using IV contrast must be carefully weighed. Often the potential information gain of contrast-enhanced imaging in the unstable patient far outweighs the small associated additional risk.

# Chapter 60 - Emergency Childbirth

**Lynnette Doan-Wiggins**

Since ancient times, midwives, specialists of the obstetric art, have supervised the labor of women and the delivery of babies. Physicians did not become involved in this practice until the end of the 18th century.

With improved prenatal and obstetric care, the perinatal death rate has fallen by nearly 50% in the past 25 years. [1] Similarly, the maternal death rate has decreased from 364 per 100,000 live births in 1940 to 8.0 per 100,000 live births in 1990. [1] [2] There are about 180 perinatal deaths for every maternal death. [1]

From the viewpoint of safer care during labor, the outstanding advance of the past 40 years has been the great increase in the proportion of in-hospital deliveries. As recently as 1940, only 60% of white births took place in hospitals; this figure now exceeds 99%. [1] In-hospital births have not only the advantage of better facilities but also care by individuals who are specially trained in obstetrics and perinatology.

The degree to which the emergency physician interacts in the process of labor and delivery varies among institutions, depending on the availability and readiness of inpatient obstetric services. The role of the emergency physician may be only to determine that the patient is indeed in active labor and to order transport directly to the labor and delivery area. In a hospital with little or no obstetric services, the emergency physician may alternatively be called on to manage a complicated delivery and neonatal resuscitation until transfer to another hospital is possible.

To this end the emergency physician must be able to assess the stage and timing of labor, aid the mother in delivery of the infant, and provide initial stabilization of the neonate.

## LABOR

*Labor* is defined as the coordinated effective sequence of involuntary uterine contractions that result in progressive effacement and dilation of the cervix. This, coupled with the voluntary bearing-down efforts of the mother, terminates in delivery, the actual expulsion of the products of conception.

Labor is normally divided into 3 stages. The first stage begins when uterine contractions reach sufficient force to cause cervical effacement and dilation and ends when the cervix is completely dilated. The average duration of the first stage of labor is 6 to 8 hours in multiparous patients and is 8 to 12 hours in primiparous patients. [3] The second stage of labor begins when dilation of the cervix is complete and ends with delivery of the infant. The duration of this stage is highly variable, with a median of 50 minutes in nulliparas and 20 minutes in multiparas. [1] [4] In general, if the second stage lasts >2 hours, abnormal labor has developed. [1] The third stage of labor begins after delivery of

the infant and ends after delivery of the placenta. Infrequently, a fourth stage of labor is described as that period during which myometrial contractions and vessel thrombosis occur (usually lasting approximately 1 hour), effectively controlling bleeding from the former placental implantation site. [1]

## Identification of Labor

### True vs False Labor

Before the establishment of true or effective labor, women may experience so-called false labor. Quite common in late pregnancy, false labor is characterized by irregular, brief contractions of the uterus, usually with discomfort confined to the lower abdomen and groin. The contractions, commonly referred to as *Braxton Hicks contractions*, are typically irregular in timing and strength; there is no change in the cervix and no descent of the fetus.

True labor, on the other hand, is characterized by a regular sequence of uterine contractions, with progressively increasing intensity and decreasing intervals between contractions. The discomfort produced by the uterine contractions of true labor begins in the fundal region and radiates over the uterus into the lower back. The uterine contractions of true labor are accompanied by effacement and dilation of the cervix, with descent of the presenting part of the fetus.

False labor is most common in late pregnancy and in parous women. Although false labor usually stops spontaneously, it may convert rapidly to the effective contractions of true labor (Table 60-1) (Table Not Available) . The interval between true labor contractions gradually diminishes from 10 minutes at the onset of the first stage of labor to as short as 1 minute or less in the second stage. [4]

### Show

A rather dependable sign of the approach of labor is the "show" or "bloody show." Occasionally preceding the onset of labor by as much as 72 hours, show consists of a small amount of blood-tinged mucus discharged from the vagina. Show represents extrusion of the mucus plug that filled the cervical canal during pregnancy and is evidence of cervical dilation and effacement. [1] [4] *Bloody show must be distinguished from more active third-trimester bleeding*, which is classified as a true emergency and in which vaginal examination is *contraindicated*.

### Rupture of the Membranes

Spontaneous rupture of the membranes usually occurs during the course of active labor, although it may occur before the onset of labor in approximately 10% of cases. [3] Rupture of the membranes is typically manifested by a sudden gush of a variable amount of clear or slightly turbid fluid. Rupture of the membranes can be verified if amniotic fluid is extruding from the cervical os or is found in the vaginal fornix on sterile

speculum examination.

Differentiation of amniotic fluid from vaginal fluid may be made by testing a drop of the fluid with *Nitrazine paper.* Amniotic fluid has a pH of 7.0 to 7.5 and turns the paper blue-green to deep blue. In the presence of vaginal secretions only, with a pH of 4.5 to 5.5, Nitrazine paper remains yellow. [1] [5] [6]

Because of its neutral pH, blood may cause a false-positive Nitrazine reading in women who have intact membranes and an unusually large amount of bloody show. [1] [4] [6] Abe found the Nitrazine test to be positive in 98.9% of women with known rupture of the membranes and negative in 96.2% of women with intact membranes. [5] In clinical practice, however, the test is less reliable, because it is frequently used in cases of questionable rupture in which the amount of amniotic fluid is small and therefore more subject to pH changes from admixed blood and vaginal secretions. [1]

A less frequently used method to test for amniotic fluid is *ferning.* A drop of fluid from the cervical os or vaginal fornix is placed on a clean glass slide. Owing to the high sodium chloride content of amniotic fluid, a fern pattern is seen through the microscope as amniotic fluid dries. [7]

Documentation of rupture of the membranes is important for 3 reasons. First, if the presenting part is not already fixed in the pelvis, the possibility of prolapse of the cord with cord compression and subsequent fetal distress is increased. Second, labor may be imminent. Finally, if labor does not begin within 24 hours after rupture of the membranes, the pregnancy must be considered to be complicated by prolonged premature rupture of the membranes with an increased chance of intrauterine infection. [1] [4] If rupture of the membranes is documented in the emergency department (ED), the patient's obstetrician should be notified and hospital admission of the patient should be considered.

## Evaluation of Labor

When a woman presents in labor, the general condition of the fetus and mother must be quickly ascertained by means of the patient history and physical examination. Inquiry is made as to the onset and frequency of contractions, the presence or absence of bleeding, the possible loss of amniotic fluid, and the prenatal care and condition of the mother and fetus. In the absence of active vaginal bleeding, the position, presentation, and lie of the fetus are determined by abdominal palpation and sterile vaginal examination. Staging of labor is assessed by vaginal examination. Fetal well-being is monitored by auscultation of fetal heart tones, particularly immediately after a uterine contraction.

### Lie, Presentation, and Position

In the latter months of pregnancy, the fetus assumes a characteristic posture within the uterus, usually forming an ovoid mass that corresponds roughly to the shape of the uterine cavity. Typically, the fetus becomes folded or bent on itself in such a way that

the back becomes markedly convex, with the head, thighs, and knees being sharply flexed. Usually the arms are crossed over the thorax and are parallel to the sides of the body. The umbilical cord lies in the space between the arms and the lower extremities. This characteristic posture is due in part to the mode of growth of the fetus and is also a result of accommodation to the uterine cavity.

*Lie* refers to the relation of the long axis of the fetus to that of the mother. Lie is either longitudinal or transverse (Fig. 60-1) (Figure Not Available) . Longitudinal lies occur in >99% of pregnancies at term. [1]

The *presentation*, or presenting part, refers to that portion of the body of the fetus that is nearest to or foremost in the birth canal. The presenting part is felt through the cervix on sterile vaginal examination. In longitudinal lies, the presenting part is either the fetal head or the buttocks or the feet. In *transverse lie*, the shoulder is the presenting part.

Cephalic presentations are classified by the relation of the fetal head to the body of the fetus (Fig. 60-2) (Figure Not Available) . Ordinarily, the head is sharply flexed so that the occipital fontanel is the presenting part. This is referred to as the *vertex* or *occiput presentation*. Less commonly, the neck is fully extended and the face is foremost in the birth canal; this is termed *face presentation*. Occasionally, the fetal head assumes a partially flexed or partially extended position, resulting in *sinciput* and *brow presentations*, respectively. Sinciput and brow presentations, associated with preterm infants, are almost always unstable and convert to either the occiput or face presentation as labor progresses.

Breech presentations are classified as frank, complete, and footling or incomplete (Fig. 60-3) (Figure Not Available) . When the fetus presents with the hips flexed and the legs extended over the anterior surfaces of the body, this is termed *frank breech.* Flexion of the fetal hips and knees results in *complete breech* presentation. When one or both of the feet or knees are lowermost in the canal, an *incomplete* or *footling breech* results.

At or near term, the incidence of the various presentations is approximately 96&2percnt; for vertex, 3.5% for breech, 0.3% for face, and 0.4% for shoulder. [1] [4]

*Position* refers to the relation of the presenting part to the birth canal and may be either left or right. The occiput, chin, and sacrum are the determining parts in vertex, face, and breech presentations, respectively. The presentation and position of the fetus are initially determined by abdominal palpation using Leopold maneuvers.

**Abdominal palpation (Leopold maneuvers).**

Although abdominal ultrasonography has largely replaced abdominal palpation for determination of fetal lie, these maneuvers may be helpful when ultrasound evaluation is unavailable. They can be performed throughout the latter months of pregnancy and during labor in the intervals between contractions. The findings from abdominal palpation provide information about the presentation and position of the fetus and the extent to which the presenting part has descended into the pelvis (Fig. 60-4) (Figure Not

Available) . The mother should be placed on a firm bed or examining table with her abdomen bared. For the first 3 of the 4 maneuvers, the examiner stands at the side of the bed facing the patient. During the first maneuver (see Fig. 60-4 (Figure Not Available) *A*), the upper abdomen is gently palpated with the fingertips of both hands to determine which fetal pole is present in the uterine fundus. The fetal breech gives the sensation of a large, nodular body, whereas the fetal head is hard, round, and freely movable.

During the second maneuver, the examiner places his or her hands on either side of the abdomen, exerting deep, gentle pressure (see Fig. 60-4 (Figure Not Available) *B*). On one side, the hard, resistant back is felt; on the other side, the fetal extremities or small parts are felt. By noting whether the back is directed anteriorly, posteriorly, or transversely, fetal orientation or lie is determined.

The third maneuver is performed by grasping the lower portion of the maternal abdomen just above the symphysis pubis with the thumb and forefinger of one hand (see Fig. 60-4 (Figure Not Available) *C*). If the presenting part is not engaged, the position of the head in relation to the back and extremities is ascertained. If the cephalic prominence is palpated on the same side as the small parts, the head must be flexed and therefore a vertex or occiput presentation exists. If the cephalic prominence is on the same side as the back, the head must be extended. If the presenting part is deeply engaged in the pelvis, the findings from this maneuver indicate that the lower pole of the fetus is fixed in the pelvis. The details of presentation and position are then defined by the fourth maneuver.

To perform the fourth maneuver, the examiner changes position and faces the mother's feet. With the tips of the first 3 fingers of each hand, the examiner exerts deep, gentle pressure in the direction of the axis of the pelvic inlet (see Fig. 60-4 (Figure Not Available) *D*). When the head is the presenting part, one examining hand will be stopped sooner than the other by a rounded body, the cephalic prominence, while the other hand continues more deeply into the pelvis. The cephalic prominence is felt on the same side as the small parts in vertex presentations and on the same side as the back in face presentations. In breech presentations, the information obtained from this maneuver is less precise. [1]

**Vaginal examination.**

Unless there has been bleeding in excess of a bloody show, a manual (not speculum) vaginal examination should be performed to identify fetal presentation and position and to assess the progress of labor.

First, the vulva and perineal area are prepared with an antiseptic solution such as povidone-iodine. The woman is placed on a bedpan with her legs widely separated. Scrubbing is directed from anterior to posterior and away from the vaginal introitus; each sponge should be discarded after it passes over the anal region. A dry sponge placed on the introitus prevents contaminated solution from running into the vagina.

After preparation of the vulvar and perineal regions, the examiner uses the thumb and

forefinger of a sterile-gloved hand to widely separate the labia to expose the vaginal opening; this prevents the examining fingers from coming into contact with the inner surfaces of the labia. The index and second fingers of the other hand are then introduced into the vagina to perform the examination. Cervical effacement, dilation, and fetal station are assessed. Fetal presentation and position are confirmed. [1]

*Effacement* of the cervix is the process of cervical thinning that occurs before and during the first stage of labor (Fig. 60-5) (Figure Not Available) . The degree of cervical effacement is assessed by palpation and is determined by the palpated length of the cervical canal compared with that of the uneffaced, or normal, cervical canal. Effacement is expressed as a percentage from 0%, or totally uneffaced, to 100%, or completely effaced. The completely effaced cervix is usually <0.25 cm thick. [3]

*Cervical dilation* is determined by estimating the average diameter of the cervical os. The examining finger is swept from the cervical margin on one side across the cervical os to the opposite margin. The transverse diameter is expressed in centimeters. Ten centimeters constitutes full cervical dilation. A diameter of <6 cm can be measured directly. For a diameter >6 cm, it is frequently easier to determine the width of the remaining cervical rim and subtract twice that measurement from 10 cm. For example, if a 1-cm rim is felt, dilation is 8 cm.

*Station* refers to the level of the presenting fetal part in the birth canal (Fig. 60-6) (Figure Not Available) . The ischial spines are used as the reference point. Zero station is used to denote that the presenting part is at the level of the ischial spines. When the presenting part lies above the spines, the distances are stated in negative figures (-1 cm, -2 cm, -3 cm, and floating). If the presenting part is below the spines, the distances are stated in positive figures (+1 cm, +2 cm, +3 cm).3 Determination is made by simple palpation. Progressive cervical dilation with no change in fetal station suggests fetopelvic disproportion.

*Position and presentation* of the fetus may be inconclusive before labor, because the presenting parts must be palpated through the lower uterine segment. After dilation and effacement of the cervix, however, further delineation of presentation and position of the fetus may be made by vaginal examination.

After the perineal area has been appropriately prepared, as described previously, 3 maneuvers are used to determine fetal presentation and position. In the first maneuver, 2 fingers of the examiner's gloved hand are introduced into the vagina and advanced to the presenting part, differentiating face, vertex, and breech presentations. In vertex presentations, the examiner's fingers are carried up behind the symphysis pubis and then swept posteriorly over the fetal head toward the maternal sacrum, identifying the course of the sagittal suture. The positions of the 2 fontanels, located at opposite ends of the sagittal sutures, are then defined by palpation. The anterior fontanel is diamond shaped; the posterior fontanel is triangular (Fig. 60-7) (Figure Not Available) .

In face and breech presentations, the various parts are more readily distinguished. In breech presentations, the fetal sacrum is the point of reference; in face presentations,

the easily identifiable fetal chin is used.

**Fetal Well-Being**

**Auscultation.**

Auscultation of fetal heart tones is necessary to determine fetal well-being. The heart rate of the fetus can be identified with a stethoscope, a fetoscope, or preferably a Doppler flow detector placed firmly on the maternal abdominal wall overlying the fetal thorax and repositioned until fetal heart tones are heard. When a Doppler flow detector is used, a conducting gel should be applied to the abdominal wall, interfacing with the Doppler receiver. The region of the abdomen in which fetal heart sounds are heard most clearly varies with fetal presentation and the degree to which the presenting part has descended. In cephalic presentations, fetal heart sounds are heard best midway between the maternal umbilicus and the anterosuperior spine of the maternal ilium. [1] To avoid confusion of maternal and fetal heart sounds, the maternal pulse should be palpated as the fetal heart rate is auscultated.

Normal baseline fetal heart rate is 120 to 160 beats/min. Changes in the fetal heart rate that are indicative of fetal distress are usually evident immediately after a uterine contraction. During labor, fetal distress is suspected if the fetal heart rate repeatedly drops below 120 beats/min immediately after a contraction. If prolonged monitoring of labor is necessary in the ED, fetal heart sounds should be assessed during and for 30 seconds after a contraction at 15-minute intervals during the first stage of labor and at 5-minute intervals during the second stage of labor. [1] [9] Additional information on the use of a Doppler monitor to measure fetal heart rate is provided in Chapter 70 . If trained personnel and equipment are available, external tocography provides a noninvasive method for continuous assessment of fetal heart rate and maternal uterine contractions.

**Management of fetal distress.**

If fetal distress is suspected on the basis of resting fetal heart rate or changes after contractions, changing of maternal position, typically into the left lateral recumbent position, may be beneficial. Maternal O2 should be administered to improve fetal oxygenation.

In the absence of bleeding, a vaginal examination should be performed to rule out the possibility of prolapse of the umbilical cord. [7] If immediate obstetric services are not available, consideration should be given to tocolytic therapy to improve fetoplacental blood flow until delivery can be accomplished. By stopping uterine contractions a more sustained placental blood flow is maintained and, in the case of cord prolapse, intermittent pressure on a compromised umbilical cord may be averted. [10] The definitive therapy for fetal distress is delivery of the infant, either vaginally or by cesarean section. [3]

*Cord prolapse* usually occurs at the same time as rupture of the membranes and is

diagnosed by palpation of the umbilical cord on vaginal examination or by visualization of the cord protruding through the introitus. The incidence of cord prolapse in labor is approximately 0.5% and most often occurs when the fetal presenting part does not completely fill the lower uterine segment during labor or when there is unusual mobility of the cord. [11] [12] Cord prolapse is frequently encountered with breech presentation, multiple pregnancies, prematurity, and premature rupture of the membranes. [12] [13]

The management of cord prolapse is directed at sustaining fetal life until delivery is accomplished. Unless immediate delivery is feasible or the fetus is known to be dead, preparations should be made for an emergency cesarean section. If immediate obstetric services are not available, tocolytic therapy may be instituted to decrease uterine contractions and improve fetoplacental perfusion. [11] [12] Compression of the umbilical cord should be minimized by exerting manual pressure through the vagina to lift and maintain the presenting part away from the prolapsed cord. The patient should be placed in the knee-chest or deep Trendelenburg position, and this position should be maintained until delivery is accomplished. Some physicians recommend that after manual elevation of the presenting part, 500 to 700 mL of saline be instilled into the bladder to maintain cord decompression. Once the bladder is filled, the vaginal hand may be removed. [11] [12]

With cord prolapse, the fetal prognosis is dependent on presentation, gestation, and the timing of diagnosis and management. Partial cord compression for <5 minutes may not be harmful. Complete occlusion for the same period or partial occlusion for a longer time is likely to cause death or severe central nervous system damage to the fetus. Perinatal mortality rate ranges from 5.4 to 49.0%. [11] [12]

## Inhibition of Labor

Tocolytic therapy may be indicated in the ED to prevent the progression of labor when fetal distress is noted, particularly that due to cord compression, and to treat preterm labor.

### Preterm Labor

Premature delivery complicates 8 to 10% of all births in the United States, and prematurity continues to be the single greatest cause of neonatal morbidity and mortality. [7] A wide variety of treatments for the inhibition of labor have been advocated, including bedrest, beta-agonist agents such as terbutaline and ritodrine, magnesium sulfate ($MgSO_4$ ), prostaglandin inhibitors, and calcium-channel blockers. Efficacy of each of these regimens is difficult to establish, because of a paucity of well-controlled clinical studies and inconsistently defined indications for the use of tocolytic therapy. [14] [15] Currently, the cornerstones of pharmacologic management of preterm labor are the use of beta-adrenergic receptor agonists and $MgSO_4$ . [7] [15]

The criteria for defining preterm labor vary among investigators. A presumptive diagnosis of premature labor may be made in the woman who is between 20 and 37 weeks' gestation in the presence of regular uterine contractions occurring at intervals of 5 to 8 minutes or less that are accompanied by one or more of the following: (1)

progressive cervical effacement and dilation, (2) cervical dilation of 2 cm, or (3) cervical effacement of 75 to 80% or more. [1] [15] Because rupture of the membranes frequently means that delivery is imminent, uterine contractions accompanied by membrane rupture may also be used to establish the diagnosis. [1] [15] External monitoring devices, when available, are helpful by providing objective evidence of the character of uterine contractions as well as the condition of the fetus. Extended

| TABLE 60-2 -- Agents Used for the Emergency Management of Preterm Labor | |
|---|---|
| **Ritodrine** | |
| Dose | 50 to 100 mug/min IV infusion |
| | 50 mug/min q 10 to 20 min |
| Endpoint | Cessation of uterine contrations |
| | Intolerable maternal side effects |
| | Maximum dose 350 mug/min |
| **Terbutaline** | |

| Dose | 0.25 mg SQ, may repeat q 20 to 60 min |
|---|---|
| | or |
| | 2.5 to 5 mug/min IV infusion, |
| | 2.5 to 5 mug/min q 20 min |
| Endpoint | Cessation of uterine contractions |
| | Intolerable maternal side effects |
| | Maximum dose 25 mug/min |

**Magnesium sulfate**

| Dose | 4 to 6 g IV over 20 min 2 to 4 g/hr IV infusion |
|---|---|
| Endpoint | Cessation of uterine contractions |
| | Signs of magnesium toxicity (e.g., depressed respirations, hypotension, somnolence) |

observation is undesirable because the effectiveness of tocolytic therapy diminishes as labor advances.

Ideally, when preterm labor is suspected, obstetric consultation should be obtained and the patient transferred immediately to the labor and delivery area for monitoring and

determination of fetal maturity. When appropriate obstetric facilities are not available, attempts to arrest labor should be initiated in the ED.

**Tocolytic Therapy**

Basic maneuvers to improve uterine and fetal status should be initiated before instituting pharmacologic tocolytic therapy when either preterm labor or fetal distress is suspected (Tables 60-2 and 60-3) . Because uterine hypoxia may induce uterine contractions, supplemental $O_2$ , IV infusion of 500 mL of crystalloid, and assumption of the left lateral decubitus position should be attempted to improve uterine perfusion.  Because uterine, cervical, or urinary tract infections account for 30 to 40% of cases of preterm labor, a specific cause should be sought and treated as appropriate. [7]

| TABLE 60-3 -- Procedure for Instituting a Ritodrine Infusion | |
|---|---|
| **Dose**\* **(mug/min)** | **Infusion Rate (mL/hr)** |
| 50  } Initial dose | 10 |
| 100 | 20 |
| 150 | 30 |
| 200 | 40 |
| 250 | 50 |
| 300 | 60 |
| 350 | 70 |

*Note:* This is *not* a dose/weight calculation.
If fluid restriction is necessary, the concentration can be increased.

 If contractions persist and cervical changes are documented despite these basic interventions, pharmacologic tocolytic therapy with either the selective beta2 -adrenergic agents or MgSO4 may be indicated. [7] Relative contraindications to tocolytic therapy include uncorrected fetal distress or fetal death, chorioamnionitis, ruptured or bulging membranes, or obstetric complications requiring early delivery, such as severe pregnancy-induced hypertension, maternal hemorrhage, or polyhydramnios. [3]

**beta $_2$ -Receptor agonists.**

The mainstays of pharmacologic tocolytic agents are the selective beta2 -adrenergic agents ritodrine (Yutopar) and terbutaline (Brethine, Bricanyl).  Ritodrine was approved by the U.S. Food and Drug Administration (FDA) for the inhibition of preterm labor in 1980 and is the only agent approved for tocolysis in the United States. Terbutaline has also been used extensively as a tocolytic agent, has documented efficacy in inhibiting labor, is less expensive than ritodrine, and is frequently more readily available to the emergency physician.

The beta-adrenergic agents prevent contraction of the myometrium through activation of the enzyme adenyl cyclase, which causes an increase in the intracellular concentration of cyclic adenosine monophosphate. This increased level of cyclic adenosine monophosphate initiates a series of cellular reactions that leads to the phosphorylation of cell membrane proteins and the sequestration of intracellular calcium. The reduced intracellular calcium concentration prevents activation of myosin light-chain kinase, which prevents the interaction of the smooth muscle filaments actin and myosin, which normally results in uterine smooth muscle contraction. [16] [18] Although both ritodrine and terbutaline stimulate beta2 -receptors primarily, both have some beta1 -activity, which is responsible for their cardiovascular side effects. Both drugs should therefore be used cautiously in patients with cardiovascular disease, hypertension, hyperthyroidism, or diabetes and in patients with bronchospastic disease concurrently on sympathomimetic bronchodilator agents.

Intravenous administration of ritodrine is used to stop contractions; therapy may be repeated if further episodes of preterm labor occur. [17] When given as an IV infusion, 150 mg ritodrine is mixed in 500 mL diluent, yielding a final concentration of 0.3 mg/mL (i.e., 300 mug/mL). Because of the risk of pulmonary edema, saline diluents should not be used unless use of dextrose-containing solutions is undesirable. [17] The initial infusion rate of ritodrine is 50 to 100 mug/min (i.e., 10 to 20 mL/hr), which is increased by 50 mug/min (i.e., by 10 mL/hr) every 10 to 20 minutes until uterine contractions cease, intolerable maternal side effects develop, or a maximum dose of 350 mug/min (i.e., 70 mL/hr) is reached. If unacceptable side effects develop, the infusion is reduced to a tolerable level. If labor is successfully arrested, IV therapy is continued for at least 12 hours after contractions cease. A controlled infusion device is required.

The usual clinical side effects of ritodrine are related to its inherent activity as a

beta-mimetic drug. Maternal cardiovascular effects include a dose-related increase in heart rate, an increase in systolic pressure, and a decrease in diastolic blood pressure. Fetal heart rate also increases slightly. About one third of patients experience palpitations, and up to 15% have chest pain, shortness of breath, tremor, nausea and vomiting, headache, or erythema. Pulmonary edema has also been reported with beta-mimetic tocolytic therapy both with and without concurrent glucocorticoid therapy for fetal lung maturation. Maternal metabolic effects during IV therapy include transient increases in glucose, free fatty acids, lactic acid, and insulin with a moderate decrease in potassium and calcium. Side effects are usually self-limited and resolve with dosage reduction or discontinuation of the drug. Treatment of the majority of side effects is supportive; severe cardiovascular effects may be treated with beta-blocking agents. [16]

Although not approved by the U.S. FDA for use as a tocolytic, terbutaline has gained widespread popularity for the treatment of preterm labor in the United States. Available in both a parenteral and an oral form, terbutaline may be given subcutaneously or by IV infusion for the emergency treatment of preterm labor. Subcutaneously administered terbutaline is rapidly absorbed with an absorption half-life of only 7 minutes. [20] Subcutaneous terbutaline is administered as a 0.25-mg dose, which may be repeated every 20 to 60 minutes until contractions cease or intolerable maternal side effects occur. Alternatively, terbutaline may be administered as an IV infusion. Although recommended dosage regimens vary, a conservative approach is to begin the infusion at a rate of 2.5 to 5 mug/min, increasing the rate by 2.5 to 5 mug/min every 20 minutes until a maximum dose of 25 mug/min is reached, uterine contractions cease, or adverse maternal side effects develop. Oral maintenance therapy should be individualized and typically ranges from 2.5 to 5 mg every 4 hours. Oral terbutaline, 30 mg/day, has been shown to be superior to ritodrine, 120 mg/day, in preventing recurrent labor. [21]

The side effects associated with the parenteral use of terbutaline are similar to those seen with ritodrine. Many authors consider subcutaneous terbutaline preferable to ritodrine for the treatment of preterm labor because of terbutaline's lower cost, ease of administration, and comparable efficacy and side effects.

**Magnesium sulfate.**

Magnesium sulfate ($MgSO_4$ ) is not approved in the United States for use as a tocolytic agent. Nevertheless, many perinatal centers prefer $MgSO_4$ over the beta-mimetic agents owing to its comparable efficacy and low incidence of intolerable side effects. [7] [22] Although its effect is not fully understood, magnesium probably acts by competitively inhibiting calcium uptake into the muscle cell, thereby inhibiting the actin/myosin interaction.

When used as a tocolytic agent, 4 to 6 g of $MgSO_4$ is given IV over 20 to 30 minutes, followed by a maintenance IV infusion beginning at 2 g/hour and titrated to the cessation of uterine contractions or clinical evidence of magnesium toxicity. [17] [22] Infusion rates of 3 to 4 g/hour are usually required to maintain tocolysis. [18]

Intravenous infusion of $MgSO_4$ typically produces sweating, warmth, and flushing; and rapid parenteral administration may cause transient nausea, vomiting, headache, or

palpitations.Infrequently, pulmonary edema or chest pain has necessitated stopping the drug. [22] The major side effect of magnesium therapy is related to impairment of the muscles of respiration with subsequent respiratory arrest, an effect usually not seen until the serum magnesium level exceeds 10 mEq/L. The first sign of magnesium toxicity, decrease of the patellar reflex, typically occurs as serum magnesium levels exceed 4 mEq/L, with loss of the reflex as levels approach 10 mEq/L. Therefore, the patellar reflex should be monitored throughout therapy. Respiratory depression typically occurs when serum magnesium levels exceed 10 mEq/L; and at 12 mEq/L or greater, respiratory arrest may occur. Although high concentrations of magnesium (10 to 15 mEq/L) can increase cardiac conduction manifested by lengthened PR and QRS intervals on the electrocardiogram, the major cause of cardiac dysfunction in humans results from respiratory arrest.

Because magnesium is almost totally excreted by the kidney, magnesium is contraindicated in the presence of renal failure, and urinary output and renal function should be monitored throughout therapy. If respiratory depression develops, 10 mL of a 10% solution of calcium gluconate or calcium chloride injected over 3 minutes is an effective antidote. For severe respiratory depression and arrest, prompt endotracheal intubation may be lifesaving. MgSO$_4$ should be used with caution in patients with impaired renal function or heart disease and is contraindicated in patients with myasthenia gravis.

## VAGINAL BLEEDING DURING THE THIRD TRIMESTER

Bleeding during the third trimester should always be considered an emergency. Profound shock secondary to exsanguinating hemorrhage may occur within minutes. Although bleeding may result from local vaginal and cervical lesions, genital lacerations, circumvallate placenta, vasa previa, or rupture of the uterus, the most frequent causes are that due to placenta previa and that resulting from placental abruption. [3] *Placenta previa* refers to implantation of the placenta in the lower uterine segment with varying degrees of encroachment on the cervical os. Complicating 1 in 200 to 1 in 250 pregnancies that continue beyond the 28th week of gestation, [3] placenta previa is characterized clinically by vaginal bleeding with little or no abdominal or pelvic pain. Premature separation of the placenta, or *abruptio placentae*, refers to separation of the placenta from its site of implantation in the uterus before delivery of the fetus. Placental abruption occurs in 1 in 86 to 1 in 206 advanced pregnancies and is responsible for approximately 30% of all late pregnancy bleeding. [3] In contrast to placenta previa, abruptio placentae is associated with varying degrees of abdominal pain and uterine irritability. The degree of clinical shock may be out of proportion to the amount of apparent hemorrhage. Fetal distress is present in more than half the cases. [3]

As noted previously, third-trimester vaginal bleeding should always be considered an emergency. Stabilization should be initiated with at least 2 large-bore IV lines. In addition to routine laboratory work-up and the taking of blood for type and crossmatching, clotting studies, including a fibrinogen level, should be done. Vaginal examination is *contraindicated* in the ED because of the possibility of tearing or dislodging a placenta previa, which may result in profuse, potentially fatal hemorrhage. [1] The patient should be immediately transferred to the care of her obstetrician for

further evaluation. Definitive diagnosis by means of the "double set-up" examination has been replaced by localization of the placenta by ultrasound. The accuracy of placental localization and, therefore, the confirmation of placenta previa is between 93 and 98%. For unknown reasons, blood separating the placenta from its attachment is difficult to detect with ultrasound. *Ultrasonography, therefore, has limited value in excluding the presence of abruptio placentae* but is of great value in excluding the diagnosis of placenta previa.

## DELIVERY

Full dilation of the cervix signifies the second stage of labor, heralding delivery of the infant. Typically, the patient begins to bear down and, with descent of the presenting part, develops the urge to defecate. Uterine contractions may last 1.5 minutes and recur after a myometrial resting phase of <1 minute.

The mechanism of labor in vertex and breech presentations consists of engagement of the presenting part, flexion, descent, internal rotation, extension, external rotation or restitution, and expulsion (Fig. 60-8) (Figure Not Available) . The mechanism of labor is determined by the pelvic dimensions and configuration, the size of the fetus, and the strength of uterine contractions. Essentially, the fetus will follow the path of least resistance by adaptation of the smallest achievable diameters of the presenting part to the most favorable dimensions and contours of the birth canal.

The sequence of movements in vertex presentations is as follows:

1. *Engagement*. Usually occurring in the last 2 weeks of pregnancy in the primiparous patient and at the onset of labor in the multiparous patient, engagement refers to the mechanism by which the greatest transverse diameter of the head, the biparietal diameter in occiput presentations, passes through the pelvic inlet.
2. *Flexion*. Flexion of the head is necessary to minimize the presenting cross-sectional diameter of the head during passage through the smallest diameter of the bony pelvis. In most cases, flexion is necessary for both engagement and descent.
3. *Descent*. Descent is gradually progressive and is effected by uterine and abdominal contractions as well as by straightening and extension of the fetal body.
4. *Internal rotation*. Internal rotation occurs with descent and is necessary for the head or presenting part to traverse the ischial spines. This movement is essentially a turning of the head such that the occiput gradually moves from its original, more transverse position, anteriorly toward the symphysis pubis or, less commonly, posteriorly toward the hollow of the sacrum.
5. *Extension*. After internal rotation, the sharply flexed head reaches the anteriorly directed vulvar outlet, undergoing extension. With increasing distention of the perineum and vaginal opening, an increasingly larger portion of the occiput appears gradually. The head is born by further extensions as the occiput, bregma, forehead, nose, mouth, and finally chin pass successively over the anterior margin of the perineum. Immediately after its birth, the head drops downward such that the chin lies over the maternal anal region.

6. *External rotation.* External rotation or restitution follows delivery of the head as it rotates to the transverse position that it occupied at engagement. After this movement, the shoulders descend in a path similar to that traced by the head, rotating anteroposteriorly for delivery. First, the anterior shoulder is delivered beneath the symphysis pubis, followed by the posterior shoulder across the perineum. Expulsion of the remainder of the fetal body occurs with ease.

The mechanism of the labor for breech presentations varies (Fig. 60-9) (Figure Not Available) . Usually the hips engage in one of the oblique diameters of the pelvic inlet. As descent occurs, the anterior hip generally descends more rapidly than the posterior hip. Internal rotation occurs as the intertrochanteric diameter assumes the anteroposterior position. Lateral flexion occurs as the anterior hip catches beneath the symphysis pubis, allowing the posterior hip to be born first. The infant's body then rotates, allowing for engagement of the shoulders in an oblique orientation. There is gradual descent, with the anterior shoulder rotating to bring the shoulders into the anteroposterior diameter of the outlet. The anterior shoulder follows lateral flexion to appear beneath the symphysis, with the posterior shoulder delivered first as the body is supported. The head tends to engage in the same diameter as the shoulders. Subsequent flexion and descent of the head occurs, following the path of the shoulders. Internal rotation occurs toward the hollow of the sacrum.Delivery of the infant usually occurs spontaneously. The role of the physician or attendant is principally to provide control of the birth process, preventing forceful, sudden expulsion or extraction of the infant with resultant fetal and maternal injury.

## Equipment for Delivery

The following typical obstetric equipment pack should be available in the ED (Fig. 60-10) . The optional equipment may or may not be needed.

**Obstetric pack (sterile)**

   1 large basin (for placenta)

   1 pair of scissors

   2 medium Kelly clamps

   1 bulb syringe

   1 plastic umbilical cord clamp

   3 sterile towels

   1 package of gauze sponges

   1 baby blanket

   2 pairs of sterile gloves

Sterile tubes for placental blood collection

**Optional**

Infant resuscitation tray

Warm blankets

Name bands

Heated isolette

## Technique for Uncomplicated Delivery

Owing to the high bacterial content of the vagina and perineum, complete sterility is not a priority. [25] When time permits, however, sterile technique should be used. The hands should be cleansed and sterilely gloved. The perineum should be cleansed as described for vaginal examination and draped such that only the immediate area about the vulva is exposed. Care should be taken to avoid fecal contamination of the infant. Equipment in the obstetric pack should be sterile.

The patient should be positioned on a stretcher with her hips and knees partially flexed, her thighs abducted, and the soles of her feet placed firmly on the stretcher. The delivery position may be enhanced by placing the patient's buttocks on the underside of a sterile bedpan, providing up to 12 cm of additional space between the bed and the perineum.

**Vertex Delivery**

Spontaneous delivery of the vertex-presenting infant is divided into 3 phases: delivery of the head, delivery of the shoulders, and delivery of the body and legs.

Delivery should be anticipated when the presenting part reaches the pelvic floor. With each contraction, the perineum bulges increasingly and the vulvovaginal opening becomes more and more dilated by the fetal head. Just before delivery, "crowning" occurs; the head is visible at the vaginal introitus, and the widest portion, or biparietal diameter, of the head distends the vulva.

*Gentle, gradual, controlled delivery is desirable.* As the fetal head becomes progressively more visible, one palm of the physician's hand is placed over the occipital area, providing gentle pressure to control delivery of the head. *Explosive delivery of the head should be avoided.* The other hand, preferably draped with a sterile towel to protect it from the anus, may exert forward pressure on the chin of the fetus through the perineum just in front of the coccyx in a modified Ritgen maneuver (Fig. 60-11) (Figure Not Available) . This maneuver extends the neck at the proper time, thereby protecting the maternal perineal musculature.

The head is gently supported during subsequent delivery of the forehead, face, chin, and neck.

After the head has been delivered, the infant's face and mouth should be quickly wiped and the oral cavity and nares should be suctioned with a bulb syringe. This minimizes the chance of aspiration of amnionic fluid, debris, and blood, which may occur with inspiration during delivery of the thorax.

With delivery of the neck, a finger should be passed around the neck to determine whether it is encircled by one or more coils of the umbilical cord (Fig. 60-12) . If a cord is felt, it should cautiously be loosened and gently slipped over the infant's head. If this cannot be done easily, the cord should be doubly clamped and cut and the infant should be delivered promptly. In approximately 25% of deliveries, the umbilical cord is around the infant's neck but is rarely tight enough to cause fetal hypoxia. [1]

Just before external rotation, the head usually falls posteriorly, bringing it almost into contact with the mother's anus. As rotation occurs, the head assumes a transverse position and the transverse diameter of the thorax or bisacromial diameter rotates into the anteroposterior diameter of the pelvis. In most cases the shoulders are born spontaneously. Delivery may be aided by grasping the sides of the head and exerting *gentle* downward (posterior) traction until the anterior shoulder appears beneath the symphysis pubis. The head is then *gently* lifted upward to aid the delivery of the posterior shoulder (see Fig. 60-8) (Figure Not Available) . The remainder of the body usually follows without difficulty.

Delivery may be assisted by *gentle* traction on the head after the shoulders have been freed. Hooking the fingers in the axilla during delivery may result in brachial plexus injury, hematoma of the neck, or fracture of the clavicle; thus, it should be avoided. Furthermore, traction should always be exerted in the direction of the long axis of the child; if applied obliquely, traction may cause bending of the neck and excessive stretching of the brachial plexus.

Immediately after delivery the infant should be held with the head lower than the body, at an angle of *not* more than 15° to facilitate drainage of accumulated mucus and bronchial secretions in the airway. The infant's airway should be thoroughly suctioned. Although some controversy exists as to the optimal position of the infant in relation to the mother during this stage, most authorities recommend that the infant be placed at or slightly below the level of the vaginal introitus for 30 seconds before the cord is clamped. [1] [26] This allows up to 80 mL of blood to be transfused from the placental circulation into the infant. [4]

The umbilical cord should be cut 30 to 60 seconds after delivery. Blood samples from the placental end of the cord should be collected for determination of infant serology, including rhesus factor (Rh) studies. Two sterile clamps should be placed several inches apart, and the cord between the clamps should be cut with sterile scissors. A sterile cord clamp or cord tie of umbilical (cloth) tape is then placed around the cord, 2 to 3 cm

from the infant abdomen. [1]

Immediately after the cutting of the umbilical cord, the infant should briefly be evaluated and, if necessary, resuscitation should be initiated. Because of the relatively large surface area of the neonate, attention should be directed toward maintaining body temperature by drying the neonate and placing the baby in a heated isolette, under a radiant warmer, or in warm blankets. [1] [7]

*Meconium staining* of the amniotic fluid occurs in infants older than 34 weeks' gestation who have suffered intrauterine asphyxia and stress. [27] It is more common in post-term pregnancy and that complicated by fetal growth retardation. [1] Fetal aspiration of meconium may lead to meconium pneumonitis, which carries a mortality of 20 to 35%. [27] To minimize the risk of aspiration, all infants with meconium-stained amniotic fluid should have the nares and oropharynx thoroughly suctioned after delivery of the head but before delivery of the thorax and the first thoracic expansion. [1] [7] [28] As soon as possible after delivery, the traditional approach mandates direct visualization of the vocal cords with aspiration of supraglottic meconium followed by tracheal intubation and thorough suctioning of the trachea. [1] [7] The traditional approach has been questioned, and some authorities now recommend a selective approach to direct visualization of the glottis and tracheal suctioning, reserving this procedure for those infants with moderate to thick meconium staining and respiratory depression or distress at birth. [3] [28]

**Delivery of the Placenta**

Placental separation usually occurs within 5 minutes after delivery of the infant and may be recognized by the following signs:

1. The uterus becomes globular and firmer as it contracts.
2. The uterine fundus rises in the abdomen.
3. There is a sudden gush of blood.
4. The umbilical cord protrudes further out of the vagina, indicating placental descent.

The placenta usually delivers spontaneously within 20 to 30 minutes. [7] Intra-abdominal pressure produced by the mother's bearing down efforts may be enough to effect complete expulsion of the placenta. If maternal force alone is insufficient, delivery of the placenta may be aided by the physician. After ensuring that the uterus is firmly contracted, the physician uses one hand to exert gentle pressure through the abdominal wall to lift the uterine fundus cephalad while keeping the umbilical cord slightly taut with the other hand. This is repeated until the placenta reaches the introitus, at which time uterine pressure is stopped and the placenta is gently lifted upward out of the vagina (Fig. 60-13) (Figure Not Available) . Membranes that are adherent to the uterine lining should be grasped with a clamp or ring forceps and removed by gentle traction. Excessive traction should never be used to pull the placenta out of the uterus, because traction may result in uterine inversion with catastrophic hemodynamic consequences. The placenta should be examined for completeness and saved for later evaluation by the obstetrician. [1] [3] [7] The vulva, vagina, and cervix should be examined to assess for traumatic lacerations. Cervical lacerations most typically occur at the 9 or 3 o'clock

position; vaginal lacerations typically occur at the point of the ischial spines. If found, these injuries are best repaired in the operating room or delivery suite. [7] After delivery of the placenta the uterus should be palpated to ensure that it is well contracted. A normally contracted uterus will be firm with its upper margin just below the maternal umbilicus. If persistent bleeding occurs from a flaccid uterus, gentle uterine massage through the abdominal wall and oxytocic agents may be used as necessary. [1] Oxytocic agents should not be used before delivery of the placenta because the resultant uterine contraction may entrap the placenta within the uterus. Use of oxytocics before delivery of an undiagnosed second twin may prove fatal to the entrapped fetus.Oxytocin (Pitocin, Syntocinon) is the most commonly used oxytocic drug and is usually given by IV infusion. Twenty units of oxytocin is added to 1 L of normal saline and is given at a rate of 10 mL/min for several minutes until the uterus remains firmly contracted and bleeding is controlled. The infusion rate is then reduced to 1 to 2 mL/min. [1] [7] If bleeding persists, an ergot derivative, such as methylergonovine maleate (Methergine), 0.2 mg, may be given intramuscularly. [7] Because of their vasoconstrictive properties, ergot preparations are relatively contraindicated in patients with hypertension and preeclampsia. [16] Occasionally, the placenta may fail to separate completely, resulting in a retained placenta or placental fragments, with persistent uterine bleeding. Manual removal of the placenta, exploration of the uterine cavity for retained products, and, occasionally, hysterectomy are indicated.  These procedures are beyond the scope of ED care and should be left to the obstetrician. The patient should be supported with IV fluid and blood transfusion as indicated until definitive therapy is available. Constant firm uterine massage can lessen hemorrhage and may be life saving.

## Complex Deliveries

### Shoulder Dystocia

The term *shoulder dystocia* refers to impaction of the fetal shoulders in the pelvic outlet occurring after delivery of the head in vertex presentations. Shoulder dystocia complicates 0.15 to 0.6% of all vaginal deliveries and 1.7% of deliveries involving infants weighing >4000 g.Impaction of the fetal shoulders and thorax in the maternal pelvis prohibits adequate respiration, and compression of the umbilical cord frequently compromises fetal circulation, making shoulder dystocia a serious, and at times fatal, complication of delivery. Although shoulder dystocia occurs more commonly with large infants, it cannot be reliably predicted from any antepartum risk factors. Shoulder dystocia is associated with post-term pregnancy, fetal macrosomia, diabetes mellitus, maternal obesity, and multiparity.

**Management.**

General anesthesia is desirable but is seldom available in the ED. A wide episiotomy reduces the incidence of major perineal lacerations and provides additional space for manipulation. Because the techniques used to treat shoulder dystocia frequently require an assistant and delivery can result in fetal injury or hypoxia, assistance, including that of a pediatrician and obstetrician, should be emergently sought. Although reduction in the interval of time from delivery of the head to delivery of the body is of great importance to infant survival, overly vigorous traction on the fetal head or neck or

excessive rotation of the body may seriously damage the infant.

Most cases of shoulder dystocia can be resolved with 1 of 2 simple maneuvers. First, placing the mother in the extreme lithotomy position with her hips completely flexed, allowing the knees to rest on her chest (McRobert's maneuver), may cause the fetal shoulders to engage appropriately and enable delivery to progress. Alternatively, moderate suprapubic pressure applied to the maternal abdomen by an assistant while gentle downward traction is exerted on the fetal head may also result in delivery. [1] [32] If these simple maneuvers fail to effect delivery, several other techniques exist, the choice of which will depend on operator preference and experience. In the first maneuver, the operator places 2 fingers into the vagina and exerts pressure on the fetal scapula, rotating the posterior shoulder 180° in a corkscrew fashion (Wood's corkscrew maneuver). This may cause the impacted anterior shoulder to be released and delivery to progress (Fig. 60-14) (Figure Not Available) . Alternatively, delivery of the posterior shoulder may be attempted. Here, the physician's hand is inserted into the vagina along the hollow of the maternal sacrum. The posterior arm of the fetus is then carefully swept across its chest, followed by delivery of the arm. The shoulder girdle is then rotated into one of the oblique diameters of the pelvis with subsequent delivery of the anterior shoulder. Although this procedure may fracture the clavicle or humerus, these injuries are preferable to permanent brachial plexus or neurologic injury. [7] If all of these strategies fail, controlled destructive procedures such as fracture of the fetal clavicle may be necessary.

**Breech Delivery**

When compared with cephalic presentations, the breech delivery is associated with a greater incidence of prematurity, prolapsed cord, low implantation of the placenta, uterine and congenital abnormalities, multiple pregnancies, and increased perinatal morbidity and mortality rates. [1] [36] [37] The incidence of breech presentation varies inversely with gestational age and weight. At term, the incidence of breech presentation is 3 to 4%; from 28 to 38 weeks' gestation, 17%; and at <28 weeks' gestation, 40%. [38]

Increased rates of prematurity and congenital anomalies associated with breech presentation account for much of the perinatal loss. [36] [39] In addition, fetal distress also occurs more frequently. Whereas umbilical cord prolapse occurs in only 0.3 to 0.5% of vertex presentations, the incidence rate increases to 3.8 to 7.4% in breech presentations generally. [38] [39] Cord prolapse is more common among non-frank breech presentations, reaching an incidence of 8 to 16% and from 10 to 28.5% in double footling and single footling presentations, respectively. [39]

The increased use of cesarean section has greatly decreased the morbidity and mortality associated with breech delivery. Although cesarean section has been traditionally considered the standard of care, vaginal delivery may be the method of choice in carefully selected cases. The emergency physician seldom is called on to make the decision as to the most appropriate means of delivery but, rather, could be faced with the imminent vaginal delivery of the breech infant. Breech delivery is most appropriately performed with both a physician and an assistant present.

**Types.**

There are 3 types of vaginal breech delivery. *Spontaneous breech* is a breech delivery in which the infant is delivered spontaneously without any manipulation or traction other than supporting the infant. Although this form of delivery is rare with term infants, there is little associated traumatic morbidity. *Partial breech extraction* is when the infant is delivered spontaneously as far as the umbilicus and the remainder of the body is extracted. *Total breech extraction* is when the entire body of the infant is extracted by the physician.

Delivery is easier and perinatal morbidity and mortality are reduced when the breech is born spontaneously to the level of the umbilicus. [1] If fetal distress develops before this time, however, a decision must be made whether to perform a total breech extraction or prepare for cesarean section. Total breech extraction is indicated only if there is a definite diagnosis of fetal distress unresponsive to routine maneuvers and cesarean section cannot be performed promptly. To perform any vaginal breech delivery, the birth canal must be sufficiently large to allow passage of the fetus without trauma and the cervix must be completely effaced and dilated. If these conditions do not exist, a cesarean section is indicated. To ensure full cervical dilation in the footling or complete breech, it is important that the feet, legs, and buttocks advance through the introitus to the level of the fetal umbilicus before the physician intervenes in delivery and further extraction is attempted. The mere appearance of the feet through the vulva is not in itself an indication to proceed with delivery. This may be a footling presentation through a cervix that is not completely dilated. In this case there may be time to transfer the patient to the labor and delivery suite, preferably in the knee-chest position to minimize the risk of cord compression. [32]

**Technique.**

If fetal distress is documented and total extraction is deemed necessary, the following procedures are carried out. The physician's hand is introduced into the vagina, and both feet of the fetus are grasped, with the index finger placed between the fetal ankles. Gentle traction is applied until the feet are pulled through the vulva (Fig. 60-15) (Figure Not Available) . At this point, a wide episiotomy is usually made. Because the legs are slippery and difficult to hold due to vernix caseosa, they should be wrapped in a sterile towel as they emerge through the vulva. Downward gentle traction is continued as successively higher portions of the legs and thighs are grasped (Fig. 60-16) (Figure Not Available) . When the breech appears at the vulva, gentle traction is applied until the hips are

delivered. As the buttocks emerge, the fetal back usually rotates anteriorly. The thumbs of the physician are then placed over the sacrum while the fingers are placed over the hips, and gentle downward traction is continued (Fig. 60-17) (Figure Not Available) . As the scapulas emerge, the infant usually rotates back to its original position, with the back directed laterally.

If spontaneous rotation does not occur, slight rotation should be added to the traction to bring the bisacromial diameter of the fetus into the anteroposterior diameter of the pelvis (Fig. 60-18) (Figure Not Available) . Delivery of the shoulders should not be attempted until the lower halves of the scapula are delivered outside the vulva and the axilla becomes visible at the introitus. Two methods of shoulder delivery are commonly used. In the first method, with the scapulas visible, the trunk is rotated such that the anterior arm and shoulder appear at the vulva and can be easily released and delivered. The body of the fetus is then rotated in the reverse direction to deliver the other shoulder and arm beneath the symphysis pubis. In the second method, if trunk rotation is unsuccessful, the posterior shoulder must be delivered first. The feet are grasped in one hand and drawn upward over the mother's groin. In this manner, leverage is exerted on the posterior shoulder, which slides out over the perineal margin, usually followed by the arm and hand. The anterior shoulder, arm, and hand are then delivered beneath the symphysis pubis by downward traction on the fetal body (Fig. 60-19) (Figure Not Available) .

Occasionally, spontaneous delivery of the arm and hand does not follow delivery of the shoulder. If this occurs, upward traction of the fetal body should be continued after delivery of the posterior shoulder. Two fingers of the physician are then passed along the fetal humerus until the fetal elbow is reached. The fingers are used to splint the fetal arm, which is then swept downward and delivered. The anterior arm may then be delivered by depression of the fetal body alone. In some cases it may be necessary to sweep the anterior arm down over the thorax using 2 fingers as a splint.

After the shoulders appear, the head usually occupies one of the oblique diameters of the pelvis, with the chin directed posteriorly. The head may then be extracted using the Mauriceau maneuver. With the fetal body resting on the physician's palm and forearm, the index and middle finger of the hand are placed over the infant's maxilla, flexing the fetal head. Two fingers of the other hand are hooked over the fetal neck, and, grasping the shoulders, the physician applies downward traction until the suboccipital region appears under the symphysis pubis (Fig. 60-20) (Figure Not Available) . As the body of the fetus is then elevated toward the mother's abdomen, the fetal mouth, nose, brow, and eventually occiput successively emerge over the perineum. Suprapubic pressure applied by an assistant is helpful in delivery of the head. If delivery of the head is not effected by the Mauriceau maneuver, forceps delivery may be necessary. Forceps application is beyond the scope of this text.

At times, delivery of a frank breech may be necessary. Facilitated by an episiotomy, the breech should be allowed to deliver spontaneously as far as possible. Moderate traction may be exerted by a finger placed in each fetal groin (Fig. 60-21) (Figure Not Available) . Once the knees appear outside the birth canal, the legs may be slowly flexed to assist delivery, which usually occurs without trauma.

**Complications.**

Traumatic morbidity associated with breech presentations is approximately 12 times that of vertex presentations and is directly related to the manipulations used to effect

delivery and the relationship of fetal size to that of the maternal pelvis. [41] One of the most significant features of breech delivery is that progressively increasing diameters and less distensible fetal parts must traverse the maternal pelvis.

If cephalopelvic disproportion is not diagnosed until the fetal body has been delivered and if the fetal head will not descend, the prognosis is grave. In addition, whereas in vertex presentations, in which molding the fetal head is gradual, occurring over the course of hours, in breech presentation, molding is abrupt, subjecting the delicate supporting tissues of the aftercoming head to sudden and often violent stresses. Intracranial hemorrhage is the most frequent cause of death in breech delivery, with injury to the spinal cord, liver, adrenal glands, and spleen occurring in decreasing order of frequency. [1] [42]

Both assisted breech delivery and breech extraction involve a change in motive powers for delivery, substituting traction from below for pressure from above and increasing the probability of occurrence of unilateral or bilateral nuchal arms or hyperextension of the fetal head. Decomposition and extraction of a frank breech infant carry significantly increased fetal risks and impose additional maternal risks because of intrauterine manipulation. The fact that manipulation does indeed correlate with traumatic morbidity was illustrated by Rovinsky and coworkers, who found that, whereas spontaneous breech delivery was associated with no traumatic complications, increasing degrees of obstetric manipulation corresponded to progressive elevations of the traumatic morbidity rate. [41] The highest traumatic maternal morbidity rate, 8.3%, which is 10 times the rate for assisted breech delivery and 4 times the rate for breech extraction, occurred with breech decomposition and extraction.

## EPISIOTOMY

An episiotomy is an incision of the posterior vaginal wall and a portion of the pudenda, which is made to enlarge the vaginal introitus to permit easier passage of the fetus and theoretically to prevent perineal lacerations of the mother, preserving the structure and function of the vaginal introitus. In 1987, >60% of women in the United States received an episiotomy during childbirth. [43] The routine use of the procedure, however, has been questioned, with studies indicating a higher incidence of third- and fourth-degree perineal lacerations, a longer healing period, and increased perineal pain in women receiving episiotomies. ] When used appropriately, an episiotomy helps to minimize compression and trauma of the fetal head; facilitates the second stage of labor by removing the resistance of the pudendal musculature; and substitutes a straight surgical incision for the ragged laceration that may result from tearing of the musculature, making repair easier. [3] The traditional beliefs that episiotomy also results in less postoperative pain and may improve recovery of the pelvic floor musculature appear not to be true. Therefore, *the routine use of episiotomy, particularly in the primiparous patient, is no longer recommended, and a selective approach is now encouraged*. Indications for selective episiotomy include preterm or breech delivery, shoulder dystocia, occiput posterior presentations, and when perineal tear is imminent. [1] [3]

Two types of episiotomy are used: the median or midline episiotomy and the mediolateral episiotomy (Fig. 60-22) (Figure Not Available) .  The median incision is the

easiest type of episiotomy to perform and repair, results in the least amount of blood loss, and heals rapidly with minimal discomfort. The major disadvantage to the median incision is a 5 to 10% incidence of accidental extension of the incision into the anal sphincter or rectum, resulting in third- and fourth-degree lacerations, respectively. Although prompt recognition and proper repair of third-degree lacerations frequently have excellent results, [47] fourth-degree lacerations can result in significant morbidity. Although a mediolateral episiotomy seldom results in extension through the anal sphincter, blood loss is generally greater, repair is more difficult, and painful healing may result.

## Equipment

The following equipment is required for episiotomy and repair:

> Tissue scissors or scalpel (with tongue blade)
>
> 3-0 or 2-0 absorbable suture on atraumatic needle (e.g., chromic catgut or polyglycolic acid)
>
> Needle holder
>
> Suture scissors
>
> Gauze pack

## Technique

The episiotomy should be timed so that it precedes trauma to the maternal tissues and fetus but avoids excessive maternal blood loss before delivery. With vertex presentations, the episiotomy should be performed when the fetal head begins to distend the perineum and the caput becomes visible to a diameter of 3 to 4 cm during a contraction. [1] [49] With breech delivery, the episiotomy is usually performed just before breech extraction. [3] Anesthesia for episiotomy in the ED is usually limited to local infiltration of the perineum with 1 or 2% lidocaine.

The episiotomy is a simple incision that extends through the skin and subcutaneous tissues, the vaginal mucosa, the urogenital septum, the superior fascia of the pelvic diaphragm, and, if the episiotomy is mediolateral and deep, through the lowermost fibers of the puborectalis portion of the levator ani muscles. The incision may be made with either a scissors or a scalpel. When using a scalpel, a tongue blade is placed between the infant's head and the maternal perineum as the perineum is incised. For the median episiotomy, the incision is made through the median raphe of the perineum almost to the anal sphincter (Fig. 60-23) (Figure Not Available) . For the mediolateral episiotomy, the incision is directed downward and outward in the direction of the lateral margin of the anal sphincter and may be either to the right or to the left (see Fig. 60-22) (Figure Not Available) .

After delivery of the infant and placenta, the episiotomy is repaired. The goals of

episiotomy repair are to restore anatomy and achieve adequate hemostasis with a minimum of suture material. It is preferable to perform the closure after delivery of the placenta and after inspection and repair of the cervix and upper vaginal canal. The same principles of repair are followed for both the median (Fig. 60-24) (Figure Not Available) and mediolateral (Fig. 60-25) (Figure Not Available) episiotomies.

Because there is minimal tension on the closed wound, most authorities recommend the use of 2-0 or preferably 3-0 absorbable suture such as chromic catgut or polyglycolic acid on a large, atraumatic needle. The first step is to close the vaginal mucosa using a continuous suture from just above the apex of the incision to the mucocutaneous junction, reapproximating the margins of the hymenal ring. Burying the closing knot in the incision minimizes the amount of scar tissue and prevents tenderness and dyspareunia. All large actively bleeding vessels should be separately ligated during closure with separate absorbable suture ligatures. Next, the perineal musculature is reapproximated with 3 or 4 interrupted sutures. Closure of the superficial layers may be accomplished by 1 of 2 methods. In the first method, a continuous suture is used to close the superficial fascia from the mucocutaneous junction outward and is then continued upward as a subcuticular skin closure, returning to and ending at the mucocutaneous junction. Alternatively, several interrupted sutures may be placed through the skin and subcutaneous fascia and loosely tied. This last method of skin closure avoids burying 2 layers of suture in the more superficial layers of the perineum.

During episiotomy closure, it is helpful to maintain a dry field by inserting a large gauze pack in the vagina, leaving a tail or portion of the pack outside the vagina to aid in removal after the repair.

The most common complication of episiotomy is hematoma formation, owing to inadequate hemostasis; a hematoma is treated by evacuation and drainage. Occasionally, large bleeding vessels will require delayed ligation. Postpartum episiotomy pain can usually be controlled by analgesics and local heat or sitz baths. Infection is a rare complication and responds readily to adequate drainage and appropriate antibiotic therapy.

## IMMEDIATE POSTPARTUM HEMORRHAGE

Postpartum hemorrhage is traditionally defined as maternal blood loss >500 mL after delivery. Although this is the classic definition, ample evidence indicates that normal blood loss after vaginal delivery is frequently 500 mL. [1] [50] Therefore, in clinical practice the diagnosis of postpartum hemorrhage is usually made when the amount of bleeding exceeds the clinician's estimate of "normal." The most common cause of serious obstetric hemorrhage, postpartum hemorrhage accounts for a significant proportion of maternal deaths. [37] Postpartum hemorrhage is conventionally divided into immediate hemorrhage occurring within 24 hours of delivery and delayed hemorrhage occurring >24 hours after delivery.

Postpartum hemorrhage is frequently characterized by steady moderate bleeding that persists until serious hypovolemia develops rather than by sudden massive hemorrhage. Because of the relative hypervolemia that occurs during normal

pregnancy, blood loss may exceed 1500 mL before significant clinical changes in pulse and blood pressure become manifest. [51] Careful observation for blood loss, including evaluation of uterine size and consistency, is therefore mandatory during the early postpartum period.

The most common causes of immediate postpartum hemorrhage, in order of decreasing frequency, are uterine atony, lacerations of the vagina and cervix, and retained placenta or placental fragments. Less commonly, coagulation disorders, uterine rupture, uterine inversion, and paravaginal vessel laceration result in postpartum blood loss.

## Management

Management of postpartum hemorrhage consists of replacement of intravascular volume with crystalloid and blood products as needed, as well as therapy directed toward the cause of hemorrhage. The diagnosis of uterine atony, the most common cause of bleeding, is made when uterine palpation reveals a soft boggy uterine corpus. Although the diagnosis may be suspected on the basis of abdominal examination alone, bimanual pelvic examination is frequently necessary to confirm the diagnosis.

*Uterine atony* is managed by manual massage of the uterine fundus (Fig. 60-26) (Figure Not Available) . One hand of the physician is used to compress and massage the posterior aspect of the uterus through the abdominal wall while the knuckles of the other sterile gloved hand are used to gently massage the anterior aspect of the uterus through the vaginal wall. Care must be taken to avoid vigorous downward massage, which can result in acute uterine inversion. Oxytocic agents should be administered in conjunction with massage.

### Oxytocics

Oxytocin is ideally administered as an IV infusion, which is prepared by adding 20 to 40 units of oxytocin to 1 L of crystalloid and infused at a rate of 200 to 500 mL/hour, titrated to sustain uterine contractions and control uterine hemorrhage. Oxytocin should never be administered as an IV bolus because profound hypotension and myocardial arrhythmias may occur. If IV access is unavailable, 10 units of oxytocin may be given intramuscularly. [17] Because the onset of action of IV oxytocin is rapid, uterine contractions and therefore slowing of hemorrhage should be observed within minutes of administration. If bleeding persists and the uterus remains boggy despite oxytocin therapy, 0.2 mg of methylergonovine (Methergine) should be given intramuscularly to help stimulate uterine contractions. Typically, uterine contractions occur within minutes of ergot administration and last for several hours. Because of their tendency to cause vasoconstriction and severe hypertension in some patients, ergot preparations should be avoided in women who are known to be hypertensive [52] and should be used cautiously in women who have cardiac, hepatic, renal, or obliterative vascular disease. [17] Uterine atony that remains refractory to

these measures may respond to treatment with prostaglandins. Although intramyometrial injection or instillation of vaginal prostaglandin suppositories has been described, [53] most reports describing the successful management of uterine atony have

involved the intramuscular administration of 15-methyl prostaglandin F$_{2alpha}$ (carboprost tromethamine [Hemabate]).  Intramuscular carboprost is administered in a dose of 0.25 mg and may be repeated at intervals of 15 to 90 minutes as determined by the clinical course. The total dose should not exceed 2 mg. [17] The rate of successful control of uterine hemorrhage with this regimen exceeds 85%. Because nausea, vomiting, and diarrhea occur frequently in patients receiving prostaglandins, concurrent administration of antiemetic and antidiarrheal agents is recommended. [17]

**Procedures**

If vaginal bleeding persists despite uterine massage and a firmly contracted uterus, a cause other than uterine atony should be suspected. The labia, vagina, and cervix should be carefully inspected for lacerations. Bleeding from lacerations may be controlled by direct pressure or, in the case of cervical lacerations, by gentle application of the ring forceps to the bleeding point. Absorbable sutures may be used to control bleeding from easily accessible lacerations. Because adequate visualization of the cervix and upper vagina is difficult and repair of extensive lacerations frequently requires general anesthesia, repair of these lacerations is often better left to the obstetrician.

For the patient in extremis, *external abdominal aortic compression* has been recommended. This procedure is performed by pressing the operator's right fist firmly against the abdomen in the midline, just above the umbilicus with the palmar aspect of the hand directed caudad. [55] This technique compresses the aorta against the vertebral column and reduces perfusion of the uterus while the source of bleeding is identified and resuscitative measures are performed.

Although rare, occurring in approximately 1 in 4000 to 5000 deliveries, [56] *uterine inversion* is frequently the result of traction on the umbilical cord during the third stage of labor. [1] [52] Other risk factors include primiparity, fundal implantation of the uterus, delivery of a large fetus, the use of oxytocin, and placental anomalies such as placenta accreta.The principal signs and symptoms of the disorder are lancinating and often violent pelvic pain and excessive postpartum hemorrhage with up to 40% of patients developing shock. Diagnosis is made by visualization or palpation of the soft bluish-gray mass of the uterus filling the vaginal vault or protruding through the introitus. On abdominal examination, no mass representing the uterine corpus can be palpated above the symphysis pubis. Treatment is aimed at maintaining cardiovascular stability through the use of IV fluids and immediate repositioning of the uterine corpus. General anesthesia is often necessary, especially if the cervix has contracted. Tocolytic therapy, such as 0.25 mg SQ or IV terbutaline or 2 g IV MgSO$_4$ (administered over 10 minutes), has been used successfully to aid repositioning of the uterus.  Oxytocics should not be given until after the uterus is repositioned. Although timing of removal of an adherent placenta is controversial, removal is generally discouraged before repositioning of the uterus. [57] Early placental removal increases bleeding from friable myometrial vessels and may increase the risk of septic complications.

Repositioning of the uterus may be accomplished by inserting one hand into the vagina and extending the fingers to identify the margins of the cervix; the uterine corpus is

allowed to rest in the palm of the hand. Gentle pressure exerted with the fingers on the edges of the uterus closest to the cervix in the direction of the umbilicus is followed by gradual replacement of the corpus (Figs. 60-27 (Figure Not Available) and 60-28) (Figure Not Available) . Pressure should not initially be exerted centrally on the fundus, because this will cause the uterus to be compressed, forcing more "layers" of the uterus to simultaneously lie within the relatively tight cervical ring. Once the uterus has been repositioned, the placenta may be removed. Because traction on the cord of an adherent placenta is one of the principal causes of uterine inversion, this procedure may be deferred until the obstetrician arrives. Although replacement of the inverted uterus can usually be accomplished by vaginal manipulation, occasionally a dense cervical contraction ring is present, preventing repositioning. General anesthesia and laparotomy may be necessary for uterine repositioning.

## POSTMORTEM CESAREAN SECTION

Postmortem cesarean section, one of oldest and most dramatic surgical procedures known, has long been used as an attempt to preserve the life of the unborn child. [59] Earliest records trace its origin back to 715 B.C. when the legendary king of Rome, Numa Pompilious, decreed that the child be excised from the womb of any woman who died late in pregnancy. Under the emperors of Rome, the *caesars*, this law became known as the "lex caesarea," and hence the name "cesarean operation." [60]

Pliny the Elder described the first successful postmortem cesarean delivery in 237 B.C., that of Scipio Africanus who would later become one of Rome's greatest generals. In A.D. 1280 the Catholic Church at the Council of Cologne decreed that postmortem cesarean section must be performed to permit the unborn child to be baptized and undergo a proper burial. In 1500, a Swiss sow gelder, Jacob Nufer, performed the first reported successful cesarean section with maternal survival, delivering his wife of a live-born infant. His wife lived to bear 6 more children through vaginal delivery. [60]

Since 1879 there have been >188 reports of postmortem cesarean section resulting in the delivery of a live infant. [61] Because most of the literature on postmortem cesarean section involves only small numbers of cases with emphasis on those that are successful, accurate survival statistics are difficult to ascertain. When reported, infant survival rates range from 11 to 40%. [62] Because the potential for survival of a normal infant exists, cesarean delivery must be considered in any woman who suffers irreversible cardiopulmonary arrest during the third trimester of pregnancy. [61] Although the procedure is rare, the decision and its performance may fall to the emergency physician.

### Indications

Survival of the infant is directly related to the maturity of the fetus, the elapsed time from maternal death to delivery, the performance of cardiopulmonary resuscitation (CPR) on the mother, and, in certain circumstances, the availability of neonatal intensive care facilities. [61] Under the best circumstances the probability of infant survival is directly related to the neonatal birth weight and gestational age of the fetus. Although the lower limit of fetal viability varies among institutions, in general, performance of the procedure

before the point of fetal viability at 24 to 25 weeks is not indicated. [7] If the duration of gestation is not known from the history, fetal maturity may be quickly estimated by calculating gestational age on the basis of the date of the patient's last normal menstrual period or by measuring the height of the uterine fundus. Between 18 and 30 weeks' gestation, the age of the fetus in weeks will correspond to the distance in centimeters from the uterine fundus to the symphysis pubis, e.g., at 28 weeks' gestation the fundus is approximately 28 cm above the symphysis pubis or halfway between the umbilicus and the costal margin. [63] Criteria for intervention should be established prospectively at each institution and be in accordance with the institution's general neonatal policies.

The potential for infant survival decreases and the chance of neurologic damage increases as the time from maternal death (cessation of circulation) to cesarean section rises (Table 60-4) (Table Not Available) . When cesarean section is performed within 5 minutes of maternal death, neonatal outcome is generally considered excellent; from 5 to 10 minutes, good; from 10 to 15 minutes, fair; and from 15 to 20 minutes, poor. There have been no reported cases of fetal survival when cesarean delivery has been performed >25 minutes after maternal death.  Because even under optimal conditions CPR results in a cardiac output of 30 to 40% of normal and placental perfusion may be severely compromised, every attempt should be made to begin cesarean delivery within 4 minutes of the cardiopulmonary arrest, completing the procedure within 5 minutes of arrest.  The 5-minute time interval is so crucial to fetal survival that operative delivery within this period has been termed by Katz and colleagues as *perimortem* cesarean delivery. [61] Fetal prognosis is generally better after the sudden death of a previously healthy mother than after the death of a woman with a prolonged and debilitating illness.  Because volume losses incurred with perimortem cesarean section may precipitate maternal cardiac arrest, performance of the procedure in a patient who is hemodynamically unstable but not arrested is not warranted. [7]

CPR should, of course, be initiated immediately on arrest of the mother and continued until after delivery of the infant. The pregnant state produces certain physiologic changes that adversely affect the adequacy of standard CPR. Vena caval occlusion by the gravid uterus hampers venous return and thus compromises maternal cardiac output. A decreased functional residual capacity of the lung may impede ventilation. Manual displacement of the uterus away from the inferior vena cava may be attempted by an assistant. However, perimortem cesarean delivery in itself probably represents the most important variable for a successful maternal resuscitation.

**Legal and Ethical Considerations**

No physician has ever been found liable for performing a postmortem cesarean section. Indeed, it is thought that the physician has the legal right and responsibility to provide the unborn fetus with every possible chance of survival when there is no hope of maternal survival. [61] [63] Permission for the operation should be obtained from the family when possible but not at the expense of delaying the procedure. Failure to obtain permission should not preclude cesarean section.

Nonetheless, there is no standard of care relating to emergency physicians performing a

postmortem cesarean delivery. Each case must be individualized. Limited resources often place the physician in the difficult situation of deciding whether to continue efforts to resuscitate the mother or attempt to deliver the fetus in a difficult situation under less than ideal conditions. In the absence of obstetric backup immediately at hand, it is reasonable for the emergency physician to proceed with delivery of the child if the mother cannot be resuscitated. Prolonged attempts to resuscitate the mother are unlikely to benefit either the mother or the fetus.

## Equipment

The following equipment is needed for a postmortem cesarean section:

Scalpel with a No. 10 blade

Bandage scissors

Bladder retractor

Large retractors (2)

Forceps

Lap or gauze sponges

Hemostats (curved and straight)

Suction

Obstetric pack (as described earlier for vaginal delivery)

Because of the rarity of this procedure in the ED, it is unlikely that a specific instrument pack will be available. Minimally, a scalpel with a large blade and an obstetric pack are necessary.

## Technique

Postmortem cesarean section should be performed by the most experienced person present, preferably an obstetrician, and when possible a neonatologist should be in attendance. CPR should be initiated on the mother at the time of cardiac arrest and continued throughout the procedure. Although it is helpful if fetal heart tones are present premortem, time should not be wasted searching for them nor attempting to evaluate fetal viability with abdominal ultrasonography.

Rapid extraction of the infant while avoiding fetal and maternal injury is the goal of the procedure. Hence, time should not be wasted preparing a sterile operating field or transporting the patient to an operating suite outside the ED. Using a large (e.g., No. 10) scalpel, a midline vertical incision is made through the abdominal wall extending from the symphysis pubis to the umbilicus and carried through all abdominal layers to the

peritoneal cavity (Fig. 60-29) (Figure Not Available) . In most gravid women the hyperpigmented "linea nigra" is apparent and may serve as a guide for the incision. If available, retractors are placed in the abdominal wound and drawn laterally to expose the anterior surface of the uterus. The bladder is reflected inferiorly; if full it may be aspirated to evacuate it and permit better access to the uterus (Fig. 60-30) . While avoiding injury to fetal parts, a small, approximately 5-cm, vertical incision is made through the lower uterine segment until amniotic fluid is obtained or until the uterine cavity is clearly entered (Fig. 60-31 *A*). The index and long fingers are then inserted into the incision and used to lift the uterine wall away from the fetus. A bandage scissors is used to extend the incision vertically to the fundus until a wide exposure is obtained (see Fig. 60-31 *B*). The infant is then gently delivered, the mouth and nares suctioned, and the cord clamped and cut (Fig. 60-32) . Because the incision is relatively high in the uterus, the infant's head may not be readily accessible to the physician, in which case the infant's feet are grasped and the infant delivered through maneuvers similar to those of a breech delivery. Neonatal resuscitation should be carried out as necessary.

Because in rare instances relief of vena caval compression by the uterus improves maternal hemodynamics such that maternal survival is possible, maternal pulses should be checked and CPR continued after delivery of the infant.

### Conclusion

Postmortem cesarean section is a rarely performed but potentially life-saving procedure that should be considered in any woman in the third trimester of pregnancy who suffers irreversible cardiopulmonary arrest. Because neonatal survival is enhanced as the time from maternal death to delivery decreases while the irreversible nature of maternal cardiac arrest becomes more apparent as resuscitative efforts progress, the decision and timing of this procedure may be one of the most difficult that the emergency physician makes. Once the decision is made to perform a cesarean section, it should be done as quickly as possible by the most experienced person present. CPR should be continued until after delivery of the infant. Although rare, maternal survival has been reported after "postmortem" cesarean section.

## THE NEONATE

Evaluation of the neonate begins before delivery with assessment of maternal well-being, gestational age, ease and type of previous deliveries, and the recognition of fetal distress, as evidenced by meconium staining of the amniotic fluid, fetal bradycardia, or evidence of cord prolapse. Care of the newborn begins with delivery of the head while the mouth and nares are suctioned. After delivery and cutting of the umbilical cord, the infant should be placed in a supine and slight Trendelenburg position with the neck slightly extended (sniffing position). A 2.5-cm roll placed under the infant's shoulders may help in maintaining head position. Because of the relatively large surface area of the infant, temperature drops rapidly immediately after birth, with subsequent chilling, which produces shivering and increased $O_2$ demand. Care must be taken, therefore, to maintain body temperature by drying the infant with subsequent placement under a radiant warmer or, if a warmer is unavailable, in warm blankets.

**Evaluation**

Traditionally, the *Apgar scoring system*, applied at 1 and 5 minutes after birth, is the standard of neonatal evaluation (Table 60-5) . [1] [66] In general, the higher the score, the better the condition of the infant. The 1-minute Apgar score reflects the need for immediate resuscitation. A score of 7 to 10 indicates that the infant is in excellent condition, requiring no aid other than nasopharyngeal suctioning. A moderately depressed infant with depressed respirations, flaccidity, and pallor or cyanosis usually scores 4 to 6. A score of 0 to 3 indicates a severely depressed infant and mandates immediate resuscitation. Heart rate is slow and reflexes are depressed or absent. [1]

Although the Apgar score is the traditional standard for neonatal evaluation and low scores at 1 and 5 minutes are excellent indicators of the need for resuscitation, the decision to resuscitate and the infant's response to resuscitation can be more accurately assessed by evaluating the heart rate, respiratory effort, and color. [27]

Normally, the newborn takes a first breath within a few seconds of birth and cries within 30 seconds. [1] Apnea is the most common initial manifestation of depression in the neonate and unless reversed may lead to progressive hypoxemia, hypercarbia, and acidosis. Hypoventilation quickly leads to cardiovascular depression with subsequent slowing of the heart rate, the most sensitive indication of infant distress. A heart rate of <100 beats/min generally indicates the need for respiratory support.

**Equipment for Infant Stabilization**

The following equipment is required for infant stabilization:

Bulb syringe or DeLee suction trap

Oral airways (size 0, 00, and 000)

Suction catheters (size 5, 6, 8, and 10 Fr)

Endotracheal tubes (size 2.5 mm, 3.0 mm, and 3.5 mm)

Endotracheal tube stylet (optional)

Laryngoscope

Laryngoscope blade (straight; sizes 0 and 1)

Resuscitation bag

Face masks (sizes 0 and 1)

Wall suction

Wall oxygen

Radiant warmer

## Stabilization Technique

If neonatal respirations are infrequent or absent, suctioning the mouth and pharynx with a bulb syringe or DeLee suction trap and stimulating the infant by lightly slapping the soles of the feet and rubbing the back may serve to stimulate breathing. If necessary, the airway should be gently suctioned with an 8 or 10 Fr suction catheter. If wall suction is used, the pressure should be set at 60 to 80 mm Hg to prevent pharyngeal and esophageal trauma. Because deep suctioning of the oropharynx may produce a vagal response causing bradycardia or apnea, suctioning should continue for no longer than 10 seconds at a time, and heart rate should be continuously monitored. To minimize hypoxia, sufficient time should be allowed between suctioning for the lungs to be ventilated with 100% $O_2$ . [66] Because the neonate is an obligate nose breather, it is advisable to suction once through each nostril to ensure patency of the upper airway. Failure to establish effective respirations indicates either marked central nervous system depression, mechanical obstruction, or an intrinsic lung abnormality and demands active resuscitation.

If signs of airway obstruction, that is, decreased chest expansion and sternal and intercostal retractions, are present and persist after suctioning, the larynx should be directly visualized with a laryngoscope. Suctioning of the larynx should be done under direct vision. For obstruction distal to the glottic opening, it is advisable to intubate the infant and suction through the endotracheal tube. Appropriate size of the endotracheal tube varies with the size of the neonate from 2.5 mm for those weighing 1000 g or less to 3.5 mm for the term infant.

Because *meconium aspiration* is a major cause of neonatal morbidity and mortality, its prevention deserves special mention. Up to 60% of neonates with meconium staining of the amniotic fluid aspirate; approximately 20% of these neonates later develop pulmonary complications. [66] To prevent aspiration, neonates born with particulate meconium staining of the amniotic fluid require thorough suctioning of the hypopharynx before the initiation of respiration, that is, immediately after delivery of the head and before delivery of the thorax. If suctioning is inadequate and the hypopharynx has not been cleared of meconium before the onset of respiration, tracheal intubation should be performed to remove as much of the meconium as possible from the lower airway. Because

| TABLE 60-5 -- Apgar Scoring System | | | |
|---|---|---|---|
| **Sign** | **0** | **1** | **2** |

| | | | |
|---|---|---|---|
| Heart rate | Absent | Slow (<100) | >100 |
| Respiratory effort | Absent | Slow, irregular | Good, crying |
| Muscle tone | Flaccid | Some flexion of extremities | Active motion |
| Reflex irritability | No response | Grimace | Vigorous cry |
| Color | Blue, pale | Body pink, extremities blue | Completely pink |

meconium is viscous, suction should be applied directly to the endotracheal tube using the full caliber of the tube to evacuate the thick, tenacious material. The presence of watery or thin meconium does not require routine endotracheal intubation. [66] [67]

Bag and mask ventilation should be initiated as soon as it is recognized that tactile stimulation is not sufficient to establish spontaneous ventilation, that ventilation is not adequate to maintain a heart rate of >100 beats/min, or when central cyanosis persists despite a maximal oxygen environment.

| TABLE 60-6 -- Medications Commonly Used in Neonatal Resuscitations | | | |
|---|---|---|---|
| **Drug** | **Dose** | **Indications** | **Comments** |
| Epinephrine * | 0.01-0.03 mg/kg (0.1 mL/kg of 1:10,000 solution) | Bradycardia, asystole, EMD | May be repeated every 5 minutes as required. |

| Sodium bicarbonate | 1-2 mEq/kg | Correction of acidosis persisting after adequate ventilation | (1) Should not be used for brief episodes of arrest or to correct respiratory acidosis; |
| | | | (2) Because rapid infusion of hypertonic solutions may predispose to intracranial hemorrhage, each dose should be infused over 5-10 minutes in concentrations not to exceed 0.5 mEq/mL. |
| Atropine * | 0.02 mg/kg | Bradycardia, 2nd- and 3rd- degree atrioventricular block, perhaps asystole unresponsive to epinephrine | (1) Minimum initial dose is 0.1 mg; |
| | | | (2) May repeat mg/kg dose every 2-5 minutes to a total dose of 0.1 mg/kg; |
| | | | (3) Not recommended by current ACLS guidelines for acute phase of neonatal resuscitation. |
| Dextrose | 0.5-1.0 g/kg | Serum glucose < 40 mg/dL | (1) Monitor serum glucose (Dextrostix) frequently; |
| | | | (2) Administer as dilute solution, e.g., 10% dextrose solution; |
| | | | (3) Follow initial bolus with continuous infusion of 5-8 mg/kg/min. |

| | | | |
|---|---|---|---|
| Naloxone [*] | 0.01 mg/kg IV, IM, or SQ | Narcotic reversal as indicated | Duration of narcotic may be greater than that of naloxone; subsequent monitoring of infant required. |
| Calcium chloride | 5-7 mg/kg | Controversial, hypocalcemia and possibly for compromised myocardial dysfunction or cardiac arrest | Not recommended by current ACLS guidelines for acute phase of neonatal resuscitation. |

**Cardiovascular Drips**

| | | | |
|---|---|---|---|
| Epinephrine | 0.1-1.0 mug/kg/min | alpha-Adrenergic agent required to maintain systemic arterial pressure | Myocardial stimulation may cause pathologic arrhythmias; excessive dose may impair renal, splanchnic, or peripheral perfusion. |
| Isoproterenol | 0.1-1.0 mug/kg/min titrated to desired heart rate | Bradycardia, 2nd- and 3rd- degree atrioventricular block, asystole | May cause hypotension due to beta2 -adrenergic stimulation. |

**Other**

| | | | |
|---|---|---|---|
| Defibrillation | 1-2 J/kg | Ventricular fibrillation, ventricular tachycardia | Rarely indicated or effective. |

| | | | |
|---|---|---|---|
| Fluid (normal saline, lactated Ringer's solution) | 10-20 mL/kg | Hypovolemia | Rarely indicated. Hypovolemia most commonly occurs with placenta previa, abruption, twin-twin or fetomaternal transfusion. May require O-negative blood crossmatched with mother's blood or 5% albumin solution or other plasma substitute. |

ACLS, advanced cardiac life support; EMD, electrical mechanical dissociation.

\* May also be given via endotracheal tube.

A pulse oximeter strapped to the infant's Achilles tendon or thumb may provide a useful adjunct to assess neonatal well-being and the adequacy of resuscitation. [27] Intubation is indicated if prolonged positive-pressure ventilation is necessary, for the suctioning of thick meconium, or if bag-valve-mask ventilation is ineffective. [27] Perlman and Risser emphasize that optimization of ventilation is essential before administering chest compressions or medications. [68]

The heart rate should be monitored during the course of neonatal evaluation and stabilization with either direct auscultation over the chest or palpation of the pulse at the base of the umbilical cord or in the brachial or femoral areas. A readily discernible heartbeat of 100 beats/min is acceptable. If the heart rate is <60 beats/min or is between 60 and 80 beats/min and does not respond rapidly to effective ventilation and oxygenation, chest compression should be instituted while ventilation is continued. There are 2 techniques for performing chest compression in the neonate. In the two-handed method of chest compression the hands encircle the chest with the fingers placed over the back, and the thumbs are placed over the lower third of the sternum just below an imaginary line joining the 2 nipples. Because of the potential for injuring abdominal organs, the xiphoid portion of the sternum should not be compressed. If the infant is large or the resuscitator's hands are too small to encircle the chest, 2 finger compressions using the ring and middle fingers placed on the sternum one finger's breadth below the nipple line may be used. The sternum is compressed 1.2 to 2.0 cm at a rate of 120 compressions/min. Positive-pressure ventilation should be continued with 100% O2 at a rate of 40 to 60/min. Myocardial dysfunction and shock in the neonatal period are most commonly due to profound hypoxia. If these conditions persist despite adequate ventilation, an umbilical, IV, or interosseous line should be established with appropriate drug therapy. The medications most commonly used in neonatal resuscitation are found in Table 60-6 .

# Chapter 61 - Culdocentesis

**G. Richard Braen**

A number of conditions require sampling of the intraperitoneal fluid to confirm a diagnosis or to obtain material for microbial culturing. This fluid can be obtained from the peritoneal cavity in a number of ways. Culdocentesis involves the introduction of a hollow needle through the vaginal wall into the peritoneal space. Culdocentesis is a simple, rapid, and safe procedure. The technique has several indications, but it is used primarily for diagnosing ruptured ectopic pregnancies and ruptured ovarian cysts and for obtaining fluid to aid in the culture diagnosis of pelvic inflammatory disease (PID).

## ANATOMY

Before culdocentesis is attempted, the clinician must be familiar with the anatomy of the vaginal and rectouterine pouch (pouch of Douglas). In the adult female, the vagina is approximately 9 cm long. From its inferior to its superior aspect, the posterior wall of the vagina is related to the anal canal by way of the perineal body, the rectum, and the peritoneum of the rectouterine pouch. [1] The rectouterine pouch and the posterior wall of the vagina are adjacent only at the upper quarter (approximately 2 cm) of the posterior vaginal wall. The vaginal wall in this area is <5 mm thick. The uterus lies nearly at a right angle to the vagina.

The blood supply of the upper vagina comes from the uterine and vaginal arteries, which are branches of the internal iliac artery. The area is drained by a vaginal venous plexus that communicates with the uterine and vesical plexuses. The vagina has its greatest sensation near the introitus and little sensation in the area adjacent to the rectouterine pouch.

The rectouterine pouch is formed by reflections of the peritoneum, and it is the most dependent intraperitoneal space in both the upright and the supine positions. Blood, pus, and other free fluids in the peritoneal cavity pool in the pouch because of its dependent location. This pouch separates the upper portion of the rectum from the uterus and the upper part of the vagina. The pouch often contains small intestine and normally a small amount of peritoneal fluid.

## INDICATIONS

Culdocentesis is indicated in any adult female when fluid aspirated from the rectouterine pouch will help to confirm a clinical diagnosis. Ultrasound examination has largely replaced culdocentesis in the diagnosis of ruptured ectopic pregnancy and ruptured ovarian cysts, but the procedure still has an important role in emergency medicine. If ultrasound examination is not readily available in the emergency department (ED), or if the patient is too hemodynamically unstable to be transported to an off-site location for ultrasound, culdocentesis may be the fastest and most accurate diagnostic technique available to the emergency physician. [2] Analysis of peritoneal fluid is also a reliable

method of differentiating inflammatory from hemorrhagic pelvic pathologic conditions. Conditions in which culdocentesis may be of diagnostic value include a ruptured viscus (particularly an ectopic pregnancy or a corpus luteum cyst), PID, and other intra-abdominal infections (particularly appendicitis with rupture or diverticulitis with perforation), intra-abdominal injuries to the liver or the spleen, and ruptured aortic aneurysms. [3]

**Ectopic Pregnancy**

Ectopic pregnancy is often one of the most difficult gynecologic lesions to diagnose. [4] The incidence of ectopic pregnancy is on the rise, accounting for 1.6% of all pregnancies. Ectopic pregnancy is the most common obstetrical cause of maternal death in the first trimester. [4] In a series of 300 consecutive cases of ectopic pregnancy, 50% of patients received medical consultation at least 2 times before the correct diagnosis was made. [5] In 11% of the patients in this series, the diagnosis was not made until the third medical visit. Because of the severe consequences of a ruptured ectopic pregnancy, an early, accurate diagnosis is desired.

The clinical picture of ectopic pregnancy may include vascular collapse, pelvic pain, amenorrhea or abnormal menses, shoulder pain, syncope, cervical or adnexal tenderness, adnexal mass, anemia, and leukocytosis. There is often a history of salpingitis, use of an intrauterine contraceptive device, or tubal ligation; however, no combination of these signs, symptoms, or historical data is diagnostic for an ectopic pregnancy. To confuse the diagnosis further, a normal menstrual history is reported in approximately 50% of patients with ectopic pregnancy and the *urine* pregnancy test is occasionally negative. [6] Although rarely seen, the combination of a uterine decidual cast (Fig. 61-1) and a positive pregnancy test are virtually pathognomonic of an ectopic pregnancy. A uterine cast is decidua that has been hormonally stimulated by the ectopic pregnancy but is passed vaginally when the tissue can no longer be supported. The cast is an outline of the uterine cavity, but it can be mistaken for products of conception if not inspected carefully. Therefore, all tissue passed vaginally should be carefully inspected before it is sent to the laboratory for analysis for products of conception.

The greater sensitivity of the serum and urine human chorionic gonadotropin beta subunit (beta-hCG) radioreceptor assay, coupled with the proliferation of transvaginal ultrasound and laparoscopy, have greatly increased the chances for early diagnosis of unruptured and ruptured ectopic pregnancy. [7] A sensitive and specific beta-hCG test must be used. The newer urinary beta-hCG tests (enzyme-linked or solid-phase immunoassay) provide sensitivity to 20 to 50 mIU/ mL. Quantification of the serum test adds additional information being sensitive to >5 mIU/mL. Therefore, a negative *urine* beta-hCG test rules out pregnancy in >98% of cases, and pregnancy in any site can be ruled out in virtually all patients with a negative *serum* beta-hCG test. [8] A single quantitative beta-hCG is a poor predictor of size of the pregnancy or the risk of ectopic pregnancy, but serial testing is quite helpful. It is expected that the quantitative serum beta-hCG level should double approximately every 2 days in the first trimester. Serum progesterone determinations may also help to identify a normal or abnormal pregnancy. Although not infallible, a serum progesterone level of <10 ng/mL is usually associated with a nonviable intrauterine pregnancy or ectopic pregnancy, and a level >25 ng/mL is

usually associated with a viable intrauterine pregnancy.

To increase the accuracy of diagnosis, it is helpful to combine quantitative beta-hCG testing with ultrasound examination. An empty uterus by transvaginal or abdominal ultrasound combined with certain quantitative serum beta-hCG results can be quite helpful to the clinician. The quantitative range in which the ultrasonographer should detect an intrauterine pregnancy varies, but an intrauterine pregnancy should be detected if the serum beta-hCG level is in the range of 1200 to 1500 mIU/mL when using a transvaginal probe, and if the level is >6500 mIU/mL when using a transabdominal probe. It is important to note that the absence of an intrauterine pregnancy by ultrasound when the beta-hCG level is below the discriminatory zone is nondiagnostic and could represent an early viable normal pregnancy, a nonviable intrauterine pregnancy, or an ectopic pregnancy. When no intrauterine pregnancy is detected by ultrasound and the serum beta-hCG exceeds the discriminatory zone, the chance of an ectopic pregnancy ranges from 86 to 100%. [9]

Culdocentesis, for some patients, may play an important role in the diagnosis of ectopic pregnancy. The test has an accuracy rate of 85 to 95%. Romero et al. [12] reported that an ectopic pregnancy was found in 99% of cases when a positive pregnancy test and a positive culdocentesis were present. Although culdocentesis is most often positive in the presence of a frankly ruptured ectopic pregnancy, it may be diagnostic even in the nonruptured case when bleeding has been slow or intermittent. It is useful to note that many ectopic pregnancies leak varying amounts of blood for days or weeks before rupture. It is important that hemoperitoneum has been noted to occur in 45 to 60% of cases of *unruptured* ectopic pregnancy proven at surgery.

Hence, culdocentesis may be helpful in the stable patient whose ultrasound examination does not demonstrate an intrauterine pregnancy despite a quantitative serum beta-hCG level in the appropriate range. Although some clinicians opt for outpatient monitoring of serial beta-hCG levels in this setting, patients for whom the clinician has a high suspicion for ectopic pregnancy (e.g., the patient who has or has had significant discomfort) or for whom close follow-up cannot be ensured are candidates for culdocentesis. [2] Although a negative culdocentesis does not rule out an early ectopic pregnancy, patients with a nondiagnostic ultrasound and a negative culdocentesis generally represent patients at lower risk for "rupture" of an ectopic pregnancy during outpatient serum beta-hCG monitoring. Patients with a nondiagnostic ultrasound examination and a serum beta-hCG level below the threshold at which an intrauterine pregnancy should be visibile on the ultrasound examination also must be individualized. Those patients with significant pain, an unexplained low hematocrit reading, or postural vital sign changes (or near syncope) represent candidates for culdocentesis.

### Pelvic Infection

Acute PID has a polymicrobial etiology. [15] [16] [17] It has been common practice to define the etiology of PID by isolation of pathogens from the endocervix. The etiology is probably better defined by examination of the tubal flora. In fact, there is little correlation between cul-de-sac cultures and cervical cultures in PID. [18] Some medical centers routinely use aspirates obtained through culdocentesis to aid in determining the

causative microbial agents for the PID. [19] As the microbiology of PID becomes more complex and as the causative organisms develop resistance to antimicrobial agents, culdocentesis may evolve as one of the prime methods of obtaining meaningful microbiological cultures that will dictate appropriate therapy.

**Blunt Abdominal Trauma**

Although diagnostic peritoneal lavage, computed tomographic (CT) scanning, and ultrasound examination are popular and valuable techniques to detect hemoperitoneum, the use of culdocentesis also has been advocated. [3] [20] Because small amounts of blood tend to collect in the rectouterine pouch, the aspiration of clear peritoneal fluid is of great potential value in *excluding* hemoperitoneum. The procedure may be more advantageous than peritoneal lavage in some instances, because there is less risk of urinary bladder perforation or bowel injury. In addition, previous abdominal surgery is not a relative contraindication to culdocentesis, as it is with peritoneal lavage.

## CONTRAINDICATIONS

The contraindications to culdocentesis are relatively few and include an uncooperative patient, a pelvic mass detected on bimanual pelvic examination, a nonmobile retroverted uterus, and coagulopathies. Pelvic masses may include tubo-ovarian abscesses, appendiceal abscesses, ovarian masses, and pelvic kidneys. It has been suggested that the only major risk with the procedure is that of rupturing an unsuspected tubo-ovarian abscess into the peritoneal cavity. This can be avoided by careful bimanual pelvic examination to exclude patients with large masses in the cul-de-sac. [18] Although there are no data to guide the age at which culdocentesis may be safely performed, the procedure is generally limited to patients who are beyond puberty. This limitation is suggested on the basis of anatomy and with the consideration that the procedure is difficult to perform through a small, prepubertal vagina.

One should not forgo culdocentesis due to the absence of classic signs anticipated with ectopic pregnancy. It is important to note that a positive culdocentesis does not consistently correlate with peritoneal irritation, blood pressure, pulse rate, or the actual volume of hemoperitoneum. [13] In fact, bradycardia in the presence of significant intraperitoneal bleeding from a ruptured ectopic pregnancy is not unusual (Tables 61-1 (Table Not Available) and 61-2) (Table Not Available).

## EQUIPMENT

The equipment required for culdocentesis is listed in Table 61-3 . Either an 18-ga spinal needle or a 19-ga butterfly needle held by ring forceps is acceptable. It may be helpful to anesthetize the posterior vaginal wall at the site of the puncture with 1 to 2% lidocaine with epinephrine administered through a 27- or 25-ga needle. Some physicians use a cocaine-soaked cotton ball to anesthetize the mucosa before infiltration with a local anesthetic. Although local anesthesia is often unnecessary (because *puncture of the posterior vaginal wall at the upper one fourth of the vagina is generally no more painful than a venipuncture*), there is some advantage to use of a local anesthetic if multiple

attempts at culdocentesis are required, as is sometimes the case. In addition, the epinephrine may produce vasoconstriction and may reduce bleeding associated with the needle puncture. Furthermore, culdocentesis is often stressful to the patient, and all attempts should be made to render the procedure as painless as possible. Consideration of parenteral analgesia and sedation (see Chapter 35) should also be made when the patient is uncomfortable or anxious.

# TECHNIQUE

## Preparation

Culdocentesis is an invasive procedure that, in some hospitals, requires a written, witnessed, and signed consent form from the patient, parent, or guardian when the patient's condition permits. If verbal consent is obtained, this action should be witnessed and a notation in the medical record made documenting that the procedure was described, complications were discussed, and any alternatives--where appropriate--(e.g., sonography or immediate laparoscopy) were offered.

Once written or verbal consent is obtained, the patient is placed in a lithotomy position with the head of the table slightly elevated (reverse Trendelenburg position) so that intraperitoneal fluid gravitates into the rectouterine pouch. The patient's feet are placed in stirrups. In selected patients, some physicians premedicate with intravenous (IV) opioids or sedatives. The administration of nitrous oxide analgesia also is an accepted practice. When nitrous oxide is used during this procedure, it is important to document the presence of a chaperone, as some patients may develop sexual delusions under this agent. Although pain associated with culdocentesis needle passage is generally minor, the judicious use of analgesia and sedation makes the procedure easier for both physician and patient.

Radiographs, when indicated in the *stable* patient, are taken *prior* to culdocentesis, to avoid possible confusion if a pneumoperitoneum is detected following the procedure.

## Exposure

A bimanual pelvic examination must be performed before culdocentesis to rule out a fixed pelvic mass and to assess the position of the uterus. The examiner then inserts the bivalve vaginal speculum and opens it widely by adjusting both the height and the angle thumbscrews. The *posterior* lip of the cervix is grasped with the toothed uterine cervical tenaculum, and the cervix is elevated (Fig. 61-2) (Figure Not Available) . Grasping the cervix may produce minor discomfort, and the patient

---

**TABLE 61-3** -- Equipment for Culdocentesis

| |
|---|
| Adjustable examination table with stirrups |
| Bivalve vaginal speculum |
| Uterine cervical tenaculum |
| 19-ga butterfly needle or 18-ga spinal needle |
| 27- or 25-ga needle (for local anesthetic infiltration) |
| Ring sponge forceps |
| Syringes (20 mL) |
| Surgical preparation (iodinated, such as povidone-iodine) |
| Sterile water, cotton balls, 4 × 4 gauze sponges |
| Cocaine (10% solution) or benzocaine (20% solution) |
| Lidocaine (1%) with epinephrine |
| Culture media or test tube without anticoagulant |

should be warned in advance of a possible sharp pain during this part of the examination. Also, bleeding from the tenaculum puncture site and/or culdocentesis site

may produce postprocedure spotting, and the patient should similarly be informed.

Use of the tenaculum elevates a retroverted uterus from the pouch, exposes the puncture site, and stabilizes the posterior wall during the needle puncture. Some physicians prefer to use longitudinal traction on the cervix to produce the same result. The vaginal wall adjacent to the rectouterine pouch will be tightened somewhat between the inferior blade of the bivalve speculum and the elevated posterior lip of the cervix. This tightening of the vaginal wall exposes the puncture site and keeps it from moving away from the needle when the wall is punctured.

After the tenaculum is applied and the posterior lip of the cervix is elevated or traction is applied, the vaginal wall in the area of the rectouterine pouch should be swabbed with surgical preparation followed by a small amount of sterile water. Local anesthesia (1% lidocaine with epinephrine) may be administered at this point. Anesthesia may be injected with a separate 27- or 25-ga needle or by the spinal needle to be used for the culdocentesis. Also, a cotton ball soaked in cocaine or benzocaine solution can be used for topical anesthesia of the posterior vaginal wall before infiltration with a local anesthetic. The needles used for both the local anesthetic and the puncture should be attached to a 20-mL syringe. A smaller syringe may not be long enough to allow adequate control of the needle, and the physician's hand may block the view of the puncture site if a smaller syringe is used.

**Aspiration**

Following local anesthesia, the syringe and the spinal needle to be used for the culdocentesis are advanced parallel to the lower blade of the speculum. It is helpful to fill the syringe with 2 to 3 mL of saline (nonbacteriostatic) before puncture. Following needle puncture, the free flow of the fluid from the syringe expels tissue that may have clogged the needle and confirms that the needle tip is in the proper position and is not lodged in the uterine wall or the intestinal wall. Saline (rather than air) is preferred, because if air is used, one must be careful in interpreting the presence of free peritoneal air on subsequent radiographs. To avoid the need to change the syringe during the procedure, 1% lidocaine may be used for both anesthesia and confirmation of proper needle placement, but the bacteriostatic property of this agent precludes its use if the procedure is performed to obtain fluid for culture.

The vaginal wall should be penetrated in the midline 1 to 1.5 cm posteriorly (inferiorly) to the point at which the vaginal wall joins the cervix (Fig. 61-3) (Figure Not Available) . [22] The needle should penetrate a total of 2 to 2.5 cm. [22] [23] Gentle suction is then applied with the syringe while the needle is slowly withdrawn. It is important for the physician to avoid aspirating any blood that has accumulated in the vagina from previous needle punctures or from cervical bleeding, because this may give the false impression of a positive tap. Bleeding from the puncture site in the vaginal wall is minimized if epinephrine is added to the local anesthetic.

Blood or fluid may be obtained immediately but may also be obtained just before the needle is withdrawn from the peritoneal cavity. Therefore, it is important to aspirate throughout the gradual withdrawal procedure. If no fluid is aspirated, the needle should

be reintroduced and directed only slightly to the left or right of the midline. Directing the needle too far laterally may result in puncture of mesenteric or pelvic vessels. It is important to note that *if no fluid is obtained on the first attempt, the procedure should be repeated.*

Some physicians prefer the use of a 19-ga butterfly needle held with a ring forceps (Fig. 61-4) (Figure Not Available) . [22] This technique offers a built-in guide to needle depth and allows for good control of the needle during puncture. An assistant must aspirate the tubing while the physician controls positioning and withdrawal of the needle.

Fluid that is aspirated may be old, nonclotting blood; bright red blood; pus; exudate; or a straw-colored serous liquid. Any fluid that is not blood should be submitted for Gram staining, aerobic and anaerobic culture, and cell counts. Blood should be observed for clotting. Blood should also be sent for a hematocrit determination.

## INTERPRETATION OF RESULTS

An interpretation of the results of culdocentesis depends primarily on whether any fluid was obtained. In the absence of a pathologic condition, one will often aspirate 2 to 3 mL of clear, yellowish peritoneal fluid. When there is no return of fluid of any type (a so-called dry tap), the procedure has *no diagnostic value.* Because a dry tap is nondiagnostic, it should not be equated with normal peritoneal fluid. In addition, when <2 mL of clotting blood is obtained, this is also considered to be a nondiagnostic tap, because the source of this small amount of blood may be the puncture site on the vaginal wall. Such blood will usually clot. Over 2 mL of *nonclotting blood* is certainly suggestive of hemoperitoneum. However, some researchers interpret as little as 0.3 mL of *nonclotting* blood as a positive tap. [13] There is no particular significance to larger amounts of blood, because absolute volume may be related to the needle position or the rate of bleeding. Brenner and coworkers reported no blood from culdocentesis in 5% of patients with proven ectopic pregnancies, even when rupture had occurred. [5] In the series of 61 patients with surgically proven ectopic pregnancy reported by Cartwright and colleagues, culdocentesis performed within 4 hours of surgery was positive in 70%, negative in 10%, and inadequate in 20%. [13] "Positive" in their series was defined as obtaining at least 0.3 mL of nonclotting blood with a hematocrit of >3%. "Negative" was defined as obtaining 0.3 mL of fluid with a hematocrit of <3%. An "inadequate" tap was one in which no fluid was obtained. In the 252 surgically proven ectopic pregnancy patients having culdocentesis reported by Vermesh and others, 83% had a positive tap. [14] They defined a positive tap as nonclotting blood with a hematocrit >15%.

Because culdocentesis is usually used to diagnose an ectopic pregnancy, a "negative tap" is one that yields pus or clear, straw-colored peritoneal or cystic fluid. A large amount of clear fluid (>10 mL) indicates a probable ruptured ovarian cyst, aspiration of an intact corpus luteum cyst, ascites, or possibly carcinoma. The significance of these fluids and the interpretation of results are outlined in Tables 61-4 and 61-5 (Table Not Available) . Elliot and associates [24] caution that obtaining >10 mL of clear fluid should not automatically rule out an ectopic pregnancy, because the latter may coexist with other pathologic conditions.

A "positive tap" is one in which nonclotting blood is obtained, although the presence of nonclotted blood does not assure a tubal pregnancy. Intraperitoneal blood from any source (ectopic pregnancy, ovarian cyst, ruptured spleen) may remain unclotted after aspiration for days in the syringe as a result of the defibrination activity of the peritoneum. The return of a serosanguineous fluid also suggests a ruptured ovarian cyst. The hematocrit of blood from active intraperitoneal bleeding is >10%. In one series, the hematocrit of blood from a ruptured ectopic pregnancy was 15% in 97% of cases. [5]

It should be emphasized that a positive culdocentesis in the presence of a positive pregnancy test does not always prove an ectopic pregnancy. [14] A ruptured corpus luteum cyst in the presence of an intrauterine pregnancy test is probably the most common cause of a "false-positive" scenario. When possible, ultrasound may help corroborate the culdocentesis findings.

## COMPLICATIONS

Culdocentesis is one of the safest procedures performed in the emergency setting, and there are probably fewer complications with this technique than with peripheral venous cannulation. Complications have been reported, however, the most serious being rupture of an unsuspected tubo-ovarian abscess. [22] Other complications include perforation of the bowel, perforation of a pelvic kidney, and bleeding from the puncture site in patients with clotting disorders. Because the most common complications result from the puncture of a pelvic mass, careful bimanual examination of the patient should help prevent this problem. Puncture of the bowel and the uterine wall occurs relatively frequently, but this occurrence does not generally result in serious morbidity. Obviously, penetration of the gravid uterus has greater potential for harm. Occasionally, one will aspirate air or fecal matter, confirming inadvertent puncture of the rectum. Although this may be disconcerting, it is seldom of serious clinical concern and requires no immediate change in therapy.

| TABLE 61-4 -- Interpretation of Culdocentesis Fluid | |
|---|---|
| **Aspirated Fluid** | **Condition and Suggested Differential Diagnosis** |
| Clear, serous, straw-colored (usually only a few milliliters) | Normal peritoneal fluid |

| | |
|---|---|
| Large amount of clear fluid | Ruptured or large ovarian cyst (fluid may be serosanguineous); pregnancy may be coexistent |
| | Ascites |
| | Carcinoma |
| Exudate with polymorphonuclear leukocytes | Pelvic inflammatory disease |
| | Gonococcal salpingitis |
| | Chronic salpingitis |
| Purulent fluid | Bacterial infection |
| | Tubo-ovarian abscess with rupture |
| | Appendicitis with rupture |
| | Diverticulitis with perforation |

| | |
|---|---|
| Bright red blood * | Ruputured viscus or vascular injury |
| | Recently bleeding ectopic pregnancy * (ruptured or unruptured) |
| | Bleeding corpus luteum |
| | Intra-abdominal injury |
| | Liver |
| | Spleen |
| | Other organs |
| | Ruptured aortic aneurysm |
| Old, brown, nonclotting blood | Ruptured viscus |
| | Ectopic pregnancy with intraperitoneal bleeding over a few days or weeks |
| | Old (days) intra-abdominal injury (e.g., delayed splenic rupture) |

* *Note:* The hematocrit of blood from a ruptured ectopic pregnancy is usually >15% (97.5% of cases), [5]but some authors use >3% as positive.

## CONCLUSION

In the emergency setting, culdocentesis can be a helpful diagnostic procedure. In an unstable patient with a strong clinical scenario for a ruptured ectopic pregnancy, the procedure may facilitate life-saving surgery by confirming the diagnosis without the need for time-consuming ultrasound examinations. Although the procedure is mainly used in the evaluation of ectopic pregnancy, culdocentesis should also be considered as a diagnostic aid in the evaluation of PID and in abdominal trauma. This is a safe, simple procedure that every physician who deals with the emergency evaluation of women, *particularly women of childbearing age*, should know and use. It may be the most rapid way to confirm hemoperitoneum in the unstable patient with suspected ectopic pregnancy.

# Chapter 62 - Examination of the Sexual Assault Victim

**G. Richard Brae** n

Rape is reported to be the fastest-growing violent crime in the United States, [1] and it has been estimated that as many as 44% of women will be victims of a rape or an attempted rape in their lifetime, and that >50% of these women will be victims more than once. [2] The incidence of rape peaks between the ages of 16 to 19 years, but >60,000 rapes of women older than 50 years are reported annually. [3] The victims of rape can experience extensive immediate and delayed trauma (both physical and psychological), can be exposed to disease, and can become pregnant.

Society has held many misconceptions about the victims of sexual assault, particularly female victims. Included in these misconceptions are beliefs that the assault was encouraged by behavior or dress, that resistance by the victim was not sufficient, that the victim was promiscuous, or that there are ulterior motives for pressing charges. Variations on classic rape include the area of "date rape," in which *some sexual activity other than coitus* was voluntary, and spousal rape, defined as forced sexual activity between a married couple without consent by one party. [4] "Statutory rape" defines any form of coitus as criminal based solely on the young age of the victim, who is not deemed capable of consent. Many women do not report rape for fear of retaliation or embarrassment or simply because they do not know their legal rights.

Physicians and nurses dealing with an alleged sexual assault victim have a professional, ethical, and moral responsibility to provide the best medical and psychological care possible while simultaneously collecting and preserving the proper medicolegal evidence that is unique to the evaluation of alleged sexual assault cases.

Ideally, rape victims should be evaluated in a designated rape evaluation center, usually a designated hospital setting (often a designated emergency department [ED]) staffed with informed physicians, dedicated social workers, and trained counselors. However, victims may be brought to an ED that does not routinely provide this service. This may happen for several reasons, but it is often a matter of logistics--the peak hours for this crime are from 8:00 P.M. to 2:00 A.M., [5] and the most common days on which it occurs are weekends. In many localities, the only available physician at these times is the emergency physician, and it is often his or her responsibility to perform the evaluation and treatment. Emergency personnel can help change the psychological and physical impact of sexual assault. Through proper care of the victim, careful and thorough acquisition of evidence, and cooperation with the law and the legal process, ED staff can help the victim to recover from the assault and can aid society in improving the prosecution and conviction rates of rapists.

## MANAGEMENT OF THE ADULT FEMALE SEXUAL ASSAULT VICTIM

An important aspect of performing a sexual assault evaluation is preparation. This includes the establishment of a protocol and the assembly of appropriate forms and equipment. All of this must be accomplished through a cooperative effort with the police,

crime laboratory, hospital laboratory, crisis volunteers, and the ED clerical, social service, nursing, and physician staffs. Careful step-by-step planning concerning the way in which a victim is handled in the ED and in follow-up helps both to ensure the best care for the victim and to aid in the prosecution and conviction of assailants (Table 62-1) .

Major provisions in the ED protocol should include patient privacy and the designation of a separate area for the care of the victims. Emergency personnel should know the appropriate steps for evaluation and treatment of the victim, follow-up care, and maintenance of evidence. Examination kits should be available in the ED, and the staff should be familiar with them. A list of the contents of the kits should be kept in the department (Table 62-2) (Table Not Available) . The kits save a tremendous amount of nursing and physician time when a

| TABLE 62-1 -- Physicians' Responsibilities for Victims of Sexual Assault |
|---|
| **Medical** |
| Provide a safe and compassionate environment for the victim |
| Obtain a focused and nonjudgmental history of the assault |
| Obtain an accurate gynecologic history |
| Assess and treat physical injuries |
| Obtain appropriate cultures and treat existing infections |
| Provide therapy to prevent unwanted pregnancy |
| Provide prophylaxis for sexually transmitted diseases |

| |
|---|
| Provide for immediate and follow-up counseling |
| Arrange for follow-up medical care |
| Offer shelter and coordinate social services for the immediate discharge period |
| **Legal** |
| Provide accurate, objective, nonjudgmental recording of events |
| Document injuries |
| Collect forensic samples and maintain the proper chain of evidence |
| Report to authorities as required |
| Provide testimony at trial if required |

victim comes to the department. A checklist for "sexual assault examinations" (Table 62-3) should be included in the kits. This serves as a reminder of all of the medicolegal procedures to be completed.

Even though this chapter is primarily devoted to the evaluation of the adult *female* sexual assault victim, a guideline to the evaluation of the adult male sexual assault victim, the female child victim, the male child victim, and the accused assailant is provided in Table 62-4 . Each of these groups has special needs, and separate protocols should be developed for them in the ED.

**Consent**

Consent for treatment of a victim of sexual assault is mandatory. The victim has

undergone an experience in which her right to grant or deny consent was taken from her, [7] and obtaining consent for medical treatment and for the gathering of evidence has important psychological and legal implications. The victim has the right to refuse medicolegal examination and even medical treatment. She should, however, be encouraged to have physical evidence collected in the event that she later decides to seek prosecution. Witnessed, written, informed consent should therefore be obtained before evaluation and treatment are begun. If the victim refuses any part of the examination, her wishes should be honored, and the refusal should be noted in the hospital record.

When the court orders an examination but the victim does not want to be examined, the physician must realize

| TABLE 62-3 -- Checklist for Sexual Assault Examinations | |
|---|---|
| | **General** |
| _____ | Photographs |
| | Urine |
| _____ | Pregnancy test |
| _____ | Urine drug screen |
| | **Blood** |
| _____ | Serologic test for syphilis |

| | |
|---|---|
| _____ | hCG-beta subunit |
| _____ | Ethanol or complete drug screen, or both |
| _____ | Red-topped test tube for blood typing |
| | **Pelvic Examination Specimens** |
| _____ | Pap smear |
| _____ | 3 vaginal swabs |
| _____ | Potassium hydroxide slide |
| _____ | Normal saline slide |
| _____ | Dry mount slide |
| _____ | Thayer-Martin plates (gonococcal culture) |
| _____ | _Chlamydia_ screening test |
| | **Buccal Specimens** |

| | |
|---|---|
| _____ | 3 buccal swabs |
| | **Rectal Specimens** |
| _____ | 3 rectal swabs (done prior to introduction of lubricant) |
| | **Pubic Hair** |
| _____ | Combed hair |
| _____ | Cut or plucked hair [*] |
| | **Fingernails** |
| _____ | Scrapings from under nails |
| | **Clothes** |
| _____ | Panties and any other articles requested by police |

* Done later if needed

**TABLE 62-4** -- Sexual Assault Examination Protocol for Medical Personnel

|  | Female Adult Victim | Female Child Victim (Prepubertal) | Male Adult Victim | Male Child Victim | Male Suspect |
|---|---|---|---|---|---|
| I.History |  |  |  |  |  |
| A. History of the event | * | * | * | * |  |
| 1.Time, date, place | * | * | * | * |  |
| 2.Use of force, threats of force | * | * | * | * |  |
| a. Type of violence | * | * | * | * |  |
| b. Threats of violence | * | * | * | * |  |
| c. Use of restraints | * | * | * | * |  |
| d. Number of assailants | * | * | * | * |  |
| e. Use of alcohol or drugs | * | * | * | * | * |
| f. Loss of consciousness | * | * | * | * |  |
| 3. Type of assault | * |  | * | * |  |

| | | | | | |
|---|---|---|---|---|---|
| a. Fondling | * | * | * | * | |
| b. Vaginal penetration (or attempt) | * | * | | | |
| c. Oral penetration (or attempt) | * | * | * | * | |
| d. Anal penetration (or attempt) | * | * | * | * | |
| e. Ejaculation--where on or in body | * | * | * | * | |
| f. Was condom used? | * | * | * | * | |
| g. Use of lubricant | | * | | | |
| B. Sexual history | | | | | |
| 1. Use of birth control | * | I.A. | | | |
| 2. Last voluntary intercourse or sexual activity | * | I.A. | I.A. | I.A. | * |
| 3. Gravidity, parity | * | | | | |

| | | | | | |
|---|---|---|---|---|---|
| 4. Recent gynecologic surgery | * | I.A. | | | |
| 5. Recent veneral disease | * | I.A. | * | I.A. | * |
| C. Medical history | | | | | |
| 1. Current medications | * | * | * | * | * |
| 2. Tetanus immunization status | * | * | * | * | * |
| 3. Allergies | * | * | * | * | * |
| D. History of douching, bathing, urination, defecation, or enema use following the assault | * | * | * | * | |
| II. Physical Examination | | | | | |
| A. Rapid survey for airway, breathing, circulation | * | * | * | * | * |

| | | | | | |
|---|---|---|---|---|---|
| B. Inspection of clothing for signs of violence or other evidence, such as feces, semen, blood (retain appropriate evidence, consider photographs) | * | * | * | * | * |
| C. Examination of all areas of skin for signs of violence, foreign material (retain appropriate evidence, consider photographs) | * | * | * | * | * |
| D.Examination of extremities for fractures, sprains, and so forth | * | * | * | * | * |
| E. Examination of oral cavity for signs of trauma, infection | * | * | * | * | * |
| F. Examination of breasts for trauma | * | * | * | * | * |
| G. Genital/rectal examination | * | * | * | * | * |
| 1.Male genitalia | | | * | * | * |
| a. Examination for signs of trauma to penis, dried semen, infection, and so forth | | | | | |

| | | | | | | |
|---|---|---|---|---|---|---|
| b. Examination of shaft of penis for lubricant, feces, blood | | | | | | * |
| c. Examination for signs of trauma to testicles, scrotum | | | | * | * | * |
| d. Examination for vasectomy scars | | | | | | * |
| 2. Female genitalia | | | | | | |
| a. Wood's light (filtered ultraviolet) to detect seminal stains on perineum | * | * | | | | |
| b. Inspection of vulva for signs of trauma or semen | * | * | | | | |
| c. Inspection of introitus | * | * | | | | |
| d. Inspection of hymen | * | * | | | | |
| i. By speculum examination | * | I.A. | | | | |
| ii. By separating vulva manually | I.A. | * | | | | |

| | | | | | |
|---|---|---|---|---|---|
| e. Inspection of vaginal vault for trauma and foreign bodies | * | I.A. | | | |
| f. Inspection of cervix | * | I.A | | | |
| i. For parity | * | | | | |
| ii. For signs of pregnancy | * | I.A. | | | |
| iii. For menstruation | * | | | | |
| iv. For trauma | * | I.A. | | | |
| v. For signs of infection | * | I.A. | | | |
| g. Refer to Section III: H, I, J, K, L of this protocol | * | * | | | |
| h. Bimanual pelvic examination | * | | | | |
| i. Palpation of uterus per rectum | * | * | | | |
| 3. Anal and perianal area | * | * | * | * | |

| | | | | | |
|---|---|---|---|---|---|
| a. Inspection for signs of trauma | * | * | * | * | |
| b. Inspection for signs of lubricant, semen, blood, foreign material, pre-established infection | * | * | * | * | |
| c. Digital rectal examination for trauma, foreign bodies | * | * | * | * | |
| d. Refer to Section III: M, N of this protocol | * | * | * | * | |
| III.Laboratory | | | | | |
| A.Photography (optional) | * | * | * | * | * |
| 1. Signs of trauma (patient clothed) | * | * | * | * | * |
| 2. Signs of restraint (patient clothed) | * | * | * | * | |
| 3. Signs of trauma (patient unclothed) | * | * | * | * | * |
| 4. Signs of restraint (patient unclothed) | * | * | * | * | |

| | | | | | |
|---|---|---|---|---|---|
| B. Clothing collection (for secretions, blood, semen, signs of violence, and so forth) | * | * | * | * | * |
| C. Removal of dried seminal or blood stains from skin | * | * | * | * | * |
| D. Fingernail scrapings for foreign material | * | * | * | * | * |
| E. Combings from pubic hair | * | I.A. | * | I.A. | * |
| F. Plucked or trimmed pubic hair | * | I.A. | * | I.A. | * |
| G. Plucked or trimmed head hair | * | * | * | * | * |
| H. Vaginal pool aspiration | * | I.A. | | | |
| 1. Sperm motility and morphology | * | I.A. | | | |
| 2. Acid phosphatase | * | I.A. | | | |
| 3. Blood group antigens | * | I.A. | | | |

| | | | | | |
|---|---|---|---|---|---|
| 4. Sperm precipitin test | Optional | Optional | | | |
| I. Swab of posterior vaginal fornix (same tests as previously described if vaginal aspirate not available) | * | I.A. | | | |
| J. Vaginal washing with 10 mL saline solution (same tests as described previously if vaginal aspirate not available) | * | I.A. | | | |
| K. Pap smear from cervix and vaginal wall | * | I.A. | | | |
| L. Gonorrhea cultures | * | * | * | * | * |
| 1. Cervix | * | I.A. | | | |
| 2. Rectum | * | * | * | * | I.A. |
| 3. Oropharynx | * | * | * | * | I.A. |
| M. Collection of foreign material from perianal area (especially lubricant) | * | * | * | * | I.A. |
| N. Rectal washing | I.A. | I.A. | I.A. | I.A. | |

| | | | | | |
|---|---|---|---|---|---|
| 1. Sperm motility and morphology | I.A. | I.A. | I.A. | I.A. | |
| 2. Acid phosphatase | I.A. | I.A. | I.A. | I.A. | |
| O. Saliva for secretor status | See Note 1 | See Note 1 | See Note 1 | See Note 1 | See Note 1 |
| P. Blood samples | * | * | * | * | * |
| 1. Serologic test for syphilis | * | * | * | * | * |
| 2. Drug and alcohol screen | I.A. | I.A. | I.A. | I.A. | I.A. |
| 3. Blood typing | * | * | * | * | * |
| 4. Pregnancy test (beta subunit of hCG) | See Note 2 | | | | |
| Q. Urine samples | I.A. | I.A. | I.A. | I.A. | I.A. |
| 1. Pregnancy test (serum test preferred) | See Note 2 | | | | |
| 2. Drug screen | I.A. | I.A. | I.A. | I.A. | I.A. |

| | | | | | |
|---|---|---|---|---|---|
| R. Penile shaft swabs | | | | | * |
|   1. For vaginal epithelium | | | | | * |
|   2. For fecal stains | | | | | * |
| S. Penile urethral swab | | | I.A. | I.A. | * |
|   1. Gram stain for gonorrhea | | | I.A. | I.A. | * |
|   2. Culture for gonorrhea | | | I.A. | I.A. | * |
| T. Radiographs for trauma | I.A. | I.A. | I.A. | I.A. | I.A. |
| IV. Consolidation of Evidence | * | * | * | * | * |
| V. Initiation of "Chain of Evidence" | * | * | * | * | * |

I .A., If appropriate.

* Indicated.

*Note 1:* Collection of salvia for secretor status is appropriate only if samples from the body, clothing, or crime scene potentially containing antigens that could link the victim with the suspect or suspects have been or will be obtained.

*Note 2:* The beta-hCG serum pregnancy test is more sensitive and quantitative.

*Chlamydia* screening tests may also be used.

that an uncooperative victim (who indeed has the right to refuse examination) can make a thorough examination impossible. Pursuing an examination that is unwanted by the victim can add to the victim's psychological trauma; the examining physician should discuss the situation with the authority that issued the court order and resolve the conflict before proceeding with the examination.

**History**

It should be emphasized that the sexual assault victim is actually a patient whose physical stability takes priority over all else (e.g., if she is in physical danger from hemorrhage, shock, or respiratory insufficiency, treatment of these conditions takes precedence).

The history appropriate to a sexual assault case basically is divided into 3 categories: history of the event, gynecologic history, and medical history (Table 62-5) . The history of the event should include only those elements necessary for the physician to complete a thorough physical examination and to collect evidence. Questions beyond this, such as a description of the assailant, should be left to the police investigators. Limiting the history not only shortens the evaluation in the ED, but it also helps to prevent discrepancies between the

| **TABLE 62-5** -- Components of History and Physical Examination |
| --- |
| **History** |
| Accurate but objective account of events |
| Avoid detailed description of assailant to avoid discrepancies |
| Thorough sexual and gynecologic history |

| **Physical Examination** |
|---|
| Collect clothing (if victim has not changed clothes) |
| External evaluation (total body) |
| Cuts |
| Bruises |
| Bite or restraint marks |
| Ultraviolet light for semen stains |
| Toluidine dye test (optional) |
| Oral cavity |
| Swabs (washing optional) |
| Injuries due to oral penetration |
| Culture for gonorrhea |

| |
|---|
| Genitalia |
| Examination for gynecologic pathology, trauma, and foreign |
| objects |
| Hair combing |
| Vaginal swabs |
| Avoid lubricants prior to sampling |
| Do not look for spermatozoa |
| Cultures/ *Chlamydia tests* |
| Rectal |
| Swab or washings |
| Avoid lubricants prior to sampling |
| Examination for trauma |

| |
|---|
| Culture for gonorrhea |
| Other |
| Pregnancy testing, RPR/VDRL, blood typing (optional) |
| Prophylaxis for gonorrhea, *Chlamydia,* syphilis, and hepatitis B (optional) |
| Pregnancy prophylaxis |
| Assess HIV issues |

ED history and the official police investigation report, which could weaken the victim's case if it comes to trial. For the sake of time, the medical history may be obtained by a nurse and expedited by the use of a checklist, which is then reviewed by the physician.

The history of the event should include the time, date, and place of the alleged assault and a description of the use of force or threats of force and the type of assault. Elements of force include the type of violence used, threats of violence, the use of restraints, the number of assailants, the use of alcohol or drugs (forcibly or willingly) by the victim, and any loss of consciousness experienced by the victim. Elements of the type of assault include fondling; vaginal, oral, or anal penetration or attempted penetration; ejaculation on or in the body; the use of a condom; and the insertion of foreign bodies into the vagina or the anus. The use of force or violence is partly a police matter, but from a medical standpoint it is desirable to correlate the physical examination with a description of any force, restraint, or violence.

A basic gynecologic history should be obtained in preparation for treatment plans regarding venereal disease and pregnancy. For example, it might be important from a medicolegal standpoint to know of any recent gynecologic surgical procedure that could be misinterpreted as local trauma. The gynecologic history therefore includes the use of birth control before the attack (with information regarding any missed birth control pills), last normal menstrual period, last voluntary intercourse, gravidity and parity, recent gynecologic surgery, and recent venereal disease.

The medical history should include current medications, tetanus immunization status, and allergies. One should note whether the victim douched, bathed, urinated, defecated, used a mouthwash, or brushed her teeth following the rape. These can alter the recovery of seminal specimens and other sexual assault evidence. In addition, any nonperineal or nonoral trauma should be reassessed when the medical history is taken.

Several elements of the history taken together can help the physician decide which samples to obtain. For example, sperm remain motile in the cervix for up to 5 days and remain motile in the vagina for 6 to 12 hours. [8] If the victim had voluntary intercourse 48 hours before examination and was raped 3 hours before examination, the physician should obtain samples from both the vagina and the cervix, keeping the 2 specimens separate. [9] By taking a careful history, the physician is able to perform an appropriate examination given these 2 events.

While taking the history, the examiner should observe the patient's ability to understand and respond appropriately to questions. Some victims of sexual assault are not capable of consenting to intercourse because of mental retardation, immaturity, or intoxication with drugs or alcohol. If intoxication is suspected, appropriate testing should be undertaken. If mental retardation is suspected, formal examination of the patient's mental status should be planned for a later date.

**Physical Examination**

The physical examination of the sexual assault victim is an extension of the history. The purpose of the examination is twofold: to aid in the proper medical evaluation of the patient and to gather samples and observations that might serve as evidence. The findings on physical examination help the physician and the court to answer the following questions: (1) Is or was the victim capable of consenting to intercourse? (2) Was force used? (3) Did vulvar, oral, or anal penetration occur? (4) Is there collectible physical evidence from the assailant in or on the victim's body?

A number of standardized examination kits have been developed to assist the physician in gathering evidence in a systematic fashion. Each kit should be reviewed for completeness as it applies to the local legal protocol.

Photographs should be considered, particularly if the victim is still wearing the clothing worn during the attack. Some institutions take their own photographs; others use police photographers. Patient consent should be obtained for photographs taken by hospital personnel, and a chain of evidence should be maintained. Self-developing film that can be permanently labeled (e.g., subject, date, details of pictured injury) should be used. The photographs should be labeled immediately and may be added to the legal evidence. These photographs may serve as evidence or may simply refresh the examiner's memory at the time of the trial. After the victim has been photographed, she should remove her own clothing, piece by piece, placing each item in a separate *paper* bag. If semen, blood, or other foreign material is present, it will dry in a paper bag,

whereas it could become moldy if placed in a plastic bag.

At this time the entirety of the patient's body, excepting the pelvic, anal, and oral areas, should be examined for signs of trauma and foreign bodies. Important areas for evaluation are the back, the thighs, the breasts, the wrists, and the ankles (particularly if restraints were used). Even in the absence of ecchymoses, tender contusions should be reported. Leaves, grass, sand, and other materials can occasionally be found in the hair or on the skin and should be retained as evidence. Areas of trauma should be documented and further evaluated (e.g., with radiographs) as indicated by the type and extent of injury. Up to 30% of rape victims show external evidence of trauma. [5] This evidence may range from abrasions to multiple major blunt and penetrating trauma. In addition, dried semen stains may be visible on the hair or the skin of the victim. These appear as lightly crusted, flaking areas that can be removed by saline-moistened swabs; the swabs are then air-dried and preserved as evidence. In addition to the skin, the fingernails can contain hidden bits of evidence. Rape victims may have bits of the assailant's skin, blood, or facial hair or other foreign material from the rape site beneath their fingernails; fingernail scrapings should be obtained routinely.

Following this initial examination, the patient should be placed in a lithotomy position for the pelvic examination. The thighs and the perineum should be inspected for signs of trauma and for foreign materials, such as seminal stains. An ultraviolet light used in a darkened room can aid in the search for external seminal stains. Any such stains should be removed and preserved as previously described.

The pelvic examination includes careful evaluation of the vulva and the introitus for signs of trauma, foreign material, and degree of maturity or senility. The internal examination should be done with a water-lubricated speculum. [10] The hymen should be inspected and one of the 4 following conditions noted: (1) that the hymen is present, intact, and free of evidence of trauma; (2) that the hymen is present and intact and shows old scarring; (3) that the hymen is present and recently ruptured; or (4) that the hymen is absent. A recently ruptured hymen is associated with bleeding or fresh clots. The breadth of perineal findings in normal and assaulted children has recently been reviewed. [11]

The vaginal wall should be inspected for lacerations. These appear near the introitus in younger, sexually inexperienced females; in older, sexually active females they appear higher, particularly in the right fornix. [12] [13] The cervix should be inspected for signs of pregnancy, menstruation, trauma, and preexisting infection.

**General Evidence Collection**

Protocols for evidence collection vary in different jurisdictions. Many rape evaluation centers have abandoned the cumbersome rape kits that have been used in the past, substituting simple collection methods that concentrate on important and usable legal evidence. The following discussion is patterned after the model protocol suggested by Young and associates. [14] As more sophisticated techniques using DNA analysis become legally accepted and more widely available, further modification in this

approach will undoubtedly evolve.

After inspection of the introitus, the vagina, and the cervix, specimens should be obtained. If at all possible, the speculum should be lubricated with water rather than a water-soluble lubricant to avoid contamination of specimens with the commercial lubricant. Generally, the specimens collected will be determined by local protocol, but they often include the following:

1. Any vaginal tampon that may have been inserted before or after the assault.
2. Any foreign body. Items identified have included condoms, soap bars, and handkerchiefs.
3. Vaginal swabs from pooled secretions in the posterior fornix (these may contain acid phosphatase or sperm) using a *dry* sterile cotton-tipped swab. A wet mount of one swab may reveal motile sperm if the examiner has ready access to a microscope. One swab can be used to make a dry mount for the crime laboratory, and 1 or 2 swabs can be air-dried for submission to the crime laboratory. These swabs are placed in a labeled envelope or red-topped tube for submission to the crime laboratory. If no pooled secretions are seen, a vaginal washing using 5 mL of sterile (but not bacteriostatic) water can be obtained with a syringe. The washing can be collected on a swab or placed in a sealed container for later examination for sperm and acid phosphatase.
4. Swabbing the oral cavity when oral penetration is stated or suspected. It may be helpful to obtain swabs along the gum line. Some examiners prefer to have the victim also swish or gargle with sterile water and collect those washings as samples. However, seminal fluid is rapidly destroyed by salivary enzymes, making identification difficult after a few hours. Similarly, swabbing the rectum should be performed if anal penetration is stated or suspected.
5. Consider a cervical, rectal, and/or oral culture or polymerase chain reaction (PCR) specimen(s) for *Chlamydia* and for *Neisseria gonorrhoeae.* Some clinicians treat empirically and forgo the culture or PCR testing. One series found gonorrhea cultures to be rarely positive in

   children<12 years of age and always associated with either a vaginal or a urethral discharge. [17] Each sample should be labeled separately; the label should include mention of the area from which the specimen was collected.After these samples have been obtained, a bimanual pelvic examination should be performed to assess uterine size and to identify adnexal masses and tenderness.

**Collection of Hair**

The victim's pubic hair can be combed for foreign material (particularly pubic hair belonging to the assailant) prior to the pelvic examination. These combings can be placed directly into a large paper envelope; the samples are submitted along with the comb. Foreign pubic hairs can help identify race and hair color, but hair does not generally possess enough individual characteristics to enable one to state positively that a hair of unknown source came from a particular person to the exclusion of all others. [18] In the future, crime laboratories may be able to perform enzyme typing of hair roots. Currently, sufficient phosphoglucomutase activity can be found in plucked hair roots to

enable typing of individual roots by starch-gel electrophoresis. [19] This capability may eventually be extended to hair roots collected by brushing.

Unfortunately, significant hair transfer occurs in <5% of assaults. Although the routine pulling of the patient's hair from the roots provides the best sample, the act of pulling the victim's hair is considered insensitive and unnecessary during the initial evaluation. The victim can provide the hairs at a later time--often the victim is willing to pluck the hairs herself at that time. Cutting locks of hair from the victim is essentially useless because this portion of the hair has no forensic value.

**Perineal Toluidine Dye Staining**

Another test that can be performed for sexual assault victims is the toluidine blue dye test for traumatic intercourse. Genital lacerations are corroborating evidence that rape has occurred, but without specialized staining techniques the majority of small lacerations go undetected. Toluidine blue greatly increases the detection of genital lacerations by highlighting lacerations with clear, linear marks easily distinguishable from episiotomy scars, rugae, and vulvitis. This test is highly sensitive, and small toluidine blue-highlighted lacerations may occur in vigorous non-rape intercourse, especially in adolescents. [20] This test involves the application of toluidine blue to the vaginal mucosa. Tears in the mucosa expose superficial nuclei of underlying cells. These nuclei have an affinity for the toluidine blue, and lacerations (small and large) become stained. In one study, 70% of nulliparous patients and 40% of the total number of patients examined within 48 hours after complaint of sexual assault demonstrated toluidine blue-positive lacerations. [21] McCauley and coworkers demonstrated that the use of toluidine blue increased the detection of vaginal lacerations in reported rape victims from 1 in 24 (4%) to 14 in 24 (58%). [20] Toluidine blue is primarily used in gynecology for outlining cervical neoplasia. [22]

The dye is applied with cotton-tipped applicators; all excess is wiped off with cotton balls until no further dye can be removed. Lacerations retain the dye (Fig. 62-1) (Figure Not Available) . Toluidine blue should be applied to the external genitalia before the insertion of a speculum, because the speculum itself may cause small lacerations. [21] Because of the potential spermicidal activity of toluidine blue, the examiner must decide on a case-by-case basis when to perform this test. The dye is not absorbed systemically, so it is considered safe in pregnant women. Note that the dye is not used intravaginally but only on external areas. If seminal stains are noted on the perineum, samples should be collected before toluidine blue application.

**Anal Evaluation**

The anorectal area should be examined for traces of foreign material (particularly lubricants) and trauma. Because of a reluctance of some victims to admit to anal or oral sodomy, some clinicians recommend an examination of these areas in all cases. When done, anorectal swabs can be obtained for sperm and acid phosphatase testing. Culture or PCR studies also may be desired as noted above. If the patient admits to anal penetration, one can perform a rectal washing by injecting 5 to 10 mL of normal saline with a syringe and a small plastic IV catheter, aspirating and preserving the fluid as

evidence. This washing can be examined for sperm and acid phosphatase, even though acid phosphatase determination has been of little value from samples taken from the anal canal and the rectum. [23]

**Oral Evaluation**

The mouth, particularly if oral sodomy is reported, should be inspected for signs of trauma. These can include bruises about the mouth, a torn frenulum of the lower lip, a torn frenulum beneath the tongue, and contusions or lacerations of the tonsillar pillars or the posterior pharyngeal wall. The examiner can test for acid phosphatase and sperm in the oral cavity by swabbing between the teeth with cotton-tipped applicators. The acid phosphatase test is seldom positive, but spermatozoa have been identified in oral smears up to 6 hours after the attack despite toothbrushing, using mouthwash, and drinking various fluids. [23] In addition, saliva samples (2 to 3 saliva-soaked swabs) may be obtained to assess the victim's secretor status of blood group antigens if foreign blood group antigens are found in the vagina. Most people (80%) secrete blood group antigens in saliva, semen, and other bodily fluids. However, determining the victim's secretor status can be done at a later time by the local crime laboratory. Consideration of pharyngeal gonorrhea culture or PCR testing should be made.

**Blood Tests**

Blood tests in sexual assault victims may include the following: drug and alcohol testing, blood typing, serologic test for syphilis, and, if appropriate, a pregnancy test using the beta-subunit of human chorionic gonadotropin (beta-hCG). [24] [25] If a serum pregnancy test is positive within a few hours or days of the assault, the victim was probably already pregnant at the time of the assault. If the test is negative at the initial examination but positive at a 2-week follow-up visit, it can be assumed that the victim became pregnant at or near the time of the assault. [9] Given the availability of sensitive and rapid bedside urine beta-hCG tests, many EDs are using this rapid assay to document pregnancy prior to consideration of pregnancy prophylaxis.

**Spermatozoa and Semen Testing**

Motile and immotile sperm may be found microscopically in wet mounts of vaginal aspirates and in vaginal, oral, and rectal swabs. Some examiners microscopically evaluate the slide immediately after the physical examination, because the forensic pathologist may not be able to examine the samples for several hours, days, weeks, or months after they are collected. At that time, sperm motility (a good sign of recent intercourse) has been lost. However, the absence of sperm does not rule out sexual assault. The assault may have been without penetration, there may have been coitus interruptus, or the assailant may have used a condom or have had a vasectomy. However, *some recommend that only the forensic pathologist examine the specimen for sperm,* so as to avoid potential conflicting evidence should later review identify the presence of sperm. [14]

Seminal plasma components (P30 and acid phosphatase) can be sought in the dried swab specimens at the crime lab. P30 is a glycoprotein specific to the prostate [26] and is

regarded as conclusive evidence of semen (i.e., ejaculation within 48 hours), whereas acid phosphatase is presumptive only because it can occur in other body fluids, such as vaginal secretions. Furthermore, low to intermediate levels of acid phosphatase do not rule out sexual assault. [27]

### Chain of Evidence

Samples and other evidence must be given to the police, a crime laboratory, or a forensic pathologist. Each sample must be labeled with the patient's name, the hospital number, the date and time of collection, the area from which the specimen was collected, and the collector's name. These specimens should then be packaged and transferred to the next appropriate official (police officer, pathologist, or other individual) along with a written "chain of evidence" that includes a list of the specimens, the signature of each person who provided them, and the signature of each person who received them. If this chain is broken, important evidence may be considered worthless in the courtroom.

## Treatment

### Sexually Transmitted Disease (STD) Prophylaxis

The factors of venereal disease, pregnancy, psychological distress, and follow-up should be considered in the treatment of a sexual assault victim. The risk of contracting a sexually transmitted disease (STD) as a consequence of a sexual assault has been difficult to determine, and estimates are tentative or nonexistent. One report of postassault STDs found a 3% chance of development of gonorrhea and a 0.1% chance of development of syphilis. [5] Others suggest that the risk for these STDs may be as high as 12% and 3%, respectively. [28] Jenny and colleagues found the postassault incidence of STDs to be 2% for *Chlamydia*, 4% for gonorrhea, 12% for *Trichomonas*, and 19% for bacterial vaginosis. [29] It is interesting to note that the latter group of women had a high rate (43%) of preexisting STDs at the time of initial examination. Glaser and others found similar STD infection rates. [30] The risk of developing herpes, cytomegalovirus, hepatitis B, or human immunodeficiency virus (HIV) infection from being raped has not been determined. However, HIV transmission has been noted. [31]

Depending on patient reliability and other factors, the examiner may choose to treat a victim as if she had been exposed to a known case of gonorrhea or *Chlamydia*; alternatively, the examiner may choose to rely on cultures. When cultures are obtained from the cervix, the pharynx, and the rectum, 90% of established gonorrhea cases can be diagnosed at a single visit. [32] Gonorrhea resulting from a rape may possibly be culturable within hours of the attack, but it should be almost always culturable at a 2-week follow-up visit.

Because victims tend to have a relatively low compliance with keeping follow-up visits, many physicians offer gonorrhea and *Chlamydia* prophylaxis at the time of the initial examination. If this course of treatment is chosen, the patient is essentially treated as if she had been exposed to a man known to have gonorrhea and/or *Chlamydia*. With the increasing prevalence of *Neisseria gonorrhoeae* strains resistant to penicillin and

tetracycline, ceftriaxone has become the antibiotic of choice in treating gonorrhea. Ceftriaxone also treats incubating syphilis. Intramuscular spectinomycin (2 g IM) and oral ciprofloxacin (250 to 500 mg PO) are single-dose alternatives for penicillin- and cephalosporin-allergic patients, but neither of these has been shown to be effective against incubating syphilis. No single-dose regimen for gonorrhea is effective against coexisting *Chlamydia trachomatis* infection, and patients should be given either a single dose of azithromycin (1 g PO) or a 7-day course of doxycycline (100 mg PO BID) or tetracycline (500 mg PO QID); a negative pregnancy test is a prerequisite for using either of the latter 2 antibiotics. [33] Erythromycin is a second alternative for *Chlamydia* prophylaxis in the pregnant patient.

One author has recommended consideration of hepatitis B prophylaxis, but this is not standard therapy. [4] Some recommend prophylaxis for *Trichomonas* with a single 2 g oral dose of metronidazole; again, this is not standard practice.

**Pregnancy Prophylaxis**

Pregnancy occurs in approximately 2 to 4% of sexual assault victims. The examiner must be careful to determine that pregnancy did not exist before the attack, and pregnancy tests should be obtained before any postcoital therapy is offered. The greater sensitivity of the serum beta-hCG test makes it the optimal evaluation. This test is sensitive at 5 days after implantation of the products of conception. However, newer urine tests with a detection threshold approaching 20 to 25 mIU/mL provide a reasonable alternative. These urine pregnancy tests should not be relied on if the patient's urine is quite dilute (specific gravity <1.010).

Sexual assault victims should be offered pregnancy prevention as outlined in Table 62-6. Diethylstilbestrol was once approved by the U.S. Food and Drug Administration (FDA), but it currently is not approved as a morning-after contraceptive. Ovral (50 mug of ethinyl estradiol and 0.5 mg

| **TABLE 62-6** -- Alternative Drug Regimens for Pregnancy Prevention in Female Sexual Assault Victims [*] |
|---|
| Oral diethylstilbestrol in a dose of 25 mg twice daily for 5 days |
| Intravenous conjugated estrogen (Premarin) in a dose of 50 mg once daily for 2 days |
| Oral conjugated estrogen (Premarin) in a dose of 30 mg once daily for 5 days |

| |
|---|
| Oral ethinyl estradiol in a dose of 5 mg once daily for 5 days |
| Oral ethinyl estradiol and norgestrel (Ovral), 4 tablets total; 2 at a time taken 12 hr apart |

* All should be begun within 24 hours of the sexual assault. Regimens may be effective for up to 72 hours post coitus. All of these preparations may cause nausea and vomiting. Note that treatment is started only after pregnancy has been ruled out.

of norgestrel) is approved for pregnancy prevention, but it has an unknown mechanism of action as a morning-after contraceptive. [34] Ovral works best if begun within 12 to 24 hours following the deposit of semen in the vagina but not >72 hours. A total of 4 tablets are taken orally, 2 tablets at a time, 12 hours apart. Adverse effects are nausea, vomiting, and breast tenderness. If the patient vomits within 1 hour of taking a dose, the dose should be repeated. Some routinely offer prophylactic antiemetic therapy; others reserve such treatment for patients who vomit. The failure rate of this regimen is reported to be 1.8%. [34] [35] Outside of the United States, RU 486 (mifepristone) is often used for postassault pregnancy prophylaxis. [36]

**Psychological Support**

Sexual assault precipitates a psychological crisis for the patient, and psychological care should begin when the patient first arrives in the ED. [37] [38] The victim often develops a posttraumatic stress disorder, manifested by numbed responsiveness to the external world, sleep disturbances, guilt feelings, memory impairment, avoidance of activities, and other symptoms. [39] The victim is particularly vulnerable to this stress disorder because of the following characteristics of sexual assault: (1) it is sudden, and the victim is unable to develop adequate defenses; (2) it involves intentional cruelty or inhumanity; (3) it makes the victim feel trapped and unable to fight back; and (4) it often involves physical injury. [40] The initial psychological care of the rape victim in the ED is fundamental (Table 62-7) (Table Not Available) . One can learn the methods for proper psychological care with a minimum of training.

**Post-examination Follow-up**

Follow-up for rape victims is essential. A 2-week follow-up examination conducted by a physician should be arranged; medical (venereal disease and pregnancy) and psychological reevaluation is performed at this time. Further evaluations can be performed at 4 and 6 weeks at the discretion of the physician following the patient. In addition, local volunteer support groups can be of immense assistance to a rape victim, and contact with such a group should be offered to each victim of sexual assault. Determining the victim's HIV status represents a complex issue. [41] Directing the patient to obtain anonymous HIV testing at a follow-up visit will keep the test results from the

defense counsel. HIV testing of the accused also is controversial and will depend on the willingness of the accused, local legal precedent, and other special circumstances (e.g., early pregnancy in the victim, in which case antiviral therapy may be beneficial to the fetus). [41]

**Legal Issues**

It is uncommon for the examiner to be called to court to testify in cases of sexual assault; if called, however, it is best to work with the prosecuting attorney to prepare testimony. However, *all chart notes should be written with the expectation that the ED evaluation and evidence collection will have to be explained and defended in court*. In some jurisdictions it is possible to minimize the time spent away from work by arranging to be called to the courtroom just before the time of testimony or by giving a deposition before the court date. Once on the witness stand, remember that one is called as the examining physician and not necessarily as an expert witness. The law requires that one testify only to one's best recollection and to what is indicated in the chart. Factual information in answer to questions should be given only if one knows the facts, and assumptions should be avoided. One should not be afraid to acknowledge the limits of one's knowledge or expertise. Statements such as "There were marks on the body that were consistent with bite marks" are preferable to statements such as "There were bite marks." It is the court's decision whether or not a person was raped, and the clinician is there to give information about the patient's presentation, statements, what was found, and what was done for treatment. Stick to the facts and avoid interpretation.

# Chapter 63 - Removal of an Intrauterine Device

**Robin R. Hemphill, J. Alan Morgan**

In 1987 it was estimated that 84.5 million women worldwide used an intrauterine device (IUD). [1] Of these, 59 million women were in China, where the IUD was the birth control method of choice. The IUD was introduced in the 1960s and has been the object of considerable controversy. Problems with the infamous Dalkon shield in the late 1970s resulted in 2 major IUD manufacturers withdrawing from the U.S. market. As a result, use of the IUD in the United States dropped by two thirds between 1982 and 1988, from 2.2 million women in 1982 to 0.7 million in 1988. [2]

Currently, while there are several models available for worldwide use, only 2 models have recently been available for use in the United States: Progestasert and ParaGard T 380A (Figs. 63-1 (Figure Not Available) and 63-2) (Figure Not Available) . The Progestasert is a T-shaped ethylene vinyl acetate copolymer with a vertical stem containing 38 mg of progesterone and barium sulfate in a silicone base. The device is 36 mm long and 32 mm wide and has 2 dark blue-black monofilament strings attached to the base. The contained progesterone supplies approximately 65 mug/day into the uterine cavity. The ParaGard T 380A is also a T-shaped device made up of polyethylene and barium sulfate. The exposed surface areas of copper are 380 ± 23 mm2 . Two off-white strings are attached to the base. The "A" designation identifies this model as having an enlarged bulbous stem base. This device is 36 mm long and 32 mm wide. Correct placement of an IUD within the uterus is shown in Figure 63-3 .

Multiple theories have been proposed for the contraceptive action of IUDs. One of the most widely studied theories suggests that the IUD inhibits implantation of the blastocyst through a variety of endometrial effects. Specifically, the IUD itself may mechanically interfere with implantation; the IUD may cause a local inflammatory response (particularly the copper-containing devices, which appear to cause significant inflammation) or release of prostaglandin that makes the endometrium unsuitable for implantation. The progesterone-releasing device may additionally disrupt endometrial maturation. Studies also indicate that IUDs are spermicidal. This may result from the induction of an inflammatory response at the endometrium that may cause sperm to be phagocytosed. Other suggested mechanisms of contraception are interference with normal development of unfertilized oocytes, reversal of ciliary action in the fallopian tubes, and changes in cervical mucus. Even a missing IUD tail has been reported to be the source of infertility. [6] Whatever the exact mechanism, the net effect is a highly effective form of reversible birth control. When the IUD is removed, the contraceptive effects rapidly disappear, and normal fertility returns in the majority of patients. There remains some concern that young nulliparous women may have an increased rate of infertility after IUD use, and this point is still controversial.

IUDs have 1-year and long-term contraception rates second only to implantable hormonal contraceptives and are at least as effective as oral contraceptives in preventing pregnancy. The Progestasert has a failure rate of 1.8 to 2.5 pregnancies per 100 women-years, and the Copper T 380A demonstrates a pregnancy rate of 0.5 to 1.0

per 100 women-years. After the IUD is placed, the rate of spontaneous expulsion is up to 15%, although this usually occurs during menses and within the first few months of being placed. This is more common in young nulliparous women. Bleeding and pain are the most common side effects reported by users of IUDs, and 5% to 15% of patients will have the IUD removed for these problems. [3] [5] The copper-containing devices are associated with more severe bleeding problems, and the Progestasert may be better tolerated.

## COMPLICATIONS OF IUD USE

There are several complications of IUD use that are of ongoing concern and continue to be debated. Multiple published studies suggest that the rate of pelvic inflammatory disease (PID) is increased substantially in women using IUDs. The Women's Health Study, published in 1981, concluded that the relative risk of PID in IUD users was 1.6. Other studies seemed to support these data and showed particularly high risk in women using the Dalkon shield. Since that time the original data have been reanalyzed, and many investigators currently believe that the original study was markedly flawed, and there is little increased risk of PID in IUD users. Still others argue that the original data were sound and believe that there is still a marked risk of PID. What seems clear is that the greatest risk of PID occurs within the first month of insertion and is likely related to the insertion process; PID after the first few months is more likely to be related to the patient's background risk of getting a sexually transmitted disease than to the simple fact that an IUD is in place. Whether the string that passes through the uterine cavity and cervix increases the risk of PID is debatable, as is the use of prophylactic antibiotics during IUD insertion.

There also is concern regarding an increased risk of ectopic pregnancy should the IUD wearer get pregnant. Because the IUD prevents pregnancy, overall the risk of ectopic pregnancy is decreased in IUD users (specifically, IUD wearers have 40% less chance of ectopic pregnancy than those using no contraception). [23] However, should a women become pregnant while using an IUD, there is a several-fold increased risk of ectopic pregnancy; the Progestasert appears to have a higher risk than the Copper T 380A. Women who have used an IUD in the past appear to have the same risk of ectopic pregnancy as women who have never used one.

The woman who becomes pregnant with an intrauterine pregnancy while the IUD is in place may experience several complications. A higher than normal percentage of IUD-failure pregnancies result in spontaneous abortion, and with the IUD left in place, the chance of a live birth is significantly reduced. [26] The type of IUD does not affect the risk of spontaneous abortion. Should the pregnancy proceed to term, the risks of premature delivery, stillbirth, and low birth rate are much higher than in a pregnancy without an IUD in place. [5] There is no known association with birth defects. Not only is the risk of spontaneous abortion higher (50%), but a number of those abortions may be septic in nature (2- to 13-fold increased risk), resulting in significant morbidity and mortality to the mother. Removing the IUD in the first trimester when the string is visible decreases the rate of spontaneous abortion to approximately 25%, and essentially eliminates the risk of subsequent septic abortion. This 25% rate of spontaneous abortion in women who have their IUD removed in the first trimester is not too dissimilar

from the 17% incidence of spontaneous second trimester abortion seen in women who conceive with no IUD in place. [27] In recent years there has generally been agreement that IUDs should be removed from the pregnant uterus when the string is visible.  When the string is not visible, the best management is controversial. Certainly, termination of pregnancy may be offered. For those for whom this is a difficult choice, ultrasound-guided IUD removal may be a possibility. [28] When such a patient is encountered in the emergency department, prompt follow-up should be arranged with an obstetrician to discuss the options available to the patient. However, any pregnant patient released from the emergency department with an IUD still in place must be given detailed instructions advising her to return for care if she should develop flu-like symptoms, purulent vaginal discharge, abdominal pain, pelvic pain, temperature elevation, or back pain. Septic abortion can proceed rapidly to fulminate septicemia and death.

Those patients who have been using an IUD for a prolonged time will sometimes have *Actinomyces*-like structures identified in Papanicolaou smears. This may occur in an average of 12% of women (range, 1% to 35%), but there does not appear to be an increased rate of gynecologic symptoms in those patients with the organisms. [23] Once the smear becomes positive for these organisms, subsequent smears will remain positive until the device is removed. Most experts agree that if signs or symptoms of infection develop in women who harbor *Actinomyces* bodies, the IUD should be removed and antibiotic therapy started. Asymptomatic patients with positive smears can probably be followed clinically. Concern that an inflammatory stimulus that remains within the uterus for a prolonged period might increase the risk of cervical or endometrial carcinoma has not been borne out. In fact, there is some evidence to suggest that IUD wearers may have a decreased risk of endometrial and cervical cancer.

Any time a foreign object is inserted blindly into a closed cavity, there is a risk of perforation, and the use of the IUD is no exception. Perforation of the uterus is among the more serious of the complications that occur with the use of the IUD. When perforation occurs, it is usually at the time that the IUD is inserted. While an experienced operator generally avoids this complication, there is a nearly ten-fold increase in the risk of uterine perforation if the IUD is placed while a woman is lactating. [1] [3] The overall risk of perforation is from 0.5 to 2 per 1000 insertions. [1] Perforation may be complete or partial, with the IUD embedded within the uterine wall. The IUD may never move from this position, or the normal uterine contractions could cause the IUD to gradually migrate through the uterine wall. The literature is replete with cases of contiguous organs that have become involved with the migrating IUD.  Given these different mechanisms of perforation, it should be clear that the patient could present with problems any time between shortly after insertion to years after insertion of the IUD.

Most commonly, unless rare complications develop, uterine perforation by an IUD is completely asymptomatic. It is usually suspected when (1) disappearance of the string is discovered during routine examination or by the patient herself during self-examination, (2) pregnancy develops and the IUD is discovered to have migrated, or (3) symptoms of abdominal pain with or without vaginal bleeding develop in a patient with a known IUD. [34] The loss of a string does not necessarily indicate a perforation (although most studies report a 5% to 10% perforation rate in missing strings), [37] nor

does the presence of a string mean that there is not a uterine perforation. However, when the string is missing, further evaluation is indicated.

In general, IUDs have a low risk of death, with IUD-related mortality ranging from 1 to 10 deaths per 1 million women-years. This makes the IUD one of the safest methods of contraception and far safer than pregnancy. These statistics are consistent, regardless of patient age, for the child-bearing years. The IUD also is a cost-effective means of birth control; the ParaGard T 380A can be left in place for 5 years (with some studies suggesting up to 10 years), and the Progestasert has a life span of 1 year. The IUD appears to be safe and effective for birth control in a well-chosen population. [40] Certainly, neither the IUD nor oral contraceptive pills alone should be advised for young nonmonogamous women, as these forms of birth control offer no barrier protection against acquired immunodeficiency syndrome (AIDS) and other sexually transmissible diseases. Given the ongoing debate about the increased risk of tubal infertility in nulliparous women, this group may not be the best population for use of the IUD. However, in older, monogamous, parous women who desire a safe, simple, and effective means of contraception, the IUD appears to be a reasonable choice.

## INDICATIONS FOR IUD REMOVAL

The most common reasons for removal of an IUD are as follows:

1. Severe uterine cramps or lower abdominal pain
2. Excessive and persistent uterine bleeding, either with the menstrual period or between periods
3. Perforation of the uterus, with passage of the device to extrauterine sites
4. Downward displacement of the device into or partially through the cervical canal
5. Intrauterine pregnancy
6. Pelvic inflammatory disease

Increased uterine cramping and bleeding are common and expected side effects of IUD use and are the most common reasons that IUDs become unacceptable to the user. [1] [3] A patient may present with this type of complaint, asking that the IUD be removed because the pain or bleeding has become unbearable. It is important to rule out infection or pregnancy as the source of the increased pain or bleeding, after which the device can be removed.

Displacement of the IUD is another indication for removal of the device. This can present as a partially extruded IUD or as a missing string in a patient with a known history of past IUD placement. If the IUD is partially extruded from the uterus, it can cause pain or bleeding and possibly penile lacerations during coitus. For these reasons, the device should be removed if it is found to be out of place during the pelvic examination.

In the case of a missing IUD string, there are a number of possible explanations. In some cases the patient may simply be unable or unwilling to examine herself for the presence of the string and may mistakenly lead the physician to believe the string is missing; in such cases a complete examination will reveal the string to be in the

appropriate position (extending 2 cm from the external os). Another cause of a missing string is spontaneous expulsion. Up to 20% of patients with spontaneous expulsion may not be aware that they passed the device, and in this case the evaluation should continue until the device is proven to no longer be present in the woman. [34] The string may also have simply retracted up into the cervical or uterine canal, or the IUD may have turned within the uterus. Again, the important issue to address is whether the IUD is still in the appropriate position. Finally, the 2 most concerning causes of a missing IUD string are pregnancy (in which the enlarging uterus pulls the string into the uterus) and perforation of the uterus by the IUD. [5] The incidence of uterine perforation in patients with missing strings varies from 3% to 22%, with most series reporting an incidence of 5% to 10%. [34]

The final indication for IUD removal is the presence of PID. Although this is controversial, most experts recommend removing the IUD in the presence of pelvic inflammatory disease, even mild cases, as its continued presence may make antibiotics less effective in eradicating the infection. [42] In the presence of infection, broad-spectrum antibiotics should be started and continued for 10 to 14 days; these are generally administered intravenously. In the patient with a complaint of pain and bleeding, it is difficult to ensure that infection is not a contributing factor. If there is doubt about the presence of infection, it is safer to administer antibiotics. Some authors advocate administration of antibiotic 30 minutes prior to the actual IUD removal. Durack and Phil reviewed endocarditis prevention and noted that in the absence of infection, antibiotics were not needed prior to IUD removal, except in certain high-risk patients such as those with severe native valve disease or prosthetic valves. [43] Antibiotics are not required if the IUD is being removed and there is no infection present.

## EQUIPMENT

Equipment useful in IUD removal is shown in Figure 63-4 . The items include 2 different vaginal specula (the Graves speculum is broad, and the Pederson speculum is narrow), Bozeman forceps, ring forceps, a tenaculum, and a uterine sound. Sponges; large, cotton-tipped applicators; and an antiseptic solution should also be available.

## PROCEDURE

Prior to removal of the IUD, the patient should have the procedure explained carefully, with the indication for IUD removal discussed, as well as the risks and benefits. This is particularly true of the pregnant patient, who must understand that any manipulation of the cervix and uterine cavity, no matter how uncomplicated and minor, may be associated with a spontaneous abortion. The complications of the procedure, which include cramping, bleeding, infection, and uterine perforation, must be explained. Preferably, a consent form listing these risks and complications should be signed by the patient and a witness and kept with the patient's medical record.

In any patient who desires IUD removal, a pregnancy test should be done prior to any manipulation. If pregnancy is ruled out, the patient may be placed in the lithotomy position and the cervix swabbed with povidone-iodine or a soap solution. If the IUD string is visible, it may be grasped with a Bozeman forceps and pulled out using slow,

gentle traction. Stabilizing the uterus with a tenaculum may occasionally be helpful. A jerking motion should never be used as the motion may cause the string to break, or cause uterine perforation if the IUD is imbedded within the uterine wall. If the string breaks when removal is attempted, partial or complete perforation must be presumed, and the patient should be appropriately referred. [34] If the IUD does not easily come free with steady, firm traction, the device should be left in place and gynecologic consultation obtained. Excessive manipulation of the IUD or probing of the uterus to free the device increases the risk of hemorrhage, infection, and perforation.

If the patient presents with a history of IUD placement and the string is not visible at the cervical os, several scenarios are possible: the IUD may have shifted within the uterus, the IUD may have fallen out unnoticed, the IUD may have perforated the uterus, or the patient may be pregnant. After determining that the patient is not pregnant, it is important to determine the IUD location. The IUD location often can be determined with a radiograph. A uterine sound may be positioned gently at the cervical os to determine where the IUD is in relation to the uterus and cervix on posterior and lateral radiographs of the pelvis. Alternatively, sonography may help localize the missing IUD. If the IUD is found to be outside the uterus, it can usually be removed as an elective operative procedure unless there is concern regarding an acute infection or other severe complication. The device should be removed to reduce the risk of adhesions and bowel obstruction.

In the nonpregnant patient, gentle probing of the external os with Bozeman forceps can often determine whether the string has retracted slightly from view. When the string is located in this manner, the string can be advanced into the vaginal canal until resistance is met. This maneuver may be sufficient when the patient presents solely because of concern regarding a "missing" IUD. Should the patient request removal, the IUD can then be removed as discussed above. There are several methods described in the literature for locating and removing a "missing" IUD believed to remain within the uterus. These include ultrasound-guided retrieval,  as well as techniques using a Lamicel, [46] the Mi-Mark Helix, [47] the Rocket retrieval forceps from London, or a uterine sound. These techniques are beyond the scope of this text.

If the patient is found to be pregnant and the string is not visible, retrieval should not be attempted by the emergency physician. The patient should be informed that if she maintains the pregnancy, she is at high risk for a second trimester spontaneous abortion, and it may be septic. Some centers attempt ultrasound-guided intrauterine removal of the IUD in early pregnancy, but this approach is beyond the scope of this text. [28] Some obstetricians may recommend elective abortion in those women who do not feel strongly about maintaining the pregnancy.

## AFTERCARE INSTRUCTIONS AND COMPLICATIONS

Patients usually tolerate uncomplicated removal of the IUD with no difficulty. It is reasonable to give the patient a mild analgesic to take home in case of cramping. The patient should be told to return to the emergency department for severe pain, bleeding, or fever. If the uterus is believed to be perforated, the patient should be given broad-spectrum antibiotics, and a gynecologist should be consulted for admission.

When the IUD is removed, the patient should be notified that she no longer has a contraceptive device and should use another form of contraception from that point on.

Removal of the IUD should be a careful and gentle procedure. Multiple forceful attempts at removal should be avoided. Prior to a removal attempt, the patient should be counseled that the removal attempt may be unsuccessful. In this case the patient will need referral to a gynecologist for further attempts at removal. An expected complication of removal is mild bleeding and uterine cramping. Patients who develop severe, unrelenting bleeding; pain; or evidence of sepsis may have a uterine perforation as a result of removal or attempted removal. These patients need resuscitation, broad-spectrum antibiotics, and urgent admission to a gynecologist.

For the patient found to be pregnant and with a history of IUD placement, the first step is to ensure that the patient has an intrauterine pregnancy. As mentioned previously, the IUD user who becomes pregnant has an increased risk of ectopic pregnancy. It is critical to verify that the pregnancy is intrauterine so that this complication of IUD use is not missed. After confirming an intrauterine pregnancy, it is appropriate to attempt IUD removal if the IUD string is visible. However, the patient must be fully informed that any manipulation of the pregnant uterus may result in spontaneous abortion.

## CONCLUSION

In the patient with a visible IUD string and appropriate indications, IUD removal is usually a simple and safe procedure, as long as the IUD is in the proper intrauterine position. Removal of the IUD is clearly indicated and beneficial in the first trimester of pregnancy and can be done in the emergency department. However, the risk of spontaneous abortion must be understood by the patient. If the IUD string is not visible, it is important to ensure that the patient is not pregnant and then to locate the IUD. This is best done using radiographs or ultrasound. Blind attempts at uterine explorations are best left to the gynecologist. If there is a suggestion of uterine perforation, the patient should receive timely referral to a gynecologist for further evaluation.

# Chapter 64 - Management of Increased Intracranial Pressure and Intracranial Shunts

**Alan B. Storrow**

Increased intracranial pressure (ICP) has multiple causes, but its pathophysiologic consequences are similar for all of them. Increased ICP impairs cerebral perfusion and causes ischemic damage to the brain. Mass lesions lead to intracranial shifts and directly damage brain tissue. Whatever its cause, increased ICP carries a high morbidity rate and may be rapidly fatal if not recognized and treated. Hence, recognition and management of this condition are important skills for emergency physicians.

In addition, many hydrocephalus patients with the potential for long-standing increases in ICP are being treated with indwelling cerebrospinal fluid (CSF) shunts and may present with shunt-related complications. Unfortunately, the emergent management of shunt-related problems is complicated by the great variety of shunts and the variability of their emergent presentations. Key issues in the management of CSF shunt problems include device identification, physical examination, radiographic examination, and access.

## PROCEDURES TO LOWER INCREASED ICP

Normal ICP (5 to 15 mm Hg) is maintained by regulation of three compartments: brain tissue volume, cerebral blood volume, and CSF volume. Tissue volume may be increased by diseases affecting the blood-brain barrier, such as cytotoxic or vasogenic edema secondary to head trauma or anoxia. Tissue volume also may be affected by mass effect from hematoma or tumor. Cerebral blood volume is regulated by brain tissue blood flow and is affected by hypoxia, hypercapnia, vasoactive medications, and ICP. The CSF compartment is the most dynamic of the three compartments. CSF is produced by the choroid plexus, located in the lateral and fourth ventricles; flows out around the brainstem; and is reabsorbed in the brain tissue, sagittal sinus, and spinal nerve roots.

The emergency physician must become adept at identifying the symptoms, signs, and conditions associated with increased ICP. Although some patients may present with subtle symptoms and a gradual rise in ICP, others may deteriorate within minutes. It is prudent to understand the emergent nonoperative treatments available for lowering ICP.

**Intracranial Anatomy and Pathophysiology**

The cranial cavity is surrounded by the skull, a rigid nondistensible structure, and is divided into compartments by the semirigid, densely fibrous folds of the dura mater: the falx cerebri and tentorium cerebelli. There are 3 primary components within the cranial cavity: CSF, blood, and semigelatinous brain. If the volume of one of the cranial components is increased as a result of hematoma, pus, edema, or tumor, there must be a corresponding decrease in the volume of one or more of the three primary

components; otherwise, pressure within the compartment rises. Compensation can occur: (1) by expressing CSF from the ventricles and subarachnoid cisterns; (2) by expressing blood from the collapsible cerebral veins; or (3) in cases of slow-growing tumors, by expressing interstitial fluid from the brain itself. Once the volume of the mass exceeds the compensating capacity of the compartmental components, pressure rises rapidly, often within minutes, and brain shift occurs from one compartment toward another. Because of the attachment of cranial nerves and delicate blood vessels, the brain tolerates shift poorly. Progressive lateral displacement of the pineal gland on computed tomography (CT) scan is associated with progressive impairment of consciousness (e.g., 3 to 4 mm of displacement produces drowsiness, and 8 to 13 mm of displacement produces coma). [1]

In the case of an expanding temporal epidural hematoma, the most medial part of the brain, the temporal lobe uncus, will begin to pass into the tentorial notch (uncal herniation), and the cingulate gyrus will pass under the falx (subfalcine herniation) (Fig. 64-1) (Figure Not Available) . The herniating uncus commonly exerts pressure on the oculomotor nerve in the tentorial notch, resulting in pupillary dilation on the side of the hematoma (Fig. 64-2) (Figure Not Available) . Pupillary dilation is the most reliable sign for determining the side of the hematoma but is correct in only 80% of cases. [2] When pupillary dysfunction does occur contralateral to the mass lesion, it is often due to direct pressure of the tentorium cerebelli on the displaced oculomotor nerve contralateral to the mass. As brain shift continues, the opposite cerebral peduncle also can be forced against the free edge of the tentorium, producing extremity paralysis on the same side as the hematoma (Kernohan's notch; see Fig. 64-1) (Figure Not Available) . Direct pressure on the ipsilateral cerebral peduncle or, in the case of a posterior frontal hematoma, the motor cortex itself can produce a contralateral paralysis. Thus, paralysis is not as accurate a localizing sign as is pupillary dilation and can be present on the same or opposite side of the hematoma. Further herniation and shift of the midbrain will result in tearing of delicate perforating vessels. At this point, irreversible damage has occurred. Damage to the midbrain reticular formation results in deep unconsciousness and cessation of eye movements, and the pupils become fixed at midposition.

The infratentorial compartment is much smaller than the 2 supratentorial compartments; compensatory mechanisms are exhausted much more quickly, and the volume increases that can be tolerated are much smaller. Patients with initial symptoms of drowsiness, incoordination, ataxia, and nystagmus may suddenly become deeply unconscious and apneic. Bradycardia and wide pulse pressure from direct brainstem pressure are more common with infratentorial masses. There can be upward herniation through the tentorial notch or downward herniation of cerebellar tonsils through the foramen magnum (tonsillar herniation) (Fig. 64-3) (Figure Not Available) . Infratentorial hematomas are difficult to diagnose without CT. In the setting of trauma, a fracture on plain films extending through the occipital bone to the foramen magnum suggests an infratentorial hematoma.

While the relationship between increased ICP and brain shift is one mechanism for impairment of brain function, Miller also implicates the influence of ICP on cerebral blood flow. [3] A constant blood flow to the brain is normally maintained by automatic changes in cerebrovascular resistance at the arteriole level, despite fluctuations in systemic blood pressure. This is known as *cerebral autoregulation,* and it continues to

function in the presence of moderate elevations of ICP. Repeated elevations of ICP, however, are believed to damage cerebral autoregulation. [3] When autoregulation is lost, increased ICP reduces cerebral blood flow. As ICP approaches mean arterial blood pressure, blood flow ceases. This quickly leads to cerebral ischemia and infarction.

**Indications and Contraindications**

Intervention begins with identification of the symptoms and signs likely to indicate increases in ICP and the acute syndromes necessitating urgent diagnosis and treatment. [4] While the emergent nature of a comatose patient with an isolated closed head injury is easy to ascertain, the more subtle symptoms suggesting a rising ICP may be difficult to recognize.

Classic symptoms associated with increasing ICP include headache, a change in mental status, and vomiting. Headaches are more worrisome when they are nocturnal, occur on arising, worsen with cough or head motion, are new in middle age, are associated with an altered mental state, or occur before the age of 10. Early changes in mental status include lethargy, irritability, slow decision making, and loss of normal social behavior. If the condition remains untreated, mental status will deteriorate to stupor, coma, and death. Vomiting may occur without nausea early, and then can progress to projectile as ICP rises.

Examination of the pupils, fundi, extraocular movements, motor ability, arterial pressure, and pulse may reveal classic signs of increased ICP. Pupil changes include unilateral irregularity or dilation. Fundi may reveal blurring of the disc margins, loss of venous pulsations, disc hyperemia, and flame-shaped hemorrhages. Later, the disc margins become obscured, and retinal hemorrhages may be seen. Extraocular muscle examination may reveal unilateral ptosis or unilateral third and sixth nerve palsies. Later, ophthalmoplegia and loss of vestibulo-ocular reflexes may occur. Motor changes occur late and include hemiparesis, followed by bilateral hemiparesis. Late changes in vital signs include arterial pressure elevation; widened pulse pressure; and a slow, irregular pulse (the Cushing response).

Acute syndromes of increased ICP include head injury and obtundation; syncope, headache, and meningismus; focal deficit followed by obtundation; and seizures. [4] Even mild trauma may lead to hematoma formation and a rapidly expanding mass lesion. A delay in treatment may allow ICP to rise precipitously within minutes to hours. Abrupt onset of headache, especially when associated with syncope or meningismus, suggests a ruptured cerebral aneurysm or vascular lesion. Mass lesions can be associated with an initial focal deficit. When this is associated with edema formation or hemorrhage, intracranial compartment shift and increased ICP may result within minutes or hours. Seizures may be associated with mass lesions. Status epilepticus may cause a decompensation of cerebral volume regulation.

A further subset of patients who "talk and deteriorate" has been defined. Such patients utter recognizable words at some time after head injury, then deteriorate to coma within 48 hours.Although they represent a small group of patients, most have intracranial

hematomas, and outcome depends on early recognition.

A cranial CT scan should be obtained, if possible, on all patients with increased ICP. It may reveal mass lesions, hemorrhages, or enlarged ventricles. Direct measurement of ICP is desired for all high-risk patients, although this is rarely available in the emergency department. Lumbar puncture should not be performed prior to CT and has no role in the trauma patient.

Optimal treatment of increased ICP begins with airway management and maintenance of normal oxygenation. The first priority is to get oxygen to the brain. Since both hypoxia and hypercapnia produce cerebral vasodilation and increased ICP, intubation for maintenance of normal $Pa_{O2}$ and $Pa_{CO2}$ is indicated for all unconscious patients. Other nonoperative techniques, discussed below, include positioning, body temperature and seizure control, medications, anesthesia, and special techniques for patients with intracranial shunts (Table 64-1) . Operative techniques include craniotomy, shunt placement, and infant hematoma aspiration. Only infant hematoma aspiration will be discussed here.

## Equipment

Most of the procedures below do not require special equipment. Hematoma aspiration in the infant requires sterile prep and drape, gloves, razor, appropriate spinal needle (20 ga, 1½ or 2½ inch), manometer, CSF collection tubes, and local anesthesia (if desired).

## Technique

### Mechanical Ventilation

IV lidocaine (1.5 mg/kg) has been suggested to blunt the rise in ICP associated with intubation, [9] [10] perhaps by preventing the cough reflex. However, this belief has been refuted in a randomized trial that suggested that paralyzing agents were the only sure mechanism to prevent ICP increase during intubation. [11] Nonetheless, a lidocaine dose of 1.5 to 2 mg/kg IV is commonly recommended.

After intubation, mechanical oxygenation and hyperventilation can optimize $Pa_{O2}$ and $Pa_{CO2}$ to produce cerebral vasoconstriction and a lower ICP. A target $Pa_{CO2}$ of 25 to 30 mm Hg was originally suggested. [13] However, more recent work  suggests that the physician must balance the degree of cerebral vasoconstriction with the potential to cause cerebral hypoxia and impaired cerebral extraction of oxygen. For

**TABLE 64-1** -- Initial Emergent Procedures to Lower Increased ICP

| Pathway | Procedure | Initial Quantity | Time To Effect |
|---|---|---|---|
| Respiratory | Hyperventilation | $Pa_{CO2}$ 30 torr | 1-5 min |
| | Oxygenation | $Fi_{O2}$ , 100%, PEEP | 1-5 min |
| Medications | Mannitol | 0.5-2 g/kg IV | 5-60 min |
| | Furosemide | 0.5 mg/kg IV | 5-30 min |
| | Dexamethasone | 10 mg IV, IM | Hours to days |
| Seizure control | Benzodiazepine | | 1-5 min |
| Surgical | Craniotomy | Hematoma removal | Immediate |
| | Ventricle catheterization | Burr hole and catheter | Immediate |
| | Infant hematoma aspiration | | Immediate |

| | | | |
|---|---|---|---|
| Shunt patients | Chamber pumping | | 1-5 min |
| | Aspiration | | 1-5 min |
| | Proximal catheter puncture | | Immediate |
| Anesthesia | Pentobarbital | 20-35 mg/kg IV bolus | 30-60 min |
| | | 3-5 mg/kg/hr IV infusion | |

$Pa_{CO2}$, partial pressure of carbon dioxide in the arterial blood; $Fi_{O2}$, inspired oxygen content; PEEP, positive end-expiratory pressure.

the emergent management of these patients, it seems prudent to suggest a target $Pa_{CO2}$ of 30 to 35 mm Hg. After admission, more invasive monitoring of cerebral perfusion pressure, cerebral blood flow, and cerebral oxygen extraction may be undertaken to optimize this balance. An additional beneficial effect of hyperventilation may be optimization of global cerebral glucose extraction. [16]

Mechanical ventilation and positive end-expiratory pressure (PEEP) should be used to maintain a $Pa_{O2}$ of 80 to 100 mm Hg. [4] This level produces at least a 90% hemoglobin saturation, adequate to attenuate $Pa_{O2}$-mediated cerebral vasodilation.

**Positioning**

Placing the patient in a head-upright posture has been suggested to lower increased ICP. Despite concerns about the deleterious effects of an upright position on cerebral perfusion pressure and cerebral blood flow, 2 reports support moderate elevation, between 15° to 30°. This degree of elevation significantly reduced ICP and, in general, did not impair the perfusion pressure or blood flow. In addition, neck flexion or rotation to either side has been shown to significantly increase ICP. [21]

**Body Temperature and Seizures**

A rise in body temperature increases the brain's metabolic demands and should be treated with antipyretics or cooling. A recent investigation concluded that

acetaminophen was the single most effective method to lower brain temperature, performing better than isolated head cooling or nasopharyngeal cooling. [22]

Similarly, seizures markedly increase the brain's metabolic needs and cause a rise in ICP. They should be terminated with a rapidly acting benzodiazepine or barbiturate. Longer term seizure prevention may be initiated with phenytoin or phenobarbital.

**Medications**

Osmotic diuretics favor the passage of water from the brain to the systemic circulation by producing an osmolar gradient. Mannitol should be administered IV for signs of increased ICP, especially herniation. One gram per kilogram is given as a 20% solution (100 g mannitol in 500 mL 5% dextrose in water) over 10 to 20 minutes. Its therapeutic effect begins within minutes and may last several hours. It can be repeated at the same dose every 4 to 6 hours. The mechanisms of mannitol are not clearly elucidated, although animal studies suggest it decreases ICP by decreasing CSF formation rate and brain tissue water content. [23]

Loop diuretics are sometimes used in conjunction with mannitol to decrease brain water content and therefore assist in production of a negative free water balance. [4] The standard dosage is 0.5 mg/kg of furosemide IV. Use of loop diuretics is not as accepted as use of mannitol; therefore, consultation with a neurosurgeon is advised.

Corticosteroids are thought to stabilize the blood-brain barrier and reduce brain swelling. Their clear benefit in improving neurologic symptoms for patients with brain tumors has led to widespread use for this indication. The standard dosage is 10 mg dexamethasone given IV or IM, followed by 4 mg every 6 hours. The pediatric dose is 1.5 mg/kg initially (up to 10 mg), followed by 1.0 mg/kg/day. The benefits of steroid use in trauma have not been well demonstrated.

If ICP is not controlled with the above measures and surgical decompression is not imminent, many practitioners would add barbiturates in anesthetic dosages (e.g., pentobarbital 20 to 35 mg/kg bolus IV, followed by 3 to 5 mg/kg/hour IV infusion).

Newer experimental approaches to lower ICP include hypertonic saline [24] and superoxide dismutase. [25] Hypertonic saline performed as well as mannitol in a preliminary trial [24] and has the added benefit of assisting with hemorrhagic shock resuscitation. Superoxide dismutase scavenges the oxygen radical superoxide. Formation of this radical is one of the last steps leading to neuronal death after traumatic or ischemic brain injury.

**Intracranial Shunts**

Intracranial shunts are discussed in detail below. Acute shunt obstruction in the rapidly deteriorating patient is a neurosurgical emergency. Rarely, forced shunt pumping may provide temporary relief. Emergency reservoir puncture may help with distal obstructions, although unfortunately, most obstructions are proximal. As a last measure,

puncture of the entire length of the ventricular catheter may relieve the obstruction, but it will also destroy the shunt. Definitive treatment is operative shunt replacement.

**Infant Hematoma Aspiration**

Head trauma at time of delivery or within the first 6 months of life is a frequent cause of infant subdural hematoma. It likely results from disruption of the bridging veins that course to the sagittal sinus. In the majority of cases, the bleeding is bilateral and located over the dorsolateral surfaces of the frontal and parietal lobes. The bleeding may be chronic, well tolerated, and diagnosed by an excessive rate of cranial enlargement. However, bleeding may also be acute and require emergent neurosurgical intervention. In addition to the typical signs and symptoms of increased ICP, the infant may present with a bulging, tense, pulseless anterior fontanelle. If untreated, the infant may progress to transtentorial herniation. [26]

The anterior fontanelle becomes effectively closed between 9 and 18 months. Until this time it may be possible to lower increased ICP caused by a subdural hematoma in the rapidly deteriorating infant by the following tap procedure: The infant is placed supine, and excessive flexion of the neck is avoided. The scalp is shaved over the lateral margins of the anterior fontanelle. The skin is scrubbed with povidone-iodine for 10 minutes, if time allows, and then draped with sterile towels. Local anesthetic is not required but may be used. A 20-ga, 1½- or 2½-inch spinal needle is inserted through the skin at the extreme lateral limit of the anterior fontanelle where it meets the coronal suture (Fig. 64-4) (Figure Not Available) .

A zigzag puncture is used to prevent later leakage of subdural fluid. The needle is first inserted through the skin, and then the skin is moved with the needle in place before the pericranium is penetrated. The needle is then pushed through the pericranium into the subdural space. The subdural fluid is allowed to drain spontaneously, pressure is measured with a manometer, and specimens are collected for Gram staining, culture, cells, glucose, and protein. The fluid is never aspirated for fear of drawing pial vessels into the point of the needle. If a pial vessel is punctured, bleeding will usually cease spontaneously. The procedure is then repeated on the opposite side. A firm sterile dressing should then be applied. If continued leakage occurs from the puncture site, applying a collodium-impregnated cotton fluff over the puncture wound and elevating the head 20° to 30° will usually stop it.

### Complications

The use of mechanical ventilation and PEEP can increase intrathoracic pressure and cause ICP to rise. The use of mannitol requires attention to serum osmolality, which should not rise above 315 mOsm. [4] Discontinuation of mannitol may result in a rebound increase in ICP.  Infant subdural aspiration may be complicated by bleeding, infection, or intracranial vessel rupture.

## INTRACRANIAL SHUNT ASSESSMENT

**Physiology**

CSF is continuously produced by the choroid plexus and cerebral ventricles. It is partially reabsorbed by the surface of the brain and drains through the central nervous system. Hydrocephalus, which means "water head," results from an excess accumulation of CSF, usually in the ventricular system.

A wide variety of terms have been developed to describe the anatomic cause of this problem. *Communicating hydrocephalus* indicates continuity between the ventricular system and the subarachnoid spaces of the brain and spinal cord. In *noncommunicating hydrocephalus* the ventricular system or its outlets to the subarachnoid space are blocked. *Obstructive hydrocephalus* implies CSF flow blockage at any point. Since CSF overproduction is virtually never a cause of hydrocephalus, most hydrocephalus is obstructive. Furthermore, hydrocephalus can be described as acute, subacute, or chronic, indicating a time course of days, weeks, or months, respectively. [27]

Congenital and acquired disorders are most often responsible for hydrocephalus. Examples include myelomeningoceles, encephaloceles, meningitis, atriovenous malformations, traumatic hemorrhage, cysts, and calcifications. [28]

The long-term management of hydrocephalus has been revolutionized by the use of CSF shunts. Kausch first described CSF diversion to the peritoneal cavity in 1905. [29] CSF shunting has been described to such unusual sites as the bowel, thoracic duct, gallbladder, retroperitoneal space, ureter, mastoid, Stenson duct, and fallopian tube. However, ventriculovenous (VV) and ventriculoatrial (VA) shunts were the commonly used early shunts. Problems with thrombosis and obstruction due to backflow of blood in these shunts were largely overcome with the development of a one-way valve in 1952. [30] However, complications with septicemia, endocarditis, glomerulonephritis, pulmonary embolism, and a high revision rate led to the current preference for ventriculoperitoneal (VP) shunts. [31]

Early VP shunts used polyethylene tubing and were plagued by a high incidence of breakage, kinking, and obstruction secondary to foreign body reaction. [32] Silicon polymer shunts, developed in the 1960s, were more pliable, caused less tissue reaction, and were less likely to become obstructed. Most of the shunts currently encountered and used are made from this material. VP shunts also need fewer revisions for growth, since excess shunt tubing can be placed in the peritoneal cavity.

**Indications and Contraindications**

A shunt assessment, which includes physical and radiographic examination, is warranted for a myriad of patient presentations. The leading cause of shunt malfunction is mechanical failure resulting in obstructed CSF outflow. Closed fontanelles associated with sudden CSF obstruction may lead to rapid deterioration, herniation, and death. More commonly, patients have partial CSF obstruction with less dramatic symptoms. Signs and symptoms include headache; vomiting; increase in seizure frequency;

lethargy; ataxia; coma; and, for young children or infants, a change in level of activity, head size, feeding, consolability, or the presence of a full fontanelle. Presence of papilledema has been reported to occur in less than 2% of patients. [33] These symptoms may be slowly progressive over several days.

Unfortunately, these presentations are often vague and similar to many less serious conditions. The emergency physician must be aggressive in considering a shunt-related problem in any shunted patient. An understanding of the variety of shunt types and mechanisms is necessary for a safe evaluation. Shunt malfunction may be due to obstruction, disconnection, migration, equipment failure, or a variety of complications unique to the distal or proximal catheter.

## Technique

Identification of the shunt type and its components is a necessary and often difficult step in assessment. A great number of shunting systems are in use; the VP shunt is the most common encountered in clinical practice (Fig. 64-5) (Figure Not Available) . The similarity of different shunt systems should prompt a review of the patient's past medical record, operative note, and the patient's or parent's knowledge of the shunt implanted.

### Radiographic Survey: Plain Films and CT

Assessment includes a radiographic survey of the entire shunt length and may aid in shunt identification. One film may be sufficient for small children, but older patients and adults require an anteroposterior (AP)/lateral skull, chest, and supine abdominal radiographs (Figs. 64-6 through 64-9 ). Catheters are made of silicon, have a 2- to 3-mm outside diameter, and have at least 1 type of radiopaque component. Some are barium impregnated throughout, and some have radiopaque tantalum dots a fixed distance apart. Less common are catheters with metal springs fixed throughout their length and catheters with tantalum dots only at their end points (Fig. 64-10) . The ventricular catheter has multiple tip perforations to allow flow of CSF and may have flanges to decrease obstruction (Fig. 64-11) (Figure Not Available) . The distal catheter may be open or closed with slit valves (Fig. 64-12) . The slit valves are used as the sole device for flow regulation in simple systems and as adjuncts in systems with other unidirectional components. They are classified as low, medium, and high pressure to describe their variable opening pressures. The metal parts of some systems will also be visible on radiographic assessment.

Radiographic assessment of shunt malfunction includes attention to disconnection, which may occur at any point in the system. Sites of connection (Fig. 64-13) and mobility, such as the lateral neck, are at increased risk. Some portions of shunts, such as certain valves, connectors, or parts of the tubing itself, may be radiolucent and appear as disconnections. Comparison with older radiographs and a thorough knowledge of the shunt components will help avoid this erroneous conclusion. [34] Older shunts may undergo mineralization and biodegradation and become fixed by fibrous tissue. They are at greater risk for breakage. [35] The distal catheter may migrate to a wide variety of sites, [36] generally into the peritoneal cavity (Figs. 64-14 through 64-17 ). Distal catheter retraction from out of the peritoneal cavity may occur due to patient

growth.

Cranial CT scans are an important component of shunt assessment. The ventricular tips of most shunts are placed in the frontal horn of a lateral ventricle, anterior to the foramen of Monro. They generally exit through a posterior parietal burr hole, although a frontal approach is sometimes used. [37] CT may confirm appropriate shunt placement and help rule out hematoma or enlarging tumor. An increase in ventricular size (Figs. 64-18 and 64-19) or blurring of the ventricular margins suggests obstruction, although older scans from the patient may be necessary to determine a baseline ventricular size. In 1 series, 87% of patients with ventricles that were enlarged compared with prior scans had shunt malfunction, while 93% of patients with small or normal ventricles had normal shunt function. [38] However, this implies that a CT scan can sometimes appear normal or unchanged in the presence of shunt malfunction and high ICP. If a baseline CT scan is unavailable, a bicaudate distance of greater than one sixth of the brain diameter is suggestive of enlargement. [38] Other serious signs include mass effect and obliteration of the perimesencephalic cistern.

Slit-ventricle syndrome is the association of episodic signs and symptoms of raised ICP with small or slit-like ventricles. It is uncommon and occurs in only 1% to 3% of shunted children. [39]

**Shunt Identification**

Identification and further assessment may be simplified by considering the shunt as a 3-component system: a ventricular catheter, a shunting device, and a distal catheter leading to the receiving site. [31] The shunting device may consist of 1 or more components, including a reservoir, valve, filter, anti-siphon device, and pump. Older systems likely consist of a distal flow valve only. The need for cerebral flushing and instillation of drugs prompted the addition of reservoirs and double-lumen domes. These components may be attached together by nylon or metal connectors or come integrated in a single device.

A *reservoir* is a device that allows physician access to CSF or the injection of medications. [40] *Valves* are unidirectional components that regulate flow and prevent reflux. *Filters* are less common; they are used to prevent the transfer of cells to the ventricles when neoplasm is suspected or known. An *anti-siphon device* closes under negative distal pressure to negate the siphon effect and possible overdrainage associated with the upright position. The *pump* describes any shunt part that can be used to determine shunt patency or to manually move CSF through the catheter. The shunt may be a separate component with bidirectional flow or a function of the reservoir or valve unit. An easy way of classifying shunts is to consider whether the pumping chamber is a "dome" or "cylinder." [42]

**Dome devices.**

Dome devices are manufactured in many configurations and may serve as a pumping chamber, access chamber, valve, or a combination of these functions. They may lie partially over or in a burr hole, or they may be placed in close proximity to it. Some may

be used as a ventriculostomy only and have no palpable distal catheter. Most, however, are an integral part of a complete shunt system.

A classic dome device is the Pudenz flushing valve (American Heyer-Schulte, Santa Barbara). It has a valve diaphragm over the ventricular end of the catheter and can be used as a pump and a reservoir. It is surgically placed into a burr hole so that only the reservoir protrudes above the skin and can be palpated (Fig. 64-20) (Figure Not Available) .

Dome-shaped reservoirs without valves can be placed separately for a simple ventriculostomy or combined with a cylindrical valve device for use as a shunt. They were originally developed to allow drug instillation and for periodic relief of increased ICP. [40] Examples are the Rickham reservoir, the cerebral catheter reservoir, and the Ommaya reservoir (American Heyer-Schulte, Santa Barbara). The Rickham reservoir (Fig. 64-21) has a stainless steel funnel that protrudes into the burr hole and is covered with a silicon dome. It may be used alone or, more commonly, in combination with a palpable cylindrical shunt valve placed distal to the reservoir. The cerebral catheter reservoir is a 1-piece silicon reservoir and catheter (Fig. 64-22) . It can serve as a simple ventriculostomy, or it can be used in combination with a distal valve and serve as the proximal portion of a shunt. The larger Ommaya (Fig. 64-23) (Figure Not Available) is a 1-piece silicon device used as a ventriculostomy, or, with a side arm, for continuous shunting. All 3 of these reservoirs may be confused with the Pudenz valve.

Other dome devices may be placed near, but not over, the burr hole. Examples include Delta valves and CSF flow control valves (Pudenz-Schulte Medical Corporation, Goleta, Calif). The tantalum markings on these domes (Figs. 64-24 (Figure Not Available) and 64-25) (Figure Not Available) indicate flow direction and opening pressures. The Delta valve serves to maintain a near-constant pressure gradient, regardless of patient position. It incorporates a device that automatically adjusts for the hydrostatic pressure difference between the ventricles and the peritoneal cavity. [43]

The Accu-Flo system (Codman and Shurtleff, Inc., Randolph, Mass) includes 4 types of reservoirs made from 1-piece silicon with polypropylene bases. It includes both single domes and double domes (Fig. 64-26) .

One of the least complex systems is the Uni-Shunt (Codman and Shurtleff, Inc., Randolph, Mass), which is a continuous catheter from the ventricular end to the distal slit valve (Fig. 64-27) . Since it has no connectors, it is less prone to disconnection. It is barium impregnated throughout. The Uni-Shunt is manufactured with or without a double-dome reservoir for pumping and CSF access (Fig. 64-28) . The catheter's distal slit valves serve to regulate flow.

The Multi-Purpose Valve (American Heyer-Schulte) incorporates an on-off device that is controlled percutaneously. [42] Its single unit includes a proximal occluder, dome reservoir, on-off switch, and an anti-siphon device.

Anti-siphon devices (Fig. 64-29) (Figure Not Available) may also be found as separate units. [44] They have both flat and round components and have a thin membrane

covering their dome. Shunt filters (see Fig. 64-22) are silicon cased and designed to prevent transfer of cells. They may be used when malignancy is known or suspected. In this case, they are commonly placed just behind the mastoid air cells in order to be exposed to radiotherapy.

**Cylinder devices.**

Cylinders are of variable sizes and since 1970 have been placed distal to a reservoir. Prior to this, reservoirs were rarely attached. [42]

The Holter valve system (Codman and Shurtleff, Inc., Randolph, Mass) incorporates 2 silicone slit valves encased in stainless steel bodies. The bodies are connected by a silicone tube that serves as the pumping chamber (see Fig. 64-21 and Fig. 64-30) (Figure Not Available) . It is commonly placed just distal to a Rickham reservoir.

The Denver shunt (Codman and Shurtleff, Inc., Randolph, Mass) incorporates a Rickham reservoir with a valved cylindrical pump. It is manufactured as 1 inseparable unit (Fig. 64-31) or with the reservoir and pump as separate units.

The Hakim shunt (Cordis Corporation, Miami) incorporates a cylindrical valve unit with a rectangular reservoir (Fig. 64-32) (Figure Not Available) . The reservoir includes a metal plate visible on radiographs. It is longer and thinner than the Holter.

**Shunt Palpation**

Continuity of a shunt may be assessed by palpation throughout its length. Most shunts are placed through posterior parietal burr holes, although a few are placed in the frontal area. The feel of the reservoir or pump may assist in identifying the shunt type.

Disconnections may be suspected by palpation. However, fibrous tracts may feel, and even perform, like shunt tubing. Single-dome devices may be difficult to palpate due to fibrous or bony encasement. Hardening of the plastic after placement has also been described. [45]

**Assessing Shunt Patency**

Patency of a single-dome device may be determined with transcutaneous digital pressure. An understanding of how CSF may flow through a particular system is necessary to interpret the results.

A valveless dome (e.g., Ommaya or Rickham without an attached Holter valve) is usually attached to a distal catheter with slit valves. Easy depression of the dome while occluding the distal catheter suggests proximal patency, whereas difficult depression suggests a proximal obstruction. Lack of prompt refilling suggests poor CSF flow toward the dome. If distal occlusion is not held, CSF will flow both proximally and distally through the path of least resistance. [42] In this case, a firm, noncompressible dome

suggests both proximal and distal occlusion.

For a dome with a valve (e.g., Pudenz), lack of easy depression suggests distal obstruction, and lack of prompt refilling suggests proximal obstruction. For double domes (e.g., Uni-Shunt with reservoir, Accu-Flo), while digital occlusive pressure is held over the proximal dome, easy distal dome pumping implies distal patency. After applying digital occlusive pressure over the distal dome, prompt refilling of the proximal dome when pressure is released implies proximal patency. The multipurpose valve contains an on-off switch composed of a silicon dome containing a tantalum-impregnated ball. Digital pressure over the dome forces the ball into a cup at the dome's base and prevents distal flow. The valve can be reopened by applying pressure over the proximal occluder and pumping the reservoir. Patency is determined by applying pressure over the proximal occluder and then pressing on the reservoir. Prompt emptying suggests distal patency. When releasing pressure off the occluder, prompt refilling suggests proximal patency.

Cylinder devices (e.g., Holter, Hakim) are tested by digital pressure on the tube. Difficult depression suggests distal obstruction, while delayed filling suggests proximal obstruction.

Acute obstruction with deterioration is a neurosurgical emergency. The role of the emergency physician consists of diagnosis, supportive care, and consultation. Forced pumping may be attempted but provides only temporary relief in a minority of cases. [31] [37]

Anti-siphon devices and filters are not designed for pumping or determination of shunt patency.

### Complications

Pumping of the shunt should not be done repeatedly unless forward flow of CSF is desired for therapeutic purposes. Repeated pumping may lead to breakage of the shunt or shunt mechanism, as well as the possibility of pumping CSF retrograde into the ventricle. Not all shunt obstructions may be determined by simple digital pressure or a normally functioning pumping chamber. However, information on this maneuver is an important component of overall shunt assessment.

## CULTURING INTRACRANIAL SHUNTS

The second most common cause of shunt malfunction is infection, which unfortunately carries a 30% to 40% mortality rate. [46] Reported incidence rates vary widely, from 2.6% to 38%, with an average of 8% to 10%. VA shunts have a slightly higher rate of infection, approximately 15%. [51]

Shunt infection is generally considered a complication of surgery, since 70% are diagnosed within the first month after surgery and 90% within 6 months. Because of this, there may be reluctance to associate minor signs and symptoms of shunt infection

with actual infection after 6 months. A variety of operative antibiotic regimens, accessory equipment, and surgical techniques have been explored to prevent shunt infection. [52]

Increased infection rates have been reported for patients with Dandy-Walker syndrome, aqueductal stenosis, or encephaloceles [46] ; premature infants [53] ; newborns, as compared with adults [54] ; patients with successive shunt revisions or taps; and those who have undergone laparotomy. Transient bacteremia has not been shown to cause shunt infection. [55]

Shunt infections may be internal, describing colonization on the inner surface of the shunt with or without ventriculitis. External shunt infections describe wound infections around the shunt secondary to the operation or erosion of the overlying skin. Infection may follow perforation of the abdominal contents by the distal catheter.

Coagulase-negative staphylococci are responsible for the majority of internal shunt infections; *Staphylococcus epidermidis* is the most common organism. Almost every other bacterial species has reportedly caused infection at one time, although *Staphylococcus aureus*, diphtheroids, gram-negative enterics, and *Candida* are more frequently noted. Infections resulting from *Haemophilus, Streptococcus pneumoniae* (pneumococcus), and *Neisseria meningitidis* (meningococcus) typically present late. It has been suggested that these infections often are not shunt related but probably represent meningitis in a patient who happens to have a shunt. [56]

**Indications and Contraindications**

Unfortunately, the clinical presentation of a shunt infection is nonspecific. Fever, change in sensorium, emesis, abdominal pain, diarrhea, peritonitis, irritability, and signs of shunt malfunction predominate. Meningeal signs are present in only 25% to 33% of patients. Local shunt tract inflammation may occasionally occur. [58] VA shunts may be complicated by septic emboli or shunt nephritis. [59] In addition, patients with infected VA shunts may remain apparently well for months before symptoms of septicemia become apparent. In contrast, VP shunt infections rarely present after 6 months. Often patients with VP shunt infections present with symptoms attributable to distal shunt obstruction caused by infected CSF being discharged into the peritoneal cavity and forming a cyst or adhering to omentum. [60] One series described 23 patients with gram-negative VP shunt infections as appearing relatively well at presentation. [61] Of these infections, 87% occurred within 4 weeks of shunt placement or revision, and the most common symptoms were fever, lethargy, and irritability.

In the unfortunate event that a shunt needs to be tapped but has no reservoir for access (e.g., Uni-Shunt), a lumbar puncture can be performed. However, this procedure may not be diagnostic for ventriculitis or infection confined to the shunt--that is, CSF from a lumbar puncture is frequently normal in the early stages. [50] The patient without a shunt access site is rarely encountered in current practice.

Because shunt tapping may introduce organisms into the central nervous system, contraindications include inexperience with the procedure and infection overlying the tap site. It is prudent to discuss the indications for the procedure with the patient's

neurosurgeon before initiating the procedure.

## Equipment

All equipment should be set up and sterilely prepared before initiating the procedure. A 25-ga needle, commonly a butterfly, is used to perform the actual puncture. Other equipment needed includes gloves, preparation and drape, anesthesia, an 18-ga needle to nick the scalp, a CSF manometer, and CSF collection tubes. The standard lumbar puncture tray could be used, with the addition of the appropriate needles.

## Technique

After the procedure is discussed with the patient (and parents, when appropriate), written informed consent is desirable. The procedure should not be particularly painful, although the sting of local anesthesia needs to be addressed.

Determine the appropriate reservoir site by palpation and radiographic assessment. *Do not puncture anti-siphon devices, filters, and Holter or Hakim valves.*

Place the patient prone and restrain him or her, if needed. Shave the scalp, if necessary, overlying the device. Meticulous attention should be paid to a standard sterile preparation and drape technique. When povidone-iodine is used for skin preparation, give the solution time to fully dry (1 to 2 minutes) to maximize its bactericidal effect. Provide anesthesia at the site with an appropriate local anesthetic. Some may elect to omit anesthesia, since its application may be as uncomfortable as the actual tap.

Nick the scalp with an 18-ga sterile needle. Puncture the reservoir with a short 25-ga butterfly needle (Fig. 64-33) . The angle of puncture should be approximately 25° to 30° to avoid placing the needle too deep and potentially damaging the reservoir floor. Many reservoirs have silicon or metal needle guards to avoid this problem. An exception is the Hakim reservoir, which may be entered at almost any angle. [31]

Watch for passive appearance of fluid, attach the manometer, and record the pressure obtained. Allow only passive withdrawal of 4 to 6 mL of CSF. Place the CSF in sterile tubes, and send for standard CSF analysis. Cell count with differential, protein and glucose levels, culture, and Gram stain are routinely desired. Other studies may be appropriate in specific clinical circumstances (see Chapter 65) .

## Complications

Puncturing anti-siphon devices, filters, and Holter or Hakim valves may result in damage to or leakage of the shunt; these structures are not designed for sampling. Infection may be introduced by the tap itself. Since this may result in increased morbidity or mortality, a thorough sterile preparation and drape is emphasized.

**Interpretation**

Assessment of CSF studies should follow standard practice (see Chapter 65) . Shunt-aspirated CSF has a reported culture sensitivity that ranges from 62% to nearly 100%.  Data on the sensitivity of the Gram stain are lacking, although generally the association with infection is considered high. [63] In 1 study, shunt-obtained CSF glucose and protein were abnormal in 18% and 66% of cases, respectively. [63] Abnormal cell counts, defined as >2 neutrophils or >10 total white blood cells per milliliter, were present in 65% of infected cases. [63] Administration of antibiotics prior to the procedure will decrease culture sensitivity.

## CONCLUSION

Increased ICP may be manifested in many ways. If unrecognized and untreated, it may lead to brain death through cerebral hypoxia or direct tissue damage. The emergency physician has several techniques to combat this problem while awaiting definitive neurosurgical treatment.

Intracranial shunts and care of shunt patients continue to improve. Emergency physicians can expect more of these patients to present with problems possibly attributable to this life-saving device. The treating physician needs to be adept at basic shunt identification, assessment of patency, and CSF sampling.

# Chapter 65 - Spinal Puncture and Cerebrospinal Fluid Examination

**Jon C. Kooiker**

Cerebrospinal fluid (CSF) examination is performed in an emergency setting for the purpose of obtaining information relevant to the diagnosis and treatment of specific disease entities. Many urgent and life-threatening conditions require immediate and accurate knowledge of the nature of the CSF. However, certain harmful consequences may result from a spinal puncture. The procedure should follow a careful neurologic examination with thought given to the risks and merits of the procedure in each given situation.

In 1885, Corning punctured the subarachnoid space to introduce cocaine anesthesia into a living patient. [1] Quincke (1891) first removed CSF in a diagnostic study and introduced the use of a stylet. [2] He studied cellular contents and measured protein and glucose levels. Quincke was also the first to record pressure with a manometer. Subsequently, increasingly sophisticated bacteriologic, biochemical, cytologic, and serologic techniques were introduced. In 1918, Dandy replaced CSF with air to determine normal brain anatomy and changes that would indicate disease. [3] Water-soluble contrast media have been used to delineate the spinal subarachnoid space and cerebral cisterns. Other uses of the spinal puncture include injection of anesthetic agents, chemotherapeutic agents, and antibiotics and drainage of fluids.

## ANATOMY OF CSF FORMATION AND CIRCULATION

In the adult, approximately 140 mL of the spinal and cranial cavities is occupied by CSF, with approximately 30 mL in the spinal canal. This volume is the result of a balance between continuous secretion (primarily by the ventricular choroid plexus) and absorption into the venous system (chiefly by way of the arachnoid villi). After formation, the fluid passes out of the ventricles by way of the midline dorsal foramen of Luschka and the lateral ventral foramina of Magendie. The fluid then flows into the spinal subarachnoid space, the basilar cisterns, and the cerebral subarachnoid space. Production is approximately 0.35 mL/min, and CSF ventricular production is such that there is a net flow out of the ventricles of 50 to 100 mL/day. Thus the usual volume of CSF removed at lumbar puncture is regenerated in 1 hour.

Cerebrospinal fluid may have an embryologic nutritive function; at maturity the CSF most likely acts as a mechanical barrier between the soft brain and the rigid fibro-osseous dura, skull, and vertebral column. It also appears to support the weight of the brain. [4] When buoyed by CSF, the functional brain weight is reduced from 1400 to 50 g. Contraction and expansion of the CSF may accommodate changes in brain volume. Additional functions, including intracerebral transport and maintenance of a stable chemical environment of the central nervous system (CNS), have been reviewed by Fishman.

## INDICATIONS FOR SPINAL PUNCTURE

In recent years, the indications for spinal puncture have been reduced with the introduction of new noninvasive diagnostic procedures--magnetic resonance imaging (MRI) and computed transaxial tomography (CT). A few clinical situations require an early, or even an emergent, spinal puncture. The primary indication for an emergent spinal tap is the possibility of CNS infection, with the exception of brain abscess or a parameningeal process. The need for early detection of meningitis results in performance of many more lumbar punctures than ultimate diagnoses of infection. [6] No other method can be used to completely exclude the diagnosis of meningitis.

CSF generally should be examined for evidence of an infection in patients with a fever of unknown origin, especially if an alteration of consciousness or impaired immune system is present, even in the absence of meningeal irritation. *Meningeal signs need not be present in patients who are old, debilitated, immunosuppressed, or receiving anti-inflammatory drugs or who have had partial treatment with antibiotics.* In a newborn, even a fever is not a dependable sign; temperatures may be normal or even subnormal. A high index of suspicion is required when evaluating infants younger than 12 months of age. Approximately 25% of infants with meningitis will not have nuchal rigidity, but most will usually appear toxic or moribund. A tense and bulging fontanel is somewhat more reliable, although this sign may be absent in a dehydrated child. Neonatal meningitis occurs in 25% of sepsis cases. In addition, 15 to 20% of infants with meningitis will have negative blood cultures. [9] [10] In a child between the ages of 1 month and 3 years, fever, irritability, and vomiting are the most common symptoms of meningitis. Typically, handling is painful for the child, and the child cannot be comforted. In addition, the older child may complain of a headache. In all ages, the patient generally looks unusually ill and appears drowsy with a dulled sensorium.

Physical signs become more useful in diagnosing meningitis in children older than 3 years of age. [11] These include nuchal rigidity, Kernig's sign (efforts to extend the knee are resisted), and Brudzinski's sign (passive flexion of one hip causes the other leg to rise, and efforts to flex the neck make the knees come up). A useful aid in distinguishing neck rigidity of meningeal origin from that caused by primary pain in the cervical muscles and the soft tissues is the usual preservation of lateral movement in meningeal irritation. A petechial rash in a febrile child should also raise the possibility of *Neisseria* meningitis. [12] Prior use of antimicrobial agents may modify the clinical presentation and alter CSF findings; partially treated children are less likely to be febrile or exhibit an altered mental status.

The second indication for an emergent spinal puncture is a suspected spontaneous subarachnoid hemorrhage. The diagnosis will usually be made by head CT scan or by the finding of blood in the CSF. [13] CT sensitivity drops in patients presenting later than 24 hours after the event to 76% after 48 hours. From 20 to 60% of aneurysmal subarachnoid hemorrhage will have "sentinel thunderclap" or "warning leak" headache before a major hemorrhage. This consists of an unusual sudden headache without nuchal rigidity caused by a "minor" leak of blood from the aneurysm and may precede major rupture by 10 to 320 days. [14] [15] Surgical treatment before a major hemorrhage is

the goal of early clinical recognition. After a "warning leak," the head CT scan is usually negative, giving added importance to the performance of a lumbar puncture. Migraine or "vascular" headache is a common misdiagnosis in patients with a subarachnoid hemorrhage who have an initially negative head CT scan.

The usual clinical picture of a subarachnoid hemorrhage is a severe and instantaneous excruciating headache. Patients will usually recall the exact moment the headache occurred. The location of the headache is variable and does not give a clue as to the site of hemorrhage. Nausea, vomiting, and prostration are common symptoms, with approximately one third of patients becoming unconscious at the onset. Examination shows an acutely ill patient with irritability or overt altered mental status. Meningeal signs are commonly present at the time of the initial examination and usually develop in all cases within 2 to 3 days. Meningeal signs may become more severe during the first week after hemorrhage and correspond to the breakdown of blood in the CSF. During the first week, many patients are febrile, reflecting a chemical hemic meningitis. [16] Failure to detect blood radiographically in an awake patient may indicate a small hemorrhage or a predominant basal accumulation of blood. If a patient is seen several days after the hemorrhage, the blood may have become isodense with brain and may no longer be visible on a CT scan. The proper diagnosis would then require spinal puncture. Because 2 to 10% of acute subarachnoid hemorrhages will not be detected by the initial CT scan (even with modern third generation scanners), a lumbar puncture is appropriate to rule out the diagnosis with certainty. In theory, a small subarachnoid hemorrhage may take several hours to reach the lumbar region. Thus, it is possible that such a patient might have normal lumbar CSF if examined soon after rupture. A second, delayed lumbar puncture may occasionally be required for diagnosis in such a situation. If the neurologic picture demonstrates localizing findings, the presence of a large intracranial hematoma should be suspected, and spinal puncture is contraindicated until CT (or arteriography) delineates the nature of the lesion.

In a patient with suspected cerebral embolus or evolving infarction, either a negative CT scan or clear CSF should be obtained before the use of anticoagulants. This seems particularly applicable if neurologic deficits or an altered mental state persists. [21]

The emergency physician may be called on to perform a therapeutic lumbar puncture in the patient with known pseudotumor cerebri (idiopathic intracranial hypertension). The patient has elevated intracranial pressure (ICP), headache, and papilledema. The headache worsens with maneuvers that increase the ICP (e.g., Valsalva, squatting, bending, coughing). The symptomatic patient may have an ICP of 250 to 400 mm $H_2O$.

Other nonemergent reasons for CSF examination include evaluation of CNS syphilis, unexplained seizures, instillation of chemotherapy and positive contrast agents, evaluation of suspected demyelinating or inflammatory CNS process, and treatment of headache from subarachnoid hemorrhage or benign intracranial hypertension. [22] Carcinomatous meningitis and suspected spinal cord compression from metastatic disease may require spinal puncture for myelography and cytologic examination. The availability of MRI is a suitable alternative for identifying compressive myelopathies.

# CONTRAINDICATIONS FOR SPINAL PUNCTURE

Spinal puncture is absolutely contraindicated in the presence of infection in the tissues near the puncture site. [4] [5] Spinal puncture is relatively contraindicated in the presence of increased ICP from a space-occupying lesion. Caution is particularly advised when lateralizing signs (hemiparesis) or signs of uncal herniation (unilateral third nerve palsy with altered level of consciousness) are present. In such cases, a tentorial or cerebellar pressure cone may be precipitated or aggravated by the spinal puncture. Cardiorespiratory collapse, stupor, seizures, and sudden death may occur when pressure is reduced in the spinal canal. [22]

The risk of herniation seems to be particularly pronounced in patients with brain abscess. Brain abscesses frequently occur as expanding intracranial lesions with headache, mental disturbances, and focal neurologic signs rather than as infectious processes with signs of meningeal irritation. In 75% of cases, a primary source of chronic suppuration is present. Common predisposing causes of brain abscess include craniofacial trauma, craniocerebral trauma, penetrating injuries with bone fragments in brain, transmitted foreign objects or large animal bites of infant skulls, postneurosurgical procedures, cardiovascular disorders with right to left shunts, bacterial endocarditis, gram-negative sepsis in neonates, dental infections, chronic sinusitis, otitis, mastoiditis, chronic abdominal pulmonary or pelvic infections, bacterial meningitis, and immunosuppression. Infarcted brain tissue may develop abscesses in the presence of sepsis because of a compromised blood-brain barrier. [25] Although the CSF is usually abnormal (elevated pressure, elevated white blood cell count, and elevated protein concentration), spinal puncture in patients with a possible abscess is contraindicated in most cases. Five of Samson and Clark's 22 patients exhibited signs of midbrain compression within 2 hours of lumbar puncture. [24] Evidence of herniation markedly reduces the patient's chances for survival.

Brain abscess may spontaneously rupture into the ventricular system producing ventriculitis and meningitis. If the history suggests possible brain abscess, CT can rapidly diagnose and localize the lesion. [26] Because the appearance of brain abscesses on CT is similar to that of neoplastic and vascular lesions, false-positive reports of brain abscess may be encountered. [27]

Trauma to the dural or arachnoid vessels may create minor hemorrhage into the CSF. This generally is of little consequence. However, the number of patients with hemophilia and human immunodeficiency virus (HIV) infection who require lumbar puncture has increased in the past decade. Spinal epidural hematomas may occur but are rare complications of lumbar puncture in individuals receiving anticoagulant therapy or in patients with disease associated with abnormal clotting mechanisms, especially thrombocytopenia. Edelson and colleagues reviewed over 100 cases of spinal epidural hematoma; approximately one third were associated with anticoagulant therapy. [28] Most articles describe isolated cases. Spinal subdural hematomas after lumbar puncture are even more rare than epidural hematomas. When a patient is anticoagulated or has a coagulopathy, the tap should be performed by experienced physicians, who are less likely to traumatize the dura. The patient should be carefully

followed for progressive back pain, lower extremity motor and sensory deficits, and sphincter impairment after the procedure. *Complaints of motor weakness, sensory loss, or incontinence after lumbar puncture should be thoroughly investigated.* Lumbar puncture may be performed in the presence of a coagulation defect if the procedure is expected to provide essential information, such as in the diagnosis of meningitis. In cases of severe thrombocytopenia, the infusion of platelets before the lumbar puncture may be desirable.

The infusion of clotting factors in the hemophiliac patient and normalization of the prothrombin time with fresh frozen plasma in the anticoagulated patient are desirable if the clinical situation permits such delay before performing a lumbar puncture. Because as many as 90% of patients with severe hemophilia are seropositive for HIV, the issue of performing a lumbar puncture in patients with coagulopathies is an increasingly common phenomenon. Silverman and colleagues [31] have demonstrated the safety of lumbar puncture in patients with hemophilia A or B who had their deficit clotting factor replaced before the procedure. In their series of 33 patients (30 with <1% normal factor level) who underwent a total of 52 spinal taps after specific factor replacement, no serious procedure-related complications were identified. Their protocol was to attain an immediate postinfusion factor level between 5% and 100%. Use of additional factor replacement after lumbar puncture is of unknown value.

If the history and physical examination suggest a treatable illness, such as meningitis or subarachnoid hemorrhage, then the physician may perform a spinal puncture after careful consideration of the entire clinical picture. In all cases, the study should be undertaken after careful thought regarding how the results will assist in patient evaluation and treatment. It is unlikely that the spinal puncture will beneficially alter management in the presence of a neoplasm, a cranial hematoma, an abscess, a completed nonembolic infarction, or cranial trauma.

## EQUIPMENT

Standard equipment to be assembled before a spinal puncture include spinal needles (generally 22 ga or smaller bore and up to 32 in. in length--longer needles may be needed for some obese patients); three-way stopcock; manometer; connecting tubing (optional, to connect needle with stopcock and manometer); 4 specimen tubes; local anesthetic; syringes and needles for local anesthesia (appropriate for both skin and subcutaneous tissue infiltration); sterile drapes, gauze, sponges, and gloves; and an antiseptic solution for skin preparation (e.g., 0.5% chlorhexidine in alcohol or 1% povidone-iodine). [32]

Some operators prefer to use an "atraumatic standard Sprotte needle" to minimize the dural injury associated with needle passage. These styletted needles (Havel's Inc., Cincinnati) have a side port for fluid withdrawal and theoretically are more likely to *separate* rather than core the dural tissue.

Although commercial kits provide most of the items (Fig. 65-1) (Figure Not Available) , it is important for the operator to bring additional supplies, including supplemental spinal needles, gauze and antiseptic solution, additional local anesthesia and

needles/syringes, and extra sterile gloves of the appropriate size. Following this advice will minimize procedural interruptions when material on the commercial kit must be supplemented.

## TECHNIQUE

Lumbar puncture is carried out with the patient in the lateral recumbent position. A line connecting the posterior-superior iliac crests will intersect the midline at approximately the L4 spinous process (Fig. 65-2) (Figure Not Available) . Spinal needles entering the subarachnoid space at this point are well below the termination of the spinal cord, and the only important neurologic structure is the cauda equina. Generally, the needle will push isolated nerves to the side during advancement. The adjacent interspace above or below may be used, depending on which area appears to be most open to palpation. The space between lumbar vertebrae is relatively wide. In the thoracic region, the spinous processes overlap and are directed caudad, and therefore there is no midline area free of overlying bone. In the adult, the spinal cord extends to the lower level of L1 or the body of L2 in 31% of persons, thus eliminating higher levels as sites for puncture. The puncture in adults and in older children may be performed from the L2 to L3 interspace to the L5 to S1 interspace. Developmentally, the spinal canal and the spinal cord are of equal length in the fetus. Growth of the cord does not keep pace with longitudinal growth of the spinal canal. At birth, the cord ends at the level of the L3 vertebra. The needle in infants should be placed at the L4 to L5 or L5 to S1 interspaces. The subarachnoid space extends to an S2 vertebral level; however, the overlying bony mass prevents entry into this lowermost portion of the subarachnoid space.

Almost all patients are afraid of a spinal puncture because they have heard stories of severe complications. Explaining the procedure in advance and discussing each step during the course of the test aids in reducing patient tension and helps the physician. The physician should inquire about history of allergies to local anesthetic agents and topical antiseptics. A standard informed consent form or detailed procedural note is recommended to document the process of patient/guardian education regarding the indications, procedural techniques, risks/benefits and alternatives to the procedure, and the patient/guardian's assent to the procedure.  This step may need to be abridged when the patient is critically ill or eliminated when the patient is mentally incapacitated and no guardian is present. In anxious patients, a benzodiazepine agent (orally or parenterally; e.g., midazolam, 0.05 to 0.075 mg/kg IV) will facilitate the procedure.

The next important step is positioning of the patient. Generally the procedure is performed in the lateral decubitus position in older children and adults. The patient is given a pillow to keep the head in the same plane as the vertebral axis. The shoulders and the hips are positioned perpendicular with the table. A firm table or bed is desirable whenever possible. Flexion of the neck does *not* facilitate the procedure to any great extent; and because severe flexion may add to the patient's discomfort, this step may be omitted. The patient's lower back should be arched toward the physician. Some physicians place the patient in an upright sitting position, because the midline is more easily identified when the patient is sitting. The higher CSF hydrostatic pressure in a sitting, dehydrated patient may aid CSF flow. Caution regarding orthostatic blood pressure changes and airway maintenance must be observed when the patient is sitting

for the procedure. Generally the sitting patient is allowed to lean onto a bedside stand using a pillow to rest the head and arms. An assistant also must help support the patient during the procedure.

Sterile gloves must be used. The examiner should wash the patient's back with an antiseptic solution applied in a circular motion. The circumference of the cleansed area should increase with each motion. A povidone-iodine solution will decrease skin flora to a bacterial count of 0 to 2 bacteria/sq in. [33] Friction during skin preparation helps remove loose debris. The patient should be warned that the solution will be cold. The excess fluid is removed with a dry sterile gauze pad. A sterile towel is placed between the patient's hip and the bed. Commercial trays have a second sterile drape with a hole that may be centered over the site selected for the tap.

The skin and deeper subcutaneous tissue are infiltrated with local anesthetic (1% lidocaine). Buffered or warmed lidocaine is preferred (see Chapter 31) . The patient should be warned about transient discomfort from the anesthetic. Anesthetizing the deeper subcutaneous tissue significantly reduces the procedural discomfort. Merely raising a skin wheal is insufficient anesthesia. Some operators not only anesthetize the interspinous ligament but also apply local anesthesia in a vertically fanning distribution on both sides of the spinous processes near the lamina. This field block on each side of the spinous processes anesthetizes the recurrent spinal nerves that innervate the interspinous ligaments and muscles.

While waiting for the anesthetic to take effect, the physician should attach the stopcock and manometer and see that the valve is working. A 3.5-in., 20-ga needle should be used in adults, and a 2.5-in., 22-ga needle should be used in children (a 1.5-in., 22-ga needle is available for infants). A needle of this size has enough rigidity to allow the procedure to be accomplished easily but makes less of a dural tear than do larger instruments. The patient should be told to report any pain and should be informed that he or she will feel some pressure.

The needle is placed into the skin in the midline parallel to the bed. The needle is held between both thumbs and index fingers. After the subcutaneous tissue has been penetrated, the needle is angled toward the umbilicus. The bevel of the needle should be facing laterally. It has been speculated that pointing the bevel laterally may allow the needle to separate the transverse fibers of the dura rather than cut through them, allowing for less CSF leakage after the needle has been withdrawn.

The supraspinal ligament connects the spinous process; the interspinal ligaments join the inferior and superior borders of adjacent spinous processes. The ligamentum flavum is a strong, elastic, yellow membrane that may reach a thickness of 1 cm in the lumbar region. The ligamentum flavum covers the interlaminar space between the vertebrae and functions to assist the paraspinous muscles in maintaining an upright posture (Fig. 65-3) (Figure Not Available) . The ligaments are stretched in a flexed position and are more easily crossed by the needle. The ligaments offer resistance to the needle, and a "pop" is often felt as they are penetrated. One should remove the stylet frequently to see if the subarachnoid space has been reached. The "pop" is occasionally not felt with the

very sharp needles contained in disposable trays.

If bone is encountered, the needle must be partially withdrawn to the subcutaneous tissue. The physician should repalpate the back and ascertain that the needle is in the midline. If bone is again encountered, the needle should be slightly withdrawn and re-angled, with the point placed so that it angles more sharply cephalad. This should avoid hitting the inferior spinous process.

Clear fluid will flow from the needle when the subarachnoid space has been penetrated. The physician should attach the manometer and record the opening pressure. Pressure readings are only valid if taken with the patient in the lateral decubitus position. A three-way stopcock is supplied in disposable trays; this allows both collection and pressure to be measured by a single needle (Fig. 65-4) (Figure Not Available) . Positioning of the manometer is often more convenient if an extension tube (provided with most disposable trays) connects the needle hub to the stopcock, which is in turn attached to the manometer. The manometer should be positioned so that the "zero" mark is at the level of the spinal needle. The patient is then asked to relax and to extend his or her legs to decrease intra-abdominal pressure. The fluid column is observed for phasic changes with respirations and arterial pulsations. This ensures placement in the subarachnoid space. If the needle is against a nerve root or is only partially within the dura, the pressure may be falsely low, and respiratory excursions will not be seen in the manometer. Minor rotation of the needle may solve these problems. Hyperventilation to relax the patient should not be attempted, because this will reduce the pressure readings owing to hypocapnia and resultant cerebral vasoconstriction.

After measuring the pressure, the physician should turn the stopcock and collect enough fluid to perform all desired studies. Even if the pressure is elevated, sufficient fluid should be removed for performance of all indicated studies, because the risk of the procedure involves the dural rent and not the amount of fluid initially removed. Presumably, more fluid will be lost subsequently through the hole in the dura. A dressing is placed over the puncture site. Commercial trays supply 4 specimen tubes. Tube 1 is used for determining protein and glucose levels and for electrophoretic studies; tube 2 is used for microbiologic and cytologic studies; tube 3 is for cell counts and serologic tests for syphilis. In the presence of bloody CSF, cell counts should be performed in tubes 1 and 3 to help differentiate traumatic taps. One may compare water placed in tube 4 with CSF in tube 3 to detect cloudiness or discoloration. Alternatively, tube 4 can be stored under refrigeration in the laboratory for subsequent studies.

Traumatic taps can be avoided by proper patient and needle positioning. A traumatic tap most commonly occurs when the subarachnoid space is transfixed at the entrance of the ventral epidural space, where the venous plexus is heavier. A plexus of veins forms a ring around the cord, and these veins may be entered if the needle is advanced too far ventrally or is directed laterally (Fig. 65-5) (Figure Not Available) . If blood is encountered and the fluid does not clear, the procedure should be repeated at a higher interspace with a fresh needle. A traumatic tap, per se, is not a particularly dangerous problem in the patient with normal coagulation, and no specific precautions are needed if blood-tinged fluid is obtained. However, observation for signs of cord or spinal nerve

compression from a developing hematoma should be routine for these patients.

**Lateral Approach in Lumbar Puncture**

The supraspinal ligament may be calcified in older persons, making a midline perforation difficult. A calcified ligament may deflect the needle. In this case, a slightly lateral approach may be used. As the lower lamina rises upward from the midline, the needle is directed slightly cephalad to miss the lamina and slightly medially to compensate for the lateral approach. The needle passes through the skin, superficial fascia, fat, the dense posterior layer of thoracolumbar fascia, and the erector spinae muscles. The needle then penetrates the ligamentum flavum (bypassing the supraspinal and interspinal ligaments), the epidural space, and the dura before CSF is obtained (Fig. 65-6) (Figure Not Available) .

**Cisternal Puncture**

In situations in which lumbar puncture is contraindicated (such as local infection or acute trauma to the lumbar spine), cisternal, or suboccipital, puncture is the usual alternative. Technical problems, such as morbid obesity, cord tumor, arachnoiditis, bony deformities, or prior spinal surgery (fusion), may make lumbar puncture impossible. Contrast material may be injected into the cisterna magna to identify the rostral extent of an obstructing lesion identified by lumbar myelography.

The patient is cleaned and anesthetized in a manner similar to that for a lumbar puncture after the neck has been shaved from the external occipital protuberance to the mastoid process laterally. The patient is preferably placed in a lateral decubitus position, but a sitting position can be used. A pillow is placed under the head to keep the neck and the vertebral axis in the same plane. The patient's neck is flexed to the chest. The spinal needle is placed in the midline halfway between the spinous process of C2 and the inferior occiput. The needle is angled cephalad through the subcutaneous tissue until it comes in contact with the bony occiput. The needle is then withdrawn and subsequently advanced at a less acute angle with the horizontal plane of the cervical spine. This is repeated until the dural "pop" is felt. As in the lumbar region, the stylet should be removed frequently so that the dura is not punctured unknowingly. Fluid is removed in the usual manner. Dural veins are less extensive, and bloody taps are less common. Low-pressure headaches are less common, presumably because the subarachnoid pressure is lower and the dural tear can heal faster.

**Lateral Cervical Puncture**

The patient is placed in a supine position and fully sterilized and anesthetized. A 20-ga lumbar puncture needle is inserted perpendicular to the neck and parallel to the bed. The landmark for insertion is a point 1 cm inferior and 1 cm dorsal to the mastoid process. The physician frequently removes the stylet to check for fluid return and, as at other sites, advances the needle slowly. If the needle goes too deeply and encounters paraspinous muscles, it is probably too deep posteriorly and should be repositioned more anteriorly. If bone is encountered, more dorsal placement is needed. Pressure and

fluid samples are collected, as in other sites. [34]

The contraindications to cisternal and cervical punctures are the same as those to lumbar puncture. Both techniques are easily mastered, but prior demonstration of the procedure by an experienced neurologist or neurosurgeon is advised. When lumbar puncture cannot be performed for technical reasons and meningitis is suspected, then placement of the needle under fluoroscopy may help obtain CSF.

**Lumbar Puncture in Infants**

Lumbar puncture in infants is usually performed to exclude meningitis. The sitting position may allow the midline to be more easily identified. Some authors use a nonstyleted needle in small infants, because this device allows the pressure to be estimated as the needle punctures the dura. [35] However, the failure to use a stylet may be the source of later development of an intraspinal epidermoid tumor. [36] A technique of lumbar puncture in the neonate using a butterfly infusion set needle has been described as a simplified procedure that may be useful in the squirming or hyperactive patient. [35]

If the child's neck is very tightly flexed, CSF may not be obtained. However, if the head is held in midflexion, CSF usually flows briskly. Prolonged severe flexion of the neck in an infant should be avoided because it may produce dangerous airway obstruction; the airway should be checked if the infant suddenly stops crying. [37] Incorrect positioning usually results in multiple punctures and a bloody tap. If CSF fails to flow, gentle suction with a 1.0-mL syringe may be used to exclude a low-pressure syndrome. Pressure readings are inaccurate in the struggling child and hence not commonly measured in infants or young children. Positioning is very important in the infant and is best accomplished by an assistant, who maintains the spine maximally flexed by partially overlying the child and holding him or her behind the shoulders and the knees. The infant has poor neck control; hence, the assistant must also ensure that the child maintains an open airway.

Newborn and preterm infants may experience significant hypoxia during lumbar puncture with clinical deterioration; a sitting position appears preferable. [38] Lumbar puncture in infants with respiratory distress syndrome may cause greater risk than benefit. [39] This is a problem primarily in neonates but may occur in younger infants with sepsis. Gleason and coworkers [40] demonstrated that performing a lumbar puncture in the ill preterm infant using the traditional lateral recumbent position with full neck flexion resulted in significant respiratory abnormalities, presumably due to a ventilation-perfusion mismatch. All infants with serious cardiopulmonary disease should be closely monitored during the procedure. Preoxygenation with or without oxygen saturation monitoring may be used as a precaution. Particular attention should be given to avoiding marked neck and trunk flexion.

Although local anesthesia or sedation has not been a routine practice during lumbar puncture in the infant or child, it is being reconsidered. Pain is perceived in the neonate, and local anesthesia neither produces physiologic instability nor makes the procedure more difficult.  Sedation in the anxious child may be considered as an adjunct as for the

adult, although these agents are relatively contraindicated in the obtunded infant or child without a protected airway or with hemodynamic instability.

## COMPLICATIONS

### Implantation of Epidermoid Tumors

An epidermoid tumor or cyst is a mass of desquamated cells containing keratin within a capsule of well-differentiated stratified squamous epithelium. Congenital lesions are rare and arise from epithelial tissue that becomes sequestered at the time of closure of the neural groove between the third and fifth weeks of embryonic life. Acquired intraspinal epidermoid tumors result from implantation of epidermoid tissue into the spinal canal at the time of lumbar puncture performed with needles without stylets or with ill-fitting needles. [36] The clinical syndrome consists of pain in the back and the lower extremities developing years after spinal puncture. Failure to use a stylet may also result in aspiration of a nerve root into the epidural space.

### Headache After Lumbar Puncture

A number of complications from lumbar puncture have been reported. By far the most common is the post-lumbar puncture headache. This occurs after 5 to 30% of spinal taps. [5] [43] The syndrome starts up to 48 hours after the procedure and usually lasts for 1 to 2 days and occasionally up to 14 days. Exceptional cases lasting months have been described. The headache usually begins within minutes after the patient arises and characteristically ceases as soon as he or she assumes a recumbent position. The pain is mild to incapacitating and is usually cervical and suboccipital but may involve the shoulders and the entire cranium. Exceptional cases include nausea, vomiting, vertigo, blurred vision, ear pressure, tinnitus, and stiff neck. The headache may improve and change to a positional backache or neckache. The headache appears in the first 1 to 3 days and may last for weeks or rarely months to years. The syndrome is caused by leakage of fluid through the dural puncture site. This results in an absolute reduction of CSF volume below the cisterna magna and a downward movement of brain tissue with displacement and stretching of pain-sensitive structures, such as meninges and vessels, which, in turn, causes a traction headache. In the recumbent position there is relief, because the weight of the brain is shifted cephalad. Dural leakage has been confirmed at surgery for disk disease and by at least one isotope myelographic study; the size of the dural rent seems to correlate with the frequency of post-lumbar puncture headaches.

The incidence of headache in normal volunteers has been found to be 1 case per 9 subjects with the use of a 26-ga needle and 1 case per 3 subjects with a 22-ga needle. [45] The headache was reported to be milder when the smaller needle was used. Technically, a 26-ga needle is difficult to place and to manipulate into a position in which it does not become intermittently obstructed by nerve roots. In addition, a syringe is needed to withdraw fluid, and pressure cannot be easily recorded. Theoretically, the incidence of headache is greater with an 18-ga needle.

Other factors claimed to influence the incidence of postspinal headache have been reviewed by Fishman. [5] The incidence is higher in young patients than in older patients and is also increased in females and individuals with a prior headache history. Psychologic factors, quantity and rapidity of CSF removal, forced bedrest, post-lumbar puncture hydration, and position during lumbar puncture have not been found to be relevant in the incidence of headache. [46] Some reports suggest a lower incidence with the lateral approach resulting from the production of holes in the dura and the arachnoid that do not overlap.

The influence of activity on postspinal puncture headache has been studied with contradicting results, including worsening of, improvement in, and no effect on the incidence of headaches when patients were mobilized. [5] [47] Brocker studied 1094 patients and reported a reduction of headache from 36.5 to 0.5% by having the patients lie prone instead of supine for 3 hours after puncture with an 18-ga needle. [47] He concluded that the prone position caused hyperextension of the spine and disrupted alignment of the holes in the dura and the arachnoid, making a leak less likely. Others have failed to show a decrease in duration, severity, or incidence of spinal headache with 24 hours of bedrest. [48] Interestingly, Vilming and colleagues noted a slight reduction in post-lumbar puncture headache if immediate mobilization was undertaken. [49]

Many medications have been advocated for treatment of post-lumbar puncture headache: barbiturates, codeine, neostigmine, ergots, diphenhydramine (Benadryl), dimenhydrinate (Dramamine), amphetamine sulfate (Benzedrine), ephedrine, intravenous fluids, magnesium sulfate, and vitamins. [5] Sechzer and Abel, [50] in a double-blind demand method, found caffeine sodium benzoate (500 mg in 2 mL normal saline given by IV push) to be effective in 75% of patients. Additional patients responded to a second injection 1 to 2 hours later. Other clinicians prefer to put 500 mg of caffeine sodium benzoate in 1 L of saline and infuse the fluid over 1 hour. [51] This approach is contraindicated in the patient at risk for xanthine toxicity (e.g., supratherapeutic theophylline level, history of cardiac dysrhythmias). Aminophylline, 5 to 6 mg/kg by IV infusion over 20 minutes, is also reported to be useful.

Most clinicians generally follow the practice of using a styleted needle that is as small as possible. A 20- to 22-ga needle is often used for adults because of its stiffness and ease of fluid flow. Multiple punctures should be avoided. There is no certainty about activity and position immediately after the procedure. Most post-spinal puncture headaches can be managed with bedrest with the head in the horizontal position. Dehydration should be avoided, because it lowers CSF pressure and might aggravate the headache. Although simple analgesics are commonly prescribed, they have no apparent advantage over bedrest and fluid intake. In the absence of a postural headache it is not necessary to stress immobilization or bedrest after lumbar puncture. [52] A patient with a prolonged headache after spinal puncture should be reassessed to rule out other structural causes of headaches. If the headache is not postural, other causes should be sought.

In cases in which a prolonged low-pressure headache exists, the placing of an epidural blood patch by experienced anesthesiologists is highly successful. An epidural tap is

performed at the level of the prior lumbar puncture. Ten to 20 mL of autologous blood is drawn aseptically into a syringe and slowly injected (1 to 2 mL every 10 seconds) into the epidural space at the site of the dural puncture. The injection is slowed or discontinued if back pain or paresthesias develop. The patient is kept supine for 1 hour while receiving intravenous hydration. Relief usually occurs within 20 to 30 minutes of the procedure. However, epidural patches are less likely to be effective if symptoms have been present for more that 2 weeks. Pain relief is due to the blood patch forming a gelatinous tamponade, hence stopping the CSF leak and providing an immediate elevation of CSF pressure. Patch failures (15 to 20%) are believed to be due to improper needle placement, injection of an inadequate quantity of blood, or an incorrect diagnosis. A second patch is often successful.

Complications reported after an epidural patch include back stiffness (15%), paresthesias, radicular pain, subdural hematoma, adhesive arachnoiditis, and bacterial meningitis. The procedure should be used in patients with refractory headaches who fail to respond to conservative therapy and should be performed by physicians trained in the procedure.

**Infection**

Spinal puncture is absolutely contraindicated in the presence of local infection at the puncture site (cellulitis, epidural abscess, or furunculosis) because of the danger of inducing meningitis.

The postulation that an association exists between performance of a lumbar puncture during bacteremia and later development of meningitis has been examined by several laboratory and clinical investigators. [55] [56] A large concentration of bacteria in the bloodstream at the time of CSF examination is associated with meningitis. The meningitis could be coincidental ("spontaneous meningitis") or could result from leakage of blood containing bacteria into the subarachnoid space after lumbar puncture ("lumbar puncture-induced meningitis"). Eng and Seligman reported that 14% of 165 cases of bacteremia caused by *Streptococcus pneumoniae, Haemophilus influenzae,* and *Neisseria meningitidis* had evidence of meningitis at the time of initial lumbar puncture. [57] They argue that one cannot easily differentiate spontaneous meningitis from lumbar puncture-induced meningitis with these organisms because of their ability to invade the meninges spontaneously. It is likely that many of these rare cases of "lumbar puncture-induced meningitis" represent cases in which the cautious physician has performed a lumbar puncture early in the course of meningitis, that is, before the infection has had time to be reflected in the CSF.

Teele and coworkers, however, reported that 7 of 46 children developed meningitis after an initial normal lumbar puncture in the presence of bacteremia. [56] *S. pneumoniae, H. influenzae,* and *N. meningitidis* were recovered in all cases. All cases of "lumbar puncture-induced meningitis" occurred in children younger than 1 year of age who received no antimicrobial therapy at the time of initial cultures. Empirical treatment for bacteremia despite a clear CSF is recommended by Teele and associates in the child younger than 1 year if a high fever and leukocytosis are present. [56]

Other studies suggest that the development of meningitis in children with occult bacteremia is more strongly associated with *S. pneumoniae, H. influenzae* type B, or *N. meningitidis* species of bacteria and *not* with the performance of lumbar puncture. [58] Suspected bacteremia is not a contraindication for performing a lumbar puncture; delay in diagnosis because of concern regarding the risks of a lumbar puncture is more serious than the risk of causing meningitis with the procedure. [59] [60]

**Herniation Syndromes After Lumbar Puncture**

Lumbar puncture is of value in confirming a diagnosis of meningitis and subarachnoid hemorrhage. Generally, when the patient has symptoms consistent with bacterial meningitis, lumbar puncture is performed before a head CT scan. However, in patients with a suspected intracranial mass lesion, a CT scan is generally performed before the lumbar puncture and antibiotics administered after blood cultures and before the CT scan when meningitis remains on the differential diagnosis list.

Particularly with supratentorial mass lesions, there may be large pressure gradients between the cranial and lumbar compartments. When brain volume is increased because of a mass lesion or edema, rostrocaudal displacement may occur after lumbar puncture if the skull is intact. Techniques for management of increased ICP are discussed elsewhere (see Chapter 64) .

Lowering the lumbar spinal canal pressure by removing CSF may increase the gradient between cranial and lumbar compartments, promoting both transtentorial and foramen magnum herniation. The frequency with which a lumbar puncture causes or accelerates transtentorial herniation is unknown, because a patient might have developed herniation spontaneously without the procedure. Duffy reported experience with 30 patients referred to a neurosurgical service with post-lumbar puncture herniation syndromes; in this selected population of adverse outcomes, he noted that complications occurred in the first 12 hours after the procedure. [22] Half of these referred patients lost consciousness immediately after the puncture. In half of these patients, the lumbar pressure was normal. Only one third had papilledema.

With the availability of CT scanning, patients at risk of herniation should be less likely to undergo lumbar puncture. However, the literature from both the pre- and post-CT eras suggests a risk of herniation after lumbar puncture to be in the range of 2 to 3%. [61] Baker and colleagues noted that CT scans at their institution were poorly correlated with CSF pressures. [62] Among 13 patients with a CSF pressure 210 mm $H_2O$, 6 had normal CT scans and 3 had generalized atrophy. Only 4 had evidence of an intracranial mass, midline shift, or hydrocephalus. Of note, no patient demonstrated herniation over the subsequent 48 hours. Similarly, Zisfein and Tuchman observed 38 patients with CT scan-documented intracranial mass lesions who underwent lumbar puncture. [63] They note that only 1 patient who had fixed, dilated pupils and absent caloric reflexes before the lumbar puncture died. All other patients were neurologically stable or had improved within 48 hours. Hence, with the current use of small-caliber spinal needles and aggressive concurrent use of ICP-lowering agents, herniation appears extremely rare

and may not be fully predictable by CT scan or opening CSF pressure readings.

Horowitz and coworkers reported herniation in 18 of 302 infants with bacterial meningitis, all of whom experienced deterioration within 8 hours of lumbar puncture. [64] Patients with meningitis who have localizing neurologic signs may have a coexisting brain abscess or infarction from arterial or venous thrombosis. Obstructive hydrocephalus and generalized cerebral edema may complicate meningitis with a secondary increase in ICP.

A careful neurologic examination should precede all spinal punctures. When there is a history of headache with progressive mental changes and the development of *localizing* neurologic signs, then spinal puncture should *not* be performed as the initial diagnostic procedure. When these findings are present and meningitis is still clinically suspected, an initial dose of antibiotic should be administered empirically while awaiting results of CT or MRI to determine the safety of a subsequent lumbar puncture. Appropriate cultures of blood and other body fluid should be obtained before antibiotic administration. Blood cultures are positive in 80% of infants with meningitis. Measures to lower ICP before spinal puncture can also be instituted.

A CT scan should identify hemorrhagic lesions and most neoplasms; it should aid in the decision regarding the need for and the risk involved with spinal puncture. Head CT may help identify patients with unequal pressures between intracranial compartments who are at greater risk for cerebral herniation. CT findings that suggest unequal pressure between intracranial compartments include lateral shift of midline structures, loss of suprachiasmatic and circum-mesencephalic cisterns, shift or obliteration of the fourth ventricle, and failure to visualize the superior cerebellar and quadrigeminal plate cisterns with sparing of the ambient cisterns. [65] The presence of a posterior fossa mass would be a strong contraindication to a lumbar puncture. Because of bone and motion artifact, the posterior fossa unfortunately may be a difficult area to visualize on CT.

**Recognition of Herniation Syndromes**

Herniation syndromes are the result of downward displacement of the hemispheres and the basal ganglia, which compress and displace the diencephalon and the midbrain rostrocaudally through the tentorial notch. Etiologic features and pathogenesis are detailed in the monograph by Plum and Posner. [66] Herniations have the potential to initiate vascular and obstructive complications that aggravate the original expanding lesion and can create an irreversible pathologic process. The anterior cerebral artery may be compressed against the falx and may increase ischemia and edema of the herniating hemisphere. Midline displacement posteriorly compresses the deep great cerebral vein and raises pressure in its area of drainage. Compression of the posterior cerebral artery at the tentorial notch can produce occipital infarction and swelling. In addition, kinking of the aqueduct may interfere with CSF circulation. This blockage may produce a normal spinal CSF pressure. Transtentorial herniation displaces the brainstem downward, stretching medial perforating branches of the basilar artery, as the artery is tethered to the circle of Willis. This produces brainstem ischemia and hemorrhages.

In general, pathologic changes with supratentorial mass lesions spread through the hemisphere and move rostrad and caudad in a progressive manner, with progressive dysfunction of the hemisphere and, subsequently, succeeding levels of the brainstem. The infrequent exceptions are seen in patients with acute cerebral-intraventricular hemorrhage and in patients with hemispheric mass lesions with incipient herniation who undergo lumbar puncture. Such conditions may rapidly progress from hemispheric dysfunction to sudden medullary failure.

**Central (Transtentorial) Herniation**

This condition occurs in response to lesions of the frontal, parietal, and occipital lobes and the extracerebral lesions lying toward the vertex or the fronto-occipital lobes (Fig. 65-7) (Figure Not Available) . Frequently, patients are subacutely or chronically ill with bilateral disease, and the diagnosis may be uncertain.  Initially, subjects exhibit a change in alertness or behavior. If the supratentorial lesion enlarges, compressing the diencephalon, stupor and then coma develop. At this point, monitoring of respiratory, ocular, and motor signs helps in diagnosing a supratentorial lesion and in determining the rostrocaudal direction of the disease process. Respirations at this time may be interrupted by deep sighs or yawns and periodic breathing of the Cheyne-Stokes type (periods of hyperpnea regularly alternating with apnea). Pupils are small but react briskly. Eye movements may be conjugate or slightly divergent with roving eye movements. Caloric testing with cold water produces a conjugate slow tonic movement to the side of irrigation. Many individuals have a hemiparesis before herniation. As the diencephalic stage of the central syndrome evolves, the contralateral hemiplegia may worsen, with the homolateral limbs developing a paratonic resistance to movement, but the individual continues to respond to noxious stimuli appropriately. At this stage both plantar responses are extensor. Decorticate responses appear and consist of flexor muscle hypertonus in the upper extremity with predominantly extensor hypertonus in the leg. Recognition of a diencephalic stage of herniation is important in that it gives warning that a potentially reversible lesion may become irreversible.

Once midbrain signs develop, they probably reflect infarction rather than reversible ischemia and compression. The chances of successfully removing or alleviating a supratentorial mass are small once the midbrain stage is reached.

Patients who develop midbrain and upper pons failure exhibit a sustained tachypnea; pupils dilate to a fixed midposition (3 to 5 mm), and oculovestibular reflexes become difficult to obtain, requiring side-to-side head movements and cold caloric irrigation. Disconjugate eye movements appear with failure to adduct (internuclear ophthalmoplegia). Motor responses give way to extensor hypertonus in all limbs (decerebrate rigidity). Midbrain damage results from ischemia and infarction, and few patients recover.

As the brainstem becomes more ischemic, the pupils maintain a fixed position, eye movements are lost, and decerebration gives way to flaccidity. The medullary stage consists of irregular respirations with long periods of apnea. The pupils dilate and the

blood pressure falls, with death being inevitable.

**Uncal Herniation**

Uncal herniation occurs when expanding lesions in the temporal fossa shift the medial temporal lobe (uncus) and the hippocampal gyrus medially over the incisural edge of the tentorium. This flattens the midbrain, pushing it against the contralateral incisura. The third nerve and the posterior cerebral artery on the side of the lesion are caught between the swollen uncus and the free edge of the tentorium (Figs. 65-8 (Figure Not Available) and 65-9) (Figure Not Available) . The earliest sign is a unilaterally sluggish or slightly dilated pupil. Because the diencephalon may not be the first structure encroached on, impaired consciousness is not consistently present as an early sign of uncal herniation. Other respiratory, ocular, and motor findings may not be appreciably changed from earlier examinations. Pupillary dilation may persist for several hours, but once the patient progresses beyond this stage there is a tendency for midbrain dysfunction to occur rapidly. Ipsilateral external ophthalmoplegia soon follows pupillary dilation along with stupor and coma. Oculovestibular reflexes disappear as ischemia spreads to the midbrain. As the opposite cerebral peduncle is compressed against the tentorial edge, hemiplegia may appear ipsilaterally to the expanding supratentorial lesion (Kernohan's notch). Decerebrate posturing develops, with the opposite pupil becoming dilated and fixed. Progression then proceeds as described for the central syndrome. As with transtentorial herniation, once midbrain failure occurs, survival is much less likely.

## Backache and Radicular Symptoms

Minor backache occurs with a frequency of 90% from the trauma of the spinal needle. Frank disk herniation has been reported from the passing of the needle beyond the subarachnoid space into the annulus fibrosis. Transient sensory symptoms from irritation of the cauda equina are also common.

Other reported complications include transient unilateral or bilateral sixth nerve palsies caused by stretching or displacement of the abducens nerve as it crosses the petrous ridge of the temporal bone, subarachnoid hemorrhage, subdural and epidural hematoma, anaphylactoid reactions to local anesthetics, settling of cord tumors, and retroperitoneal abscess produced by dural laceration in patients with meningitis. [67] Most of these are rare and seldom encountered.

Most of the complications of lateral cervical and cisternal puncture are similar to those encountered with a lumbar puncture. In addition, perforation of a large vessel with resultant cisterna magna hematoma or obstruction of vertebral artery flow has been described. Puncture of the medulla oblongata may cause vomiting or apnea, and puncture of the cord may be associated with pain. Long-lasting side effects of cord puncture are probably minor. [34] In addition, traumatic tap and post-spinal puncture headache may occur with lateral cervical and cisternal puncture.

# INTERPRETATION

## Pressure

The pressure of the CSF is of great clinical importance. It should be accurately measured whenever possible. Accurate measurement is dependent on patient cooperation. Elevated pressures are abnormal. Opening pressure is taken promptly, avoiding falsely low values due to leakage through and around the needle. Normal pressure is between 70 and 180 mm $H_2O$. Herniating cerebellar tonsils may occlude the foramen magnum and prevent increased ICP from being reflected in the lumbar pressure reading. Increased ICP can result from expansion of the brain (edema, hemorrhage, or neoplasm), overproduction of CSF (choroid plexus papilloma), a defect in absorption, or obstruction of flow of CSF through the ventricles. Cerebral edema may be associated with meningitis, $CO_2$ retention, subarachnoid hemorrhage, anoxia, congestive heart failure, or superior vena cava obstruction. Pressure may be falsely elevated in a tense patient, when the head is elevated above the plane of the needle, and, possibly, with marked obesity or muscle contraction. [5] Pressure is not usually measured in the neonate, because a struggling or crying child will have a falsely elevated pressure. The upper limits of normal are 50 mm $H_2O$ in neonates and 85 mm $H_2O$ in infants and young children. [68] Although the manometer reading is falsely elevated in the sitting position, the level should not rise above the foramen magnum.

Low pressure should suggest obstruction of the needle by meninges. Low pressure can also be seen with spinal block. Rarely, a primary low-pressure syndrome occurs in a setting of trauma, following neurosurgical procedures, secondary to subdural hematomas in elderly patients, with barbiturate intoxication, and in cases of CSF leakage through holes in the arachnoid.

The Queckenstedt test is useful for demonstrating the presence of obstruction in the spinal subarachnoid space. [4] [5] The test is seldom performed today, because myelographic techniques have been refined and the availability of MRI has reduced the number of myelograms and associated lumbar puncture. With the patient in the lateral recumbent position, jugular vein compression causes decreased venous return to the heart. This distends cerebral veins and causes a rise of ICP, which is transmitted throughout the system and is measured in the manometer. After 10 seconds of bilateral compression, CSF pressure usually rises to 150 mm $H_2O$ over the initial reading and returns to the baseline in 10 to 20 seconds after release. If there is no change in the lumbar pressure or if the rise and fall are delayed, it should be concluded that the spinal subarachnoid space does not communicate with the cranial subarachnoid space. In this situation, Pantopaque should be injected before removal to facilitate subsequent performance of a myelogram. This is necessary because the lumbar dural sac may collapse, making it impossible to reenter the canal. If cervical cord disease is suspected, the test should be repeated with the neck in the neutral position, hyperextended, and flexed. When lateral sinus obstruction is suspected, unilateral jugular venous compression may be used (Tobey-Ayer test).

**Appearance**

If the CSF is not crystal clear, a pathologic condition of the CNS should be suspected. The examiner should compare the fluid with water, viewing down the long axis of the tube or holding both tubes against a white background. A glass tube is preferred, because plastic tubes are frequently not clear. The fluid may be clear with as many as 400 RBCs/mm3 and 200 WBCs/mm3 . [5]

Xanthochromia is a yellow-orange discoloration of the supernate of centrifuged CSF. Xanthochromia is produced by red cell lysis and is caused by one or more of the following pigments: oxyhemoglobin, bilirubin, and methemoglobin. Oxyhemoglobin causes red coloration; bilirubin, yellow; and methemoglobin, brown. Oxyhemoglobin is seen within 2 hours of subarachnoid bleeding and red cell lysis. Formation reaches a maximum in 24 to 48 hours after hemorrhage and disappears in 3 to 30 days. [4] The appearance of bilirubin in the CSF involves the conversion of oxyhemoglobin by the enzyme heme oxygenase. The enzyme is found in the choroid plexus, the arachnoid, and the meninges. Enzyme activity appears approximately 12 hours after the hemorrhage. [4] Bilirubin may persist for 2 to 4 weeks. Bilirubin in CSF caused by hepatic or hemolytic disease does not appear until a serum level of 10 to 15 mg total bilirubin per 100 mL is reached, unless underlying disease associated with a high CSF protein is present. Xanthochromia may be seen with CSF protein values above 150 mg/dL. CSF may clot with complete spinal block and very high CSF protein.

Oxyhemoglobin and bilirubin may be measured chemically or by spectrophotometric analysis. Demonstration of these compounds in CSF may help in the distinction between recent intracranial hemorrhage and a traumatic tap. Oxyhemoglobin may form as a result of red cell lysis if the tube has been allowed to stand for more than 1 hour before testing, however.

Methemoglobin is a reduction product of oxyhemoglobin characteristically found in encapsulated subdural hematomas or in old intracerebral hematomas.

**Cells**

The technique involved in cell counts is reviewed in Fishman's text. [5] WBC counts over 5 cells/mm3 should be taken to indicate the presence of a pathologic condition. In a study of 135 normal university students, Tourtellotte and Shorr reported almost exclusively lymphocytes and monocytes, and the presence of 1 to 5 lymphocytes in the CSF may be normal. [4] Neonatal CSF may show up to 32 WBCs/mm3 with prominent neutrophils; infants 4 to 8 weeks old may have 22 WBCs/mm3 . [71] It has been stated that polymorphonuclear leukocytes are never seen in normal adults. However, with the use of the cytocentrifuge, an occasional specimen may show a neutrophil in an otherwise normal individual. [72] Such a finding should routinely prompt culture of the CSF, because the presence of a neutrophilic pleocytosis is commonly associated with bacterial infections or the early stages of viral infections, tuberculosis, meningitis,

hematogenous meningitis, and chemical meningitis due to foreign bodies.

As many as 30% of patients may exhibit CSF pleocytosis after a generalized or focal seizure. In a review of 102 patients without CNS infection who had lumbar puncture within 48 hours of a seizure, Prokesch reported an average of 72 cells/mm3 , noting the presence of both polymorphonuclear and mononuclear cells. [73] Although these patients did not have CNS infection, many did have serious intracranial pathologic processes (subdural hematoma, subarachnoid hemorrhage, or stroke). This finding should prompt a search for infection and assessment of the seizure as being possibly secondary to an underlying neurologic disorder.

Small lymphocytes may be seen in normal individuals. Small and large immunocompetent cells are found with a variety of bacterial, fungal, viral, granulomatous, and spirochetal diseases as well as the presence of foreign substances.

Eosinophils are always abnormal and most commonly represent a parasitic infestation of the CNS. They may also be seen after myelography and pneumoencephalography and, to a minor degree, in other inflammatory diseases, including tuberculous meningitis and neurosyphilis. CSF eosinophilia has also been reported in cases of subarachnoid hemorrhage, lymphoma, Hodgkin's disease, brucellosis, fungal meningitis, *Mycoplasma pneumoniae* infection, measles, lymphocytic choriomeningitis, rickettsial infections, leukemia, demyelinating diseases, sarcoidosis, acute inflammatory demyelinating polyneuropathy, allergic reactions, and idiopathic eosinophilic meningitis. [74]

Normal CSF RBCs are less than 10/mm3 . Counts up to 100/mm3 that are otherwise unexplained may be due to a traumatic tap. Herpes simplex virus encephalitis may elevate the CSF RBC count in many patients. Myeloid and RBC precursors may contaminate CSF with bone marrow cells from an adjacent vertebral body. [75]

**Glucose**

Glucose enters the CSF by way of the choroid plexus as well as by transcapillary movement into the extracellular space of the brain and the cord by carrier-mediated transport. It then equilibrates freely with the CSF subarachnoid space. Fishman concludes that a low CSF glucose concentration indicates increased glucose use in the brain and the spinal cord and, to a lesser degree, by polymorphonuclear leukocytes, bacteria, and inhibition of membrane carrier systems. [5] Once in the CSF, glucose undergoes glycolysis and there is an invariable rise in CSF lactate levels. Glucose levels remain subnormal for 1 to 2 weeks after the effective treatment of meningitis.

The normal range of CSF glucose is between 50 and 80 mg/dL, which is 60 to 70% of the glucose concentration in the blood. Ventricular fluid glucose levels are 6 to 8 mg/dL higher than in lumbar fluid. A ratio of CSF:blood of <0.5 or CSF levels <40 mg/dL are invariably abnormal. The ratio is higher in infants for whom a ratio of <0.6 is considered abnormal. Hyperglycemia may mask a depressed CSF glucose level, and the CSF to blood glucose ratio should be measured routinely. With extreme hyperglycemia a ratio of 0.3 has been suggested as being abnormal. [76] Between 90 and 120 minutes is required before the CSF glucose reaches a steady state with blood glucose changes

(e.g., after the intravenous injection of glucose). When CSF glucose is of diagnostic importance, CSF and blood samples ideally should be obtained after a 4-hour fast.

Low CSF glucose levels may be found in several diseases of the nervous system (see Table 65-1) . Only low concentrations

| TABLE 65-1 -- Low CSF Glucose Syndromes | |
|---|---|
| Bacterial meningitis | Syphilis |
| Tuberculous meningitis | Chemical meningitis |
| Fungal meningitis | Subarachnoid hemorrhage |
| Sarcoidosis | Mumps meningitis |
| Meningeal carcinomatosis | Herpes simplex encephalitis |
| Amebic meningitis | Hypoglycemia |
| Cysticercosis | Trichinosis |

of glucose are of diagnostic value, and elevated CSF glucose levels generally have no significance; elevation usually reflects hyperglycemia. A rapid estimate of the CSF glucose level can be obtained by using bedside reagent strip testing with a commercial autoanalyzer. However, formal laboratory testing is recommended for confirmation of bedside levels.

**Protein**

The normal range of the lumbar CSF protein level is 15 to 45 mg/dL. Infants normally have a lower level than adults, and protein levels may drop after a lumbar puncture. The

concentration is lower in the ventricles (5 to 15 mg/dL) and the basilar cisterns (10 to 25 mg/dL), reflecting a gradient in the permeability of capillary endothelial cells to proteins in the blood. Levels of CSF protein in premature infants and full-term neonates is higher than in adults with a mean of 90 mg/dL; protein levels decline by 8 weeks of age, reflecting maturation of the blood-brain barrier. Most of the proteins in CSF normally come from the blood, which normally has a protein concentration of up to 8000 mg/dL. Protein entry is determined by molecular size and relative impermeability of the blood-CSF barrier. A full range of serum proteins are found in CSF at several hundredfold dilution. Faulty reabsorption of protein by arachnoid villi may also elevate protein levels. Increases in CSF total protein levels are a nonspecific abnormality associated with many disease states. Levels >500 mg/dL are uncommon and are seen mainly in meningitis, in subarachnoid bleeding, and with spinal tumors. The high levels seen with cord tumors result from an increase in local capillary permeability. With high levels (generally 1000 mg/dL), CSF may clot (Froin's syndrome).

Hemorrhage into the CSF or the introduction of blood by a traumatic tap increases CSF protein levels. If the serum protein is normal, the CSF protein should theoretically rise by 1 mg for every 1000 red cells, but this is variable. The inflammatory effect of hemolyzed RBCs may also significantly increase CSF protein.

Selective measurement of immunoglobulin fractions in CSF has proved to be of diagnostic value in suspected cases of multiple sclerosis. Elevated CSF immunoglobulin levels may reflect blood-brain barrier disruption or local antibody response to a CNS immune response. [77] Stimuli may be infectious or antigenic, producing an inflammatory response. Immunoglobulin elevation has been found in many chronic inflammatory conditions, including syphilis, viral encephalitis including HIV infection, subacute sclerosing panencephalitis, progressive rubella encephalitis, tuberculous meningitis, sarcoidosis, cysticercosis, and acute inflammatory demyelinating polyneuropathy (Guillain-Barre syndrome).

**The Traumatic Tap**

It should not be difficult to distinguish between subarachnoid bleeding and bloodshed by the spinal needle if certain steps are taken *at the time of the initial puncture.* In traumatic punctures the fluid generally clears between the first and third tubes as the needle is washed by CSF. Decreasing cell counts on the first and third tubes help confirm this. In a recent hemorrhage, however, a declining cell count may represent layering of cells in a recumbent patient. The fluid should then be centrifuged. With moderately blood-stained fluid, the supernatant should be clear if the RBCs have been present for less than 2 hours (traumatic tap). Red cells undergo hemolysis in the CSF after a few hours to produce xanthochromia. Xanthochromia persists for up to 4 weeks, depending on the number of RBCs that were originally present. An early CSF examination may show clear fluid before the development of hemolysis, even after spontaneous subarachnoid bleeding. However, xanthochromia may be seen after a traumatic tap if the RBC count exceeds 150,000 to 200,000/mm3 . The yellow color may appear if sufficient serum is present. [78] The presence of a clot in one of the tubes strongly favors a traumatic tap.

In subarachnoid hemorrhage, clotting does not occur because blood is defibrinated at the site of the hemorrhage. Any lumbar puncture performed several days after a traumatic tap may yield stained fluid. An immediate repeat puncture at a higher interspace yielding clear CSF may also help distinguish a traumatic tap. The fluid from a traumatic tap should contain about 1 WBC per 700 RBCs if the complete blood cell count is normal, but this ratio is highly variable. Formulas to assess the contribution of total CSF cell count and protein values from traumatic taps have been proposed but do not have uniform support. [79] [80] Indeed, Bonadio and colleagues suggest that children with meningitis and traumatic taps (i.e., RBC count 1000/mm3 ) generally have other features of meningitis besides an observed-to-predicted WBC ratio 10 (e.g., predominance of neutrophils, low CSF-to-serum-glucose ratio, and positive Gram stain). [81] All blood-contaminated CSF should be cultured, especially from uncooperative infants and children being evaluated for sepsis.

Lang and colleagues suggest the use of a D-dimer test on CSF to identify subarachnoid hemorrhage. [82] Although identifying local fibrinolysis, other conditions such as disseminated intravascular coagulation, a previous traumatic tap, or prior thrombolytic therapy may produce false-positive test results.

## CSF Analysis with Infections

### Bacterial Infections

The CSF findings are essential in establishing a provisional diagnosis of acute bacterial meningitis. CSF analysis establishes the diagnosis, the causative organism, and the choice of antibiotics. CSF must be transported to the laboratory immediately and examined at once. CSF cells begin to lyse within 1 hour of collection, but this may be slowed by refrigeration. In cases of meningococcal infection, a delay in processing may cause the diagnosis to be missed, because the organism tends to autolyze rapidly. Speed is only slightly less important with other organisms, because early initiation of antibiotic therapy is crucial.

The Gram stain is of great importance, because this often dictates the initial choice of antibiotic therapy. The physician should become expert at examining Gram stains of CSF, and it is suggested that one should spend 10 minutes personally examining each CSF Gram stain specimen when the CSF WBC count is elevated. Gram-negative intracellular or extracellular diplococci are indicative of *Neisseria meningitidis*. Small gram-negative bacilli may indicate *Haemophilus influenzae*, especially in children. The presence of gram-positive cocci indicates *Streptococcus pneumoniae,* other *Streptococcus*

species, or *Staphylococcus.* Twenty percent of Gram stains may be falsely negative

because too few organisms are present. The Gram stain smear is more likely to be positive in patients who have not received prior antibiotic therapy. Acridine orange stain may improve the yield in bacterial infections with negative Gram stains.

For culture, blood and chocolate agar are required. *N. meningitidis* and *H. influenzae* grow best on chocolate agar. The plates are incubated under 10% $CO_2$ . Thioglycolate medium is used for possible anaerobic organisms. Cultures are examined at 24 and 48 hours, but plates should be kept for at least 7 days. Large volumes of CSF may improve yields. [84]

While the culture is pending, one may suspect a bacterial infection in the presence of an elevated opening pressure and a marked pleocytosis ranging between 500 and 20,000 WBCs/mm3 . The differential count is usually chiefly neutrophils. A count above 1000 cells/mm3 seldom occurs in viral infections. Occasionally, acellular fluid may be found in the severely immunosuppressed patient or others with appropriate presentations. Repeat lumbar puncture may be required in febrile patients in whom the clinical features remain compatible with meningitis.

CSF glucose levels less than 40 mg/dL or less than 50% of a simultaneous blood glucose level should raise the question of bacterial meningitis, even in the presence of a negative Gram stain and a low cell count. Glucose levels with bacterial meningitis are occasionally below 10 mg/dL; levels are normal in 9% of bacterial meningitis. [76] The CSF protein content in bacterial meningitis ranges from 500 to 1500 mg/dL and usually returns to normal by the end of therapy. CSF protein values above 220 mg/dL virtually ensure an acute meningitis of being bacterial. Of note, prior antibiotic therapy may adversely affect the sensitivity of cultures and Gram stains for bacterial meningitis but does not significantly affect WBC counts, CSF to blood glucose ratios, or CSF protein values. [87] Spanos and colleagues [88] have developed a useful nomogram to help distinguish bacterial from viral infections (Fig. 65-10) (Figure Not Available) .

A useful test that can be performed in nearly all laboratories is the measurement of CSF lactate dehydrogenase (LDH) levels. In bacterial and fungal meningitis, LDH levels are increased. [89] The LDH level may be elevated even in patients who have received antibiotics for 1 or 2 days. The LDH levels in viral infections tend to be normal, whereas values in bacterial meningitis are usually two to four times greater than the normal concentration of approximately 1.6 mEq/L.

A study by Durack and Spanos questions the use of repeat spinal taps as a test of meningitis cure. [90] Their review of 165 meningitis cases revealed 13 instances in which the repeat tap led to unnecessary intervention and 2 others in which treatment failure was *not* detected by the repeat spinal tap.

**Microbial Antigens and Polymerase Chain Reaction**

Several tests other than CSF culture and Gram stain are available to establish a bacterial etiology of meningitis. These include blood cultures, CSF counterimmunoelectrophoresis (CIE), CSF latex agglutination (LA), and coagglutination counterimmunoelectrophoresis. [7] All of the ancillary tests have a low sensitivity for

bacterial meningitis, thus limiting their use. In 50 to 80% of bacterial meningitis cases, blood cultures are positive for the etiologic agent. [91]

CIE uses wells in two rows of agarose gel. A different antiserum is placed in each well. A current is passed through the gel with the reactants then moving toward each other by electrophoretic mobilization of the antigen. A line of precipitation visualized in 1 to 4 hours represents positive reaction between antiserum and antigen. [92] Commercial kits are available to detect *S. pneumoniae*; *Listeria monocytogenes*; *H. influenzae*; *N. meningitidis* A, B, and C and W135; group B *Streptococcus*; K1 strains of *Escherichia coli*; *Klebsiella*; and *Pseudomonas* species. [93]

Particle agglutination involves staphylococcal coagglutination and latex agglutination. Antibody on the surface of a colloid combines with antigen binding sites to cross-link the colloid-forming antigen bridges. A matrix forms and appears as a macroscopic agglutination. Agglutination tests can detect approximately 10 times less antigen than CIE. False-positive tests can occur in the presence of rheumatoid factor, serum complement components, and possibly other serum proteins. The technique may be used in infections due to *H. influenzae*, *S. pneumoniae*, *N. meningitidis*, and group B *Streptococcus.*

Another technique having some potential use is the enzyme-linked immunosorbent assay. This technique may detect 100 to 1000 times less antigen than agglutination tests but is technically more difficult and requires 4 hours to perform.

A positive CSF antigen test may be expected in 70 to 90% of patients with *Neisseria* meningitis. This compares with a positive Gram stain in approximately 70% of patients. Positive latex antigen tests have been reported in approximately 60% of *S. pneumoniae* meningitis cases, with a positive Gram stain in 80%. A positive latex test and Gram stain are reported in approximately 85% of *H. influenzae* meningitis cases.Group B *Streptococcus* can be detected with a 60 to 90% sensitivity. The Gram stain may be difficult to assess after antibiotic therapy has been initiated. Bacterial antigens may persist in the CSF for several days after antibiotic therapy. Twenty-five to 33% of positive tests are lost per day in the setting of appropriate antimicrobial therapy. A negative test, however, does not rule out bacterial meningitis. [93] In addition, blood and urine should be examined for antigen. Often antigen may be found only in the urine. Urine needs to be concentrated and may have the disadvantage of reflecting urinary tract infections. The particle agglutination test for *H. influenzae* type B may be positive for up to 10 days after children have received *H. influenzae* polysaccharide vaccine. Antigen tests are not useful in diagnosing gram-negative bacillary, staphylococcal, and *Listeria* meningitis. In addition, although antigen tests may identify the bacterial pathogen, they do not provide information about the antibiotic susceptibility of the organism.

Future use of polymerase chain reaction (PCR) will aid the rapid diagnosis of CNS infection when results of current techniques are suboptimal. PCR is a method for amplifying target nucleic acid in CSF by use of repeated cycles of DNA synthesis. PCR requires use of flanking DNA sequences at the opposite ends of the target DNA. Synthetic primers anneal to their respective recognition sequences at the opposite end

of the target sequence; they serve as primers for new DNA synthesis. PCR allows detection of and quantification of organisms whose genetic material is DNA or messenger RNA. PCR permits the diagnosis of infectious disease with a high degree of sensitivity and specificity and allows rapid reliable detection of microbes present in small numbers. Laboratory false-negative and false-positive results may occur. PCR is only available at limited centers, although this seems likely to change in the near future. [94]

**Empirical Antibiotic Use Before**

Lumbar Puncture

Many patients are transported to a referral center or within a facility for a CT scan to rule out an intracranial mass after a clinical concern for meningitis is raised. In such instances, CSF examination may not be performed before transport. This may be due to technical problems (uncooperative or large patient) or concerns regarding safety of lumbar puncture in an obtunded patient with possible increased ICP.  The initial physician may have to decide whether to initiate empirical antibiotic therapy. Prior antibiotic administration could obscure the bacterial etiology, whereas a delay in initiating therapy may increase the morbidity and mortality of the illness. It may be difficult to identify individuals at risk for a fulminant course, and the diagnosis of bacterial meningitis cannot always be made with confidence; some cases may be misdiagnosed as subarachnoid hemorrhage or metabolic encephalopathy.

Talan and associates note that parenteral antibiotic treatment decreases the percentage of positive CSF cultures by 4 to 33% and of positive CSF Gram stains by 7 to 41%. [91] A minimum window of 2 to 3 hours exists after giving parenteral antibiotics when CSF cultures are not adversely affected. After 24 hours post treatment, as many as 38% of meningitis patients may still have positive CSF cultures. When CSF is cultured more than several hours after parenteral antibiotics are administered, antigen tests may be of help. Of course, blood cultures should be obtained immediately before antibiotic administrations whenever possible. Occasional patients may develop a lymphocytic pleocytosis with antibiotic therapy; but in most cases, cell count, differential, glucose and protein concentrations are not changed in the first 2 to 3 days of antibiotic therapy. [76] Although there are no data to confidently address the potential advantages or disadvantages of antibiotic therapy before lumbar puncture, it is reasonable to initiate therapy on the premise that a delay may be deleterious. If a lumbar puncture cannot be done, consultation with physicians at the referral center would seem appropriate. If a lumbar puncture is performed before a transfer, a portion of the CSF (chilled on ice) should be sent with the patient.

Bacterial meningitis occurring in children younger than 10 years of age historically has been often due to *H. influenzae*. *H. influenzae* appears to be easier to grow from early postantibiotic cultures and is more likely to be associated with positive blood cultures and antigen tests. In the pediatric population a single dose of antibiotic before transport is unlikely to prevent bacterial identification. In neonates, adults, and immunosuppressed patients, the sensitivity of blood cultures and immunologic tests is less reliable. CSF examination before antibiotic administration or *early* in the course of

treatment is preferred.

For suspected or confirmed cases of acute bacterial meningitis, antimicrobial therapy can be started based on the most likely causative organism based on age of the subject, associated diseases, and renal function. For immunocompromised and postneurosurgical patients, a third-generation cephalosporin (cefotaxime, ceftizoxime, ceftazidime, or ceftriaxone) and vancomycin should be used for coverage against staphylococci, *L. monocytogenes,* and gram-negative organisms. [7] Table 65-2 (Table Not Available) offers guidelines for emergency antibiotic therapy. The third-generation cephalosporins are efficacious in many empirical regimens or situations where the organism is known. To rely on third-generation cephalosporins for all cases of bacterial meningitis would result in treatment failures of all *Listeria* species and increasing numbers of *Enterobacter, Serratia,* and *Pseudomonas* groups. Treatment with older agents may be as or more effective in bacteria demonstrating appropriate sensitivity.

**Dexamethasone Therapy in Bacterial Meningitis**

Studies have suggested benefit from adjunctive dexamethasone therapy in reducing neurologic sequelae, especially hearing loss, in children with *H. influenzae* meningitis. The adjunctive benefit of corticosteroids during treatment of meningitis caused by other organisms is unknown.

In acute bacterial meningitis, bacterial cell wall components including lipopolysaccharides and teichoic acid initiate and exacerbate the host response. These stimulate the production of cytokines, including interleukin-1 and tumor necrosis factor, from macrophages and monocytes. Cytokines may injure vessels, diminish cerebral perfusion, and stimulate cerebral swelling. [68] The dosage of dexamethasone is 0.15 mg/kg every 6 hours for 4 days. When used, it is recommended that the corticosteroid be given before antibiotic use. Benefit in adults is less certain but experimental data in children would seem to support corticosteroid use, with caution being exercised in immunocompromised or leukopenic patients. A moderate inflammatory response in the meninges is required for penetration of the CNS by many antibiotics. Reducing meningeal inflammation will reduce the concentration of antibiotics in the CSF. Corticosteroids should be discontinued after approximately 4 days of treatment, at which time meningeal inflammation has been reduced by antibiotics. [95] [96]

Although an intriguing concept, the actual benefit from adjunctive corticosteroids is controversial. Current data do not allow for definitive recommendations regarding the use of corticosteroids in bacterial meningitis. With the increasing use of *H. influenzae* vaccines, the point may become moot.

**Neurosyphilis**

The true incidence of this disease is unknown. Approximately 5000 new cases of neurosyphilis are estimated to occur in the United States each year. [97] The natural history and clinical manifestations have been modified in the antibiotic era. The widespread use of oral antibiotics has changed neurosyphilis into a chronic partially treated meningitis. Partial therapy may clear peripheral infection and attenuate the

immune response. Therapy may be insufficient to eradicate organisms in the CNS and eye, which may then multiply. Seizures were considered in the preantibiotic era to complicate general paresis only late in the course of the illness, and then only in untreated patients. Previously, seizures were reported as an early manifestation in less than 5% of symptomatic neurosyphilis cases. Seizures now occur in 25% of symptomatic cases and may on occasion be the sole manifestation of neurosyphilis. [97] Seizures are usually partial (focal), with one third of patients having no interictal clinical findings. A treponemal serologic test should be obtained in every adult with acquired partial seizures.

Ophthalmologic findings are frequently present in syphilis. These include a slowly progressive optic atrophy, an acute optic neuritis, cranial oculomotor neuropathy, and a chorioretinitis. A more specific sign is a dissociated pupillary response to light and convergence with loss of the light reflex (Argyll Robertson pupil). Meningovascular neurosyphilis involves infection of both the meninges and the cerebral vasculature. The clinical picture is that of a cerebral infarction or an acute meningoencephalitis. Meningovascular syphilis also may present as headache, nausea, vomiting, confusion, stiff neck, and cranial nerve palsies. Arteritis may affect small, medium, or large vessels, producing infarction or ischemia of brain or spinal cord. The peak incidence is 5 to 7 years after initial infection. Tabes dorsalis, general paresis, and gummas occur 10 to 20 years after initial infection.

CSF findings suggestive of neurosyphilis include more than 5 leukocytes/mm3 , elevated protein concentration, elevated gamma-globulin concentration, and a positive serologic test for syphilis. The glucose concentration is usually normal. Higher cell and protein values are seen in early as opposed to late neurosyphilis.

Diagnostic certainty remains difficult. The diagnostic criterion standard is darkfield microscopy to identify morphology and flexing "corkscrew" motility of spirochetes. Serologic tests for syphilis are either treponemal or nontreponemal. Nontreponemal tests detect a nonspecific globulin complex called reagin. Reagin tests, such as the Venereal Disease Research Laboratory (VDRL) flocculation test, lack sensitivity and should not be used to exclude the diagnosis of neurosyphilis. One third to one half of patients with neurosyphilis have a negative VDRL test in the serum, and more than one third have a negative VDRL test in the CSF. [97] CSF VDRL is quite specific with false-positive results seen primarily with traumatic taps.

Treponemal tests provide evidence of a specific immune response to *Treponema pallidum*. These include the serum fluorescent treponemal antibody adsorption (FTA-ABS), microhemagglutination tests for *T. pallidum* (MHA-TP), and the *T. pallidum* hemagglutination assay (TPHA). These assays are believed to remain positive for life. The FTA-ABS and MHA-TP tests may revert to negative in 13 to 24% of patients after therapy for early (primary) syphilis. There is no evidence that these tests revert to negative in untreated disease. [98] The serum FTA-ABS test is the test of choice to confirm the diagnosis of neurosyphilis. The serum FTA-ABS test is reactive in 95 to 100% of cases of neurosyphilis. A negative serum FTA-ABS test makes CSF examination unnecessary. Its false-positive rate is less than 1%, but the incidence of

biologic false-positive tests may increase in the presence of collagen vascular disorders.

A positive serum treponemal test indicates past infection with syphilis and may be reactive indefinitely, even after treatment. Therefore, CSF is used as a guide to the presence and the activity of neurosyphilis. The VDRL test is the test of choice in CSF and, when positive, is strong evidence for neurosyphilis. False-positive CSF VDRL tests are rare. The FTA-ABS test is not used in CSF; the false-positive rate is between 4 and 6% and is believed possibly to represent antibodies that have passively entered from serum. [99] The FTA-ABS test measures IgG antibody and cannot differentiate active from past infection. The CSF VDRL either may be reactive by contamination with seropositive blood (traumatic tap, subarachnoid hemorrhage) or may occur with entry of serum reagin into CSF during meningitis.

There is some concern that today many patients with parenchymal neurosyphilis have normal CSF. This finding leads to the recommendation that a patient with signs of progressive neurosyphilis and a positive treponemal serologic test be treated with antibiotics regardless of the CSF findings. A CSF pleocytosis may be provoked after 1 week of therapy and may supply supportive evidence for a diagnosis of neurosyphilis. PCR may have future applications for diagnosis of neurosyphilis (see the section on neurosyphilis and HIV infection later in this chapter).

**Viral Studies**

The organisms most commonly isolated in viral meningitis are the enteroviruses (coxsackieviruses, echoviruses) and mumps virus. Enteroviruses are most commonly seen in the summer and fall, and mumps appears most frequently in the winter and spring. Viral cultures in most hospitals are not available and play little role in acute decisions regarding diagnosis and treatment. A serial rise in CSF antibody titers may be helpful but may be difficult to obtain in patients who have clinically recovered. Intrathecal production of organ-specific antibodies (IgM, IgG, and IgA isotopes) may be diagnostic of neurologic infection if there is no prior history of infections. This measurement of both serum and CSF antibody titers must be done in a specialized laboratory. Viral meningitis is diagnosed when bacterial culture and Gram stain are negative. A tentative diagnosis may be based on analysis of the CSF.

The WBC count in viral meningitis and encephalitis characteristically shows 10 to 1000 cells/mm3 . The differential cell count is predominantly lymphocytic and mononuclear in type. In the early stages of meningoencephalitis, however, polymorphonuclear cells may predominate, making the distinction between viral and bacterial infections difficult. In such cases, a repeat tap in 12 to 24 hours will assist in clarifying the diagnosis. Protein levels are usually mildly elevated, but normal levels may be seen. Antibiotic coverage pending culture results may be reasonably initiated if the diagnosis of viral meningitis is in doubt. [76] The CSF glucose is characteristically normal; however, notable exceptions include some cases of mumps meningoencephalitis and herpes simplex encephalitis. CSF pleocytosis and elevated protein levels have also been found in asymptomatic HIV-seropositive individuals. [100]

If the CSF cannot be delivered to the viral laboratory in 24 to 48 hours, it should be

refrigerated at 4 °C. Members of the enterovirus group are occasionally isolated from CSF. Herpesviruses and arboviruses are rarely found in CSF. In known viral CNS disease, the stool is more rewarding (85% positive) than CSF (10% positive). [101] Because CSF is normally sterile, any isolate is significant, whereas a stool isolate does not necessarily indicate that the agent is responsible for CNS disease. [84] The PCR is the diagnostic test of choice for herpes simplex meningoencephalitis and PCR will be increasingly applied to the diagnosis of other CNS viral infections.

## CSF Analysis in Immunocompromised Patients

The number of immunocompromised individuals is increasing because of the HIV epidemic and the increased survival of patients with cancer and autoimmune disorders. The nervous system is a major target of the HIV virus, with 40 to 60% of infected individuals developing neurologic disease during their lifetime; one third will present with neurologic complaints as the initial manifestations of the acquired immune deficiency syndrome (AIDS), and an even higher incidence of nervous system involvement is found at autopsy. [102] Risk of CNS infection depends on the underlying disease, treatment, duration, and type of immune abnormality. Abnormalities include defects in T-lymphocyte and macrophage cellular immune function, defects in humoral immunity, defects in number and function of neutrophils, and loss of splenic function with inability to remove encapsulated bacteria. [103] A summary of the major neurologic manifestations of HIV infection including their clinical characteristics is presented in Table 65-3 (Table Not Available) .

Patients with defects of their cell-mediated immunity include those with lymphoma, organ transplant recipients, daily corticosteroid therapy, and patients with AIDS. These individuals are vulnerable to infections with microorganisms that are intracellular parasites. The most common source of acute bacterial meningitis in such patients is *L. monocytogenes*. The clinical presentation includes fever, headache, seizures, focal neurologic deficits and a brainstem encephalitis.

Patients with defective humoral immunity include those with chronic lymphocytic leukemia, multiple myeloma, and Hodgkin's disease after radiotherapy or chemotherapy. These patients have difficulty controlling infection by encapsulated bacteria. They may develop a fulminant meningitis due to *S. pneumoniae*, *H. influenzae* type b, and *N. meningitidis*. Patients after splenectomy are at risk for the development of meningitis for the same reason. Neutropenic patients are at risk for meningitis due to *P. aeruginosa* and the Enterobacteriaceae.

Establishing a specific diagnosis in HIV and organ transplant patients may be difficult to impossible because of overlapping clinical and radiographic presentations, the presence of simultaneous infections with more than one organism, and CSF changes that may be nonspecific. The immune response may be altered with the absence of signs of meningeal irritation; patients may present with a diffuse encephalopathy or with focal neurologic deficits. CSF is abnormal in 60% of asymptomatic HIV-infected individuals, complicating CSF/clinical correlation. [102]

**Neurosyphilis in Patients with HIV Infection**

Both syphilis and HIV infection are sexually transmitted diseases, and patients with syphilis are probably at increased risk of HIV infection. CNS invasion is probably no more common in HIV-infected and HIV-noninfected patients. [104] Both diseases may have elevations in CSF WBC counts, protein levels, and gamma-globulin levels. Early CNS infection may be more common in patients with HIV infection. Reports also have noted an increased incidence of syphilitic meningitis, meningovascular syphilis, and ocular syphilis, which has been uncommon in the past. Studies suggest HIV patients treated for syphilis may have viable organisms in CSF after therapy or have persistent CSF VDRL titers. Treatment failures are more likely to occur with single-dose benzathine penicillin therapy.

Diagnosis of neurosyphilis may also be more difficult in HIV-infected patients. A small number of patients with secondary or ocular syphilis have been described with negative serum reagin tests. Positive treponemal tests may revert to nonreactivity after treatment, particularly in individuals with advanced symptomatic HIV disease and a low VDRL titer at the time of diagnosis. CSF pleocytosis and increased gamma-globulin levels may not help distinguish between HIV infection and CNS syphilis.

HIV-infected patients should have serum treponemal and nontreponemal tests early in their illness to minimize a false-negative result. [104] Infected patients should undergo a CSF examination. Previously treated patients without a CSF examination should have a lumbar puncture because of a probable increased risk of neurosyphilis even with a decline in serum nontreponemal titers. Asymptomatic patients with neurosyphilis should be treated to prevent CNS relapse.

**Cryptococcal Meningitis**

The most common CNS fungal infection is due to *Cryptococcus neoformans*. Approximately 5% of AIDS patients will develop infection with this common fungus. Clinical presentation in AIDS and non-AIDS patients include nonspecific symptoms of headache and altered mental status with or without meningeal signs. Most patients have increased ICP. Immunocompetent patients show a lymphocytic pleocytosis with CSF WBC counts under 500/mm3 . Glucose levels are depressed with elevation of CSF protein. India ink preparations are positive in 50% of cases. The CSF is less likely to be abnormal in HIV infection with normal cell counts and chemistries. However, CSF cultures and cryptococcal polysaccharide capsular antigens are almost always positive. False-positive antigen tests are rare but may be seen in the presence of rheumatoid factor. Blood cultures are frequently positive in AIDS patients. Cisternal puncture for fluid analysis may be helpful in undiagnosed cases of lymphocytic meningitis in which multiple lumbar punctures have not established a diagnosis. [102]

**Toxoplasmosis**

*Toxoplasma gondii*, an intracellular protozoan, is associated with CNS infection in up to

30% of AIDS patients who have antibodies to *T. gondii*. Most adults have antibodies; infection is believed to represent reactivation of latent primary infection. *Toxoplasma* encephalitis will develop within the first 2 years after the diagnosis of AIDS. Cerebral toxoplasmosis usually presents with the acute/subacute appearance of focal disease including seizures. MRI or CT shows multiple abscesses that may represent multiple pathogens. The distinction between toxoplasmosis and lymphoma may be difficult clinically. Less commonly, the presentation is of a chronic meningitis with confusion, memory loss, and lethargy similar to the AIDS-dementia complex. CSF in these patients is nonspecific with increased protein, mononuclear pleocytosis (<100 cells/mm3 ) and, rarely, reduced glucose concentration. Serum and CSF serologies may be either positive or negative and do not help make a diagnosis, although most patients with encephalitis have detectable IgG antibodies. Diagnosis is usually made based on clinical and imaging responses to antibiotics (pyrimethamine or sulfadiazine) or brain biopsy. Treatment failures with relapse occur in 50% of AIDS patients and 15 to 25% of non-AIDS patients, necessitating lifelong treatment.

**Mycobacterial Tuberculosis**

CNS mycobacteria infection is almost always the result of infection with *Mycobacterium tuberculosis*. Infection occurs in the setting of disseminated tuberculosis. Atypical *Mycobacterium* infection occurring in HIV-infected individuals uncommonly affects the CNS. Clinical presentation is that of meningitis (particularly involving the basal cistern), encephalitis, or abscess formation. If tuberculosis is suspected, a large volume of CSF (10 mL) is required for adequate culture. The cell count varies from 100 to 400 cells/mm3 , with a lymphocytic predominance; 30% may show predominately neutrophils early in the course of infection. Protein levels are elevated (100-500 mg/dL); CSF glucose level may be depressed. Acid-fast stains should be examined by experienced technicians. Fluid is inoculated onto Lowenstein-Jensen medium, and the absence of visible growth on the medium should not be considered negative for 8 weeks. CSF cultures are more sensitive than stains. Many of the current tests for mycobacteria are likely to be replaced by PCR tests. [103]

**Primary CNS Lymphoma**

The risk of developing CNS lymphoma in an AIDS patient is 2%. Presentation is that of a diffuse encephalopathy, although occasionally focal neurologic deficits occur. Leptomeningeal spread of malignancy is reflected in a modest lymphocytic pleocytosis with slightly elevated protein and decreased glucose. Cytologic yield is improved by repeat lumbar punctures and submitting large quantities of CSF or sampling fluid by cisternal puncture. [102]

**Progressive Multifocal Leukoencephalopathy**

Progressive multifocal leukoencephalopathy is an uncommon disorder in individuals with impaired cell-mediated immunity and is caused by reactivation of the JC papovavirus in the kidney. Progressive demyelination presents as combinations of dementia, blindness, aphasia, hemiparesis, and seizures that progress until death. MRI and CT demonstrate nonenhancing white matter lesions without mass effect. Definitive diagnosis is made by

brain biopsy, but CSF may show the presence of myelin basic protein, increased IgG, with acellular or a mild CSF pleocytosis (<50 WBCs/mm3 ). Average survival is 4 months. [103]

**Brain Abscess**

Brain abscesses also occur in the immune-deficient host. Diverse causes have been described as well as multiple pathogens. Lesions may be difficult to detect by CT with contrast enhancement, decreased by corticosteroid therapy and a diminished host inflammatory response. Organisms reported include *T. gondii*, *Nocardia asteroides*, *Cryptococcus neoformans*, *Mycobacterium* species, aerobic gram-negative bacteria, *Aspergillus* species, *Candida* species, and Zygomycetes.

**Cytomegalovirus Infection**

Cytomegalovirus may be detected in 30% of brains of HIV-infected persons who come to autopsy. A distinct CNS disorder has not been defined. CSF pleocytosis may be minimal. Retinitis and painful polyradiculopathies are recognized with a prominent CSF pleocytosis in the later condition.

# CONCLUSION

A spinal tap should only be performed when the treating physician believes the CSF specimen or specimens will be of diagnostic or therapeutic value, as in the case of patients with symptomatic pseudotumor cerebri. The procedure is often indicated in the diagnosis of meningitis or subarachnoid hemorrhage. Complications are uncommon, and risks are usually outweighed by the benefit of the procedure. Most contraindications are relative and not absolute, particularly if infection is an overriding consideration.

# Chapter 66 - Special Neurologic Tests

**J. Stephen Huff, Paul B. Baker**

## CALORIC TESTING

An accurate assessment of the comatose patient requires a thorough neurologic examination, including careful evaluation of the patient's responses to a variety of external stimuli. In a comatose individual with normal brainstem and cranial nerve function, stimulation of the vestibular labyrinth results in a well-described and reproducible compensatory deviation of the eyes. This response is known as *the vestibulo-ocular reflex* and forms the physiologic basis for the caloric test of the vestibular system. During caloric testing, a thermal stimulus, usually water at a specific temperature, is delivered to the external auditory canal to activate the labyrinth and produce the characteristic ocular movements. Pathologic conditions involving either the vestibular or oculomotor reflex pathways will alter or abolish the response to caloric stimulation.

Caloric tests are performed in both conscious and unconscious patients, depending on the diagnostic circumstances. Quantitative caloric examination is conducted in the ambulatory patient for evaluation of possible vestibular dysfunction. This type of testing requires precisely controlled irrigation temperatures and specialized recording devices and is best undertaken in a properly equipped laboratory under the supervision of an experienced neuro-otologist. However, the neurologist, neurosurgeon, or emergency physician may perform *qualitative* caloric testing in the comatose patient for detection of gross disruption of vestibulo-ocular reflex pathways indicative of structural lesions or metabolic abnormalities involving the brainstem. In this setting, large quantities of ice water provide maximal stimulation of the vestibular apparatus. Such testing needs no special expertise and can be done at the bedside using equipment readily available in the emergency department (ED). This simple procedure can provide valuable diagnostic and prognostic information necessary for management of the comatose patient.

### Background

In the middle of the 19th century, Brown-Sequard first described the effects of introducing cold water into the ear canal. [1] The clinical importance of the phenomenon was first realized in 1906 by Barany, who developed a caloric procedure using an ice water stimulus. [2] He postulated, correctly, that caloric stimulation of the auditory canal induced formation of convection currents within the semicircular canals of the vestibular labyrinth.

Different methods of caloric testing were later proposed by Kobrak and others, but standardization of the procedure awaited the introduction of the Fitzgerald-Hallpike technique in 1942. This technique, which uses both warm and cool water stimuli under rigidly specified conditions, permits quantification of normal and abnormal caloric responses. Today, most formal caloric testing of conscious patients is based on

variations of the original Fitzgerald-Hallpike procedure. [3]

The value of caloric testing in the assessment of the comatose patient was emphasized by the work of Klingon and Bender and associates in the 1950s. Vaernet and Ethelberg studied the changes in caloric reactions during transtentorial herniation of the brainstem. Blegvad reported the effects of barbiturate intoxication on the vestibulo-ocular reflex. [8]

More recent advances include the development of electronystagmography (ENG), which provides a graphic record of reflex eye movements and permits precise determination of the intensity of the caloric response. [9] Some authorities now advocate performing caloric testing at a single temperature (monothermal calorics) as an equally sensitive and less time-consuming alternative to standard bithermal calorics. [10] Others have recently investigated the use of heated air as an alternative to traditional water caloric tests. [11]

**Physiology and Functional Anatomy**

Proper performance and interpretation of the caloric test require a basic understanding of the structure and function of both the vestibular and oculomotor systems. The anatomic pathways underlying the vestibulo-ocular reflex begin in the posterior portion of the labyrinth of the inner ear. The peripheral vestibular apparatus is located within the temporal bone and consists of the utricle, the saccule, and the lateral, anterior, and posterior semicircular canals (Fig. 66-1) (Figure Not Available) . Because of its proximity to the external ear canal, the lateral or horizontal canal is of principal interest in caloric testing. Note that the lateral canal is oriented at a 30° angle to the horizontal (Fig. 66-2) (Figure Not Available) . Deflections of the cupula due to movement of endolymphatic fluid within the canal result in polarization changes in the underlying hair cell, which in turn are relayed to the afferent limb of the primary vestibular neuron.

Impulses of the primary neuron travel via Scarpa's ganglion and cranial nerve (CN) VIII to the brainstem to synapse with secondary vestibular neurons in the superior and medial vestibular nuclei of the upper medulla and lower pons (Fig. 66-3) (Figure Not Available) . Although the connections between the vestibular and oculomotor nuclei in the brainstem are quite complex, two main pathways exist. The direct projection runs from the vestibular complex to the nuclei of CN III and VI via the medial longitudinal fasciculus (MLF) and involves only three neurons: (1) primary vestibular, (2) secondary vestibular, and (3) oculomotor. [12] The indirect projection between these nuclei occurs over multisynaptic circuits in the tegmental reticular formation. [13] Another brainstem structure that contributes to the vestibulo-ocular interaction is the parapontine reticular formation (PPRF), a poorly characterized group of pontine neurons that coordinates both voluntary and involuntary lateral gaze. The PPRF receives multiple inputs, including projections from the vestibular system and the contralateral frontal cortex, and sends output to oculomotor neurons through both the direct and indirect pathways. Excitatory impulses originating in the lateral canal finally travel via the oculomotor and abducens nerves to the ipsilateral medial rectus and contralateral lateral rectus muscles.

Rotation of the head generates flow of endolymphatic fluid within the semicircular canals. The firing rate of the primary vestibular neuron is dependent on the direction of flow. For example, in the lateral canal, flow toward the ampulla (ampullopetal) increases the firing rate, whereas flow away from the ampulla (ampullofugal) decreases the firing rate. [14] Increased firing on one side results in conjugate deviation of the eyes toward the opposite side, whereas decreased firing causes deviation to the same side. This principle forms the physiologic basis of caloric testing. When the lateral canal is placed in the vertical position and ice water is infused into the ear, the endolymph nearest the canal cools and sinks, resulting in ampullofugal flow (Fig. 66-4) (Figure Not Available) . As the firing rate decreases, the eyes deviate conjugally toward the side of irrigation. Likewise, if warm water were used in the same position or if the canal were inverted 180°, the opposite would occur.

Eye movements induced by caloric stimulation in conscious, neurologically normal individuals are more complex. Ice water infusions induce a rhythmic jerking of the eyes that includes a slow deviation toward the irrigated side followed by a quick compensatory saccade toward the midline. This is known as *caloric nystagmus.* By convention, *caloric nystagmus is named for the fast component*, thus the popular mnemonic "fast COWS" ( *C*old irrigation-- *O*pposite beating nystagmus; *W*arm irrigation-- *S*ame-sided beating nystagmus). Most sources attribute the slow phase of nystagmus to vestibular activity transmitted over the direct pathway, whereas the fast phase is believed to be generated by the PPRF in conjunction with cortical activity and carried over indirect pathways within the reticular formation. Numerous factors, including physiologic, pharmacologic, and pathologic factors, can alter caloric-induced eye movements.

**Indications and Contraindications**

Caloric testing of the comatose patient is indicated when the physician needs information regarding the functional integrity of the brainstem. When the cause of the coma is initially unknown, caloric testing may assist in differentiation among structural, metabolic, and psychogenic causes for unresponsiveness. Even when the cause is clear, caloric testing may provide an indication of the depth of coma and the prognosis for eventual recovery.

In conscious patients who complain of vertigo, caloric testing may be indicated for the nonemergency evaluation of possible vestibular disorders. Ice water produces maximal stimulation of the labyrinth. This can be quite painful and may induce nausea and vomiting in awake, susceptible individuals. These patients are best referred to a qualified neuro-otologist, who can conduct more accurate testing in the ENG laboratory with much less discomfort to the patient.

There are few contraindications to caloric testing. An absolute contraindication is the presence of a basilar skull fracture, either documented radiologically or suspected by clinical signs, because of the risk of introducing infection into the central nervous system (CNS) through an associated dural tear. If bilateral fracture can be readily excluded, testing of the intact ear with both warm and cold water will yield results similar to those

of the standard bilateral examination.

Relative contraindications to water caloric testing include perforations of the tympanic membrane (those not due to temporal fractures), otitis media and externa, and the presence of previous otologic surgery (e.g., mastoidectomy). Although the risk of otitis media is probably small, carrying out the caloric test in the comatose patient under these conditions remains a matter of clinical judgment. When available, auditory evoked potentials provide an alternative source of information when caloric testing is contraindicated.

## Equipment

The equipment needed for performance of the caloric test is minimal and is readily available in the ED or hospital ward. Although almost any size syringe will suffice, a 12- or 35-mL plastic syringe is ideal for irrigation. The syringe may be used as is, or a short length of soft plastic tubing may be attached. A good source of tubing is a butterfly catheter with the needle cut or pulled off. At least a hundred milliliters of ice water should be available, although larger quantities of cool tap water (<25° C) can be used with similar results if ice is unavailable. Sterile or bacteriostatic saline may be used, although its advantage over tap water has not been shown. A small plastic emesis basin is useful to collect water as it drains from the ear canal.

Additional required equipment includes an otoscope, several sizes of ear speculums, and equipment for removal of cerumen. Towels and a thermometer that reads from 0° to 50° C are also helpful.

## Procedure

Caloric testing should be deferred until the patient's condition has been stabilized, including protection of the airway and evaluation of the cervical spine in trauma patients. A thorough neurologic assessment should be performed prior to caloric testing, with special attention given to the ocular examination. Pupillary responses, spontaneous ocular movements, and resting eye position should be accurately recorded. The ears should be inspected prior to insertion of the otoscope. If active bleeding or CSF otorrhea or rhinorrhea is noted in the trauma victim, caloric testing and further otoscopic examination should be curtailed and the patient should be treated for a probable basilar skull fracture. If the external ear canal appears normal, the otoscopic examination should be completed. Signs of active ear infection or perforation of the tympanic membrane are contraindications to caloric testing. Tympanic rupture, hemotympanum, and step deformities of the canal may indicate fracture of the temporal bone; caloric testing in this situation is contraindicated (Fig. 66-5) (Figure Not Available) . Excess cerumen and foreign material must be removed, and the tympanic membrane must be clearly visualized.  The ear speculum may be left in the canal as a guide for irrigation.

The patient should be placed in a supine position. Ideally, the head or upper body should be raised 30° (2 pillows will provide the appropriate angle). This angle places the lateral canal in the vertical position and ensures a maximal response. The patient should be draped with a towel, and a small basin should be positioned below the ear to

collect the water outflow. A container should be filled with ice water and placed near the bedside.

The syringe and catheter system *(minus the needle)* should be filled with 10 mL of ice water, and the irrigation stream should be directed at the upper posterior portion of the tympanic membrane. Because the goal of the qualitative caloric test is to induce a maximum response, the amount and rate of infusion are not critical. As a general guide, 5 to 10 mL of ice water should be initially infused over a period of 5 to 10 seconds; amounts less than 5 mL may be advisable in suspected cases of light coma or psychogenic unresponsiveness. If no response is noted within 1 or 2 minutes, at least 100 mL should be infused before declaring that there is no response. Testing of the contralateral ear may begin 5 or 10 minutes after the eyes have returned to their original position. At the conclusion of the testing, the otoscopic examination may be repeated to check for blanching of the tympanic membrane, a sign that the irrigation stream was properly directed.

Observation of eye deviation is easier when an assistant holds the patient's eyelids open. Movement usually occurs after a latency of 10 to 40 seconds, with persistence of the response for as long as 4 or 5 minutes. Small deviations may be detected by focusing on a small scleral vessel. Alternatively, a dermographic pencil may be used to mark the initial position of the pupil with respect to the eyelid. Variations of the caloric technique may be useful in certain situations. Warm water caloric testing may be performed if no response to bilateral ice water caloric testing is obtained or in cases in which only one ear can be tested. Water temperature should be kept below 50° C. The response elicited will be the opposite of that obtained with ice water. In patients who fail to respond to ice water caloric testing alone, additional stimulation may be obtained by combining irrigation with repeated head turning away from the irrigated side ( *doll's-eye maneuver*). *Obviously, cervical injury must be excluded prior to using this technique*. This combination of techniques may produce eye movements in patients who do not respond to caloric testing alone. [15] Eviatar and Goodhill have described a technique for caloric testing in tympanic perforations using a small latex finger cot placed in the ear canal to prevent water from entering the middle ear. [16]

The technique of bilateral caloric testing is used to evaluate vertical gaze disorders and involves the simultaneous delivery of equal amounts of ice water to both ear canals. A large syringe can be attached to a Y-connector, or two separate syringes can be used. Two persons are needed to administer the caloric challenge properly. In normal individuals, upward-beating nystagmus is seen after a latency of 1 to 3 minutes (as expected, the opposite is seen with warm water). The mechanism of this phenomenon is not clear, but it may involve stimulation of the remaining 2 semicircular canals.

**Complications**

The few complications that are possible with caloric testing can be avoided by carefully selecting both the patients to be tested and the equipment and technique to be used. Using needles or other sharp objects to irrigate the ear may result in laceration or perforation of the tympanic membrane or canal wall if the patient moves unexpectedly.

The use of plastic syringes and soft catheter tubing aid in reducing such occurrences.

Other potential complications of caloric testing include otitis media, meningitis, and the induction of vomiting and subsequent aspiration. Ice water irrigation in the presence of tympanic membrane perforation increases the chance of middle ear infection; however, the incidence of this complication following caloric testing has not been reported. Meningitis may follow basilar skull fractures with meningeal tears; the additional risk of calorics in such situations is not known. Therefore, caloric testing should be omitted in the head-injured patient if there is any suspicion of temporal fracture. Although ice water irrigation may produce nausea and even emesis in a few awake patients, vomiting or aspiration has not been reported as a complication of caloric testing in the comatose patient. Nevertheless, it is advisable to delay testing until the patient's airway is protected.

## Interpretation

### The first phase of interpretation.

Analysis of initial eye position and spontaneous movements should be made prior to irrigation. The eyes of comatose patients with intact oculomotor pathways are usually directed straight ahead or are slightly divergent. Unilateral destructive lesions of the cerebral hemisphere can cause conjugate deviation of the eyes *toward* the side of the lesion, whereas irritative foci, as might be seen in status epilepticus, can cause conjugate deviation *away* from the affected side. Deviations of this type can usually be overcome by caloric stimulation, although combined irrigation and head turning may be required in the first hours following the insult. Lesions in or near the PPRF in the brainstem cause conjugate deviation *away* from the side of the lesion that usually cannot be overcome by calorics. Conjugate downward deviation can be seen with structural lesions of the brainstem or in the deeper phases of metabolic coma. Dysconjugate gaze either indicates damage at the level of the oculomotor nuclei or below or reflects disruption of the ocular muscles themselves. [17] Dysconjugate gaze may also be seen in drug-induced coma in the presence of a structurally intact brainstem pathway. In the very late stages of brainstem dysfunction, the eyes usually return to the central position. Spontaneous roving movements of the eyes, either conjugate or dysconjugate, may be seen in supratentorial insults, but these, too, disappear with brainstem involvement. [15] Ocular "bobbing" is an intermittent, spontaneous downward jerking of the eyes that may occur with massive pontine lesions. Paralysis occurs of both voluntary and reflex lateral gaze, and caloric stimulation may increase the rate of bobbing without causing lateral deviation of the eyes. [18] Ocular "dipping" is a more prolonged, downward conjugate deviation of the eyes and has been reported in cases of severe anoxic encephalopathy (e.g., carbon monoxide poisoning). [19] The pathophysiologic basis of these eye movements is poorly understood. [20]

### The second phase of interpretation.

Analysis of ocular movements after caloric irrigation. Reactions to ice water stimuli may be divided into four categories: (1) caloric nystagmus, (2) conjugate deviation, (3) dysconjugate deviation, and (4) absent responses (Fig. 66-6 (Figure Not Available) *A*

and *B*). The first reaction, caloric nystagmus with the fast component beating away from the side of ice water irrigation, is seen in normal, alert individuals; in cases of psychogenic unresponsiveness; and in those who have very mild organic disturbances of consciousness. The intensity of nystagmus is highly variable in conscious subjects and depends on the degree of visual fixation and the level of mental alertness. The response is present in more than 90% of children by the age of 6 months and declines in magnitude only after the seventh decade of life.  Corneal reflexes and responses to facial pinprick may be abolished by hypnotic suggestion in susceptible individuals; however, caloric nystagmus appears to be immune to similar manipulation. [22]

Caloric nystagmus in the apparently comatose individual is the usual result of caloric testing in cases of psychogenic unresponsiveness due to catatonia, hysterical conversion, schizophrenia, and malingering. Hyperactive caloric responses result from testing in the presence of tympanic perforation and mastoid disease. Hypoactive caloric responses are recorded in a wide variety of vestibular and neurologic disorders.

Hypoactive or abnormal caloric responses may be further evaluated with quantitative caloric testing and ENG. Caloric nystagmus may be *inverted* (beating to the wrong side) or *perverted* (beating in the wrong plane); both responses are seen in brainstem lesions. *Pseudocaloric nystagmus* is a preexisting latent nystagmus that is brought out by the general arousal of ice water irrigation; it can be distinguished from true nystagmus by its failure to reverse direction with warm water irrigation.

As the level of coma deepens, the fast phase of nystagmus becomes intermittent and finally disappears, probably as a result of decreased activity in the cerebral cortex.

In the second type of caloric response, the eyes deviate conjugately toward the side of ice water stimulation (they "look" toward the source of irritation). When present, this reaction indicates intact brainstem function. This is seen during general anesthesia, in supratentorial lesions *without brainstem compression*, and in many metabolic and drug-induced comas. In such situations, bilateral simultaneous irrigation with ice water may result in conjugate downward deviation, implying that brainstem centers for vertical gaze are functional.

Dysconjugate reactions constitute the third type of caloric response to ice water stimuli. The most common dysconjugate reaction is internuclear ophthalmoplegia, in which a lesion of the MLF causes weakness or paralysis of the *adducting* eye following caloric irrigation. Internuclear ophthalmoplegia may be due to acute damage to the rostral pons or may be seen as a manifestation of multiple sclerosis or previous vascular insult.

In acute supratentorial lesions, the development of dysconjugate caloric responses is a significant sign that may indicate compression of the brainstem and impending herniation. Caloric responses of this type are less common with metabolic and drug-induced coma, and when present in metabolic coma, their significance is less ominous. Reversible internuclear ophthalmoplegia has been reported in hepatic coma and may occur during toxic response to phenytoin, barbiturate, or amitriptyline. Forced downward deviation of the eyes, either conjugate or dysconjugate, may be seen in

sedative-hypnotic drug-induced coma when unilateral caloric testing is performed. [23]

Palsies of the oculomotor nerves are another cause of dysconjugate reactions, although most should be apparent before irrigation. Causes include diabetic neuropathy (especially CN VI), increased intracranial pressure, and Wernicke's encephalopathy. Finally, Plum and Posner report that unusual and poorly characterized caloric responses may be obtained from the testing of comatose patients with long-standing severe brain injury. [17]

Absent caloric responses are the fourth category of reactions to ice water stimuli. As a general rule, the oculovestibular response is preserved longer than other brainstem reflexes; however, the oculocephalic or doll's eye response may persist in the absence of caloric responses due to bilateral labyrinthine disease because of additional input from proprioceptive receptors in the neck. Loss of caloric responses in comatose patients with structural lesions is usually a sign of brainstem damage. In supratentorial lesions, progressive loss of caloric responses may be seen in the final stages of transtentorial herniation. The oculovestibular reflex may also be transiently absent or decreased on the side opposite massive supratentorial damage during the first hours following injury. [24] Absent caloric responses may occur in any subtentorial lesion that affects vestibular reflex pathways, including pontine hemorrhage, basilar artery occlusion, cerebellar hemorrhage, or infarction with encroachment on the brainstem, and in any expanding mass lesion within the posterior fossa. Calorics may disappear in deep coma resulting from subarachnoid hemorrhage, perhaps owing to pressure on the brainstem.

The vestibulo-ocular reflex is usually retained until the late stages of metabolic coma. Disappearance of the reflex, as in the case of hepatic coma, is frequently a preterminal event. [25] Nevertheless, caloric responses may be transiently absent in certain types of drug-induced coma, with the *eventual complete recovery of the patient.* The vestibulo-ocular reflex seems particularly sensitive to the effects of sedative-hypnotic drugs (e.g., barbiturates, glutethimide), antidepressants (e.g., amitriptyline, doxepin), and anticonvulsants (e.g., phenytoin, carbamazepine). [8] [26] [27] Obviously, neuromuscular blocking agents (e.g., succinylcholine) will abolish caloric-induced ocular movements.

Finally, the caloric response may be absent for reasons other than the neurologic causes responsible for the coma. Inadequate irrigation due to excess cerumen or poor technique and unilateral or bilateral dysfunction of the peripheral vestibular apparatus must be considered. Bilateral loss of caloric response (arreflexia vestibularis) is uncommon in conscious patients, constituting 1.7% and 0.2%, respectively, of the ENG clinical population in two large series of patients. [28] [29] Some of the causes of unilateral and bilateral loss of oculovestibular reflexes in conscious patients are listed in Table 66-1 .

| TABLE 66-1 -- Possible Causes of Absent Vestibulo-ocular Reflex in Conscious Patients | |
| --- | --- |
| Inadequate Irrigation<br>Cerumen Impaction<br>Postinfection<br>Meningitis<br>Encephalitis<br>Syphilis<br>Neoplastic<br>Acoustic neuroma<br>Other cerebellopontine-angle tumors<br>Posterior fossa tumors<br>Inflammatory<br>Systemic lupus erythematosus<br>Cogan's syndrome | Traumatic<br>Previous temporal fracture<br>Previous head injury<br>Postlabyrinthectomy<br>Labyrinthine<br>Vestibular neuronitis<br>Suppurative labyrinthitis<br>Congenital<br>Congenital hydrocephalus<br>Hereditary spinocerebellar degeneration<br>Idiopathic<br>Drugs<br>Aminoglycoside antibiotics<br>Neuromuscular blocking agents<br>Anticonvulsants [*] |

* Reported rarely in conscious patients who have taken more than normal therapeutic dosage.

The vestibulo-ocular reflex has *prognostic* as well as diagnostic significance in the comatose patient. In a study of 100 patients who were comatose from head trauma, absence of calorics at 1 to 3 days following injury was associated with extremely high mortality. [30] Testing in the immediate post-traumatic period may yield inconsistent responses and is of considerably less prognostic value. Levy and coworkers studied 500 cases of nontraumatic, non-drug-induced coma in a large multicenter effort. Absence of the vestibulo-ocular reflex correlated with less than a 5% chance of achieving functional recovery within 1 year when tested within 6 to 24 hours of coma onset. [31] In one study of comatose patients, the combination of absent vestibulo-ocular reflex and absent pupillary light reflex at 24 hours was associated with a 100% mortality. [32] Complete loss of caloric responses is part of the criteria for the legal diagnosis of brain death in many localities and correlates with the irreversible cessation of cerebral function at least as well as an isoelectric electroencephalogram (EEG). [33] Excessive reliance on a single clinical sign must be avoided, and decisions regarding neurologic prognosis and future therapy should be based on complete consideration of all evidence available to the physician.

**Summary**

The caloric test is a simple, easily performed procedure that may enhance the neurologic assessment of the comatose patient, unless otherwise contraindicated. In the

emergency patient, this test should be reserved for the stable patient undergoing secondary assessment. Even when the cause of the coma is known, the test can provide a baseline for the evaluation of future changes in the patient's status. The examination requires minimal equipment and can be conducted in a few minutes while awaiting laboratory results or during preparation for computed tomographic scanning. Complications are few if patients are properly selected and correct technique is used. When reliably interpreted, caloric testing furnishes valuable diagnostic and prognostic information necessary for proper care of the comatose individual.

## MYASTHENIA GRAVIS TESTING

Myasthenia gravis is the most common disease of neuromuscular transmission, yet its incidence is only 1 in 20,000 in the general population. Patients with myasthenia may be grouped into two major categories. The first group shows weakness in proximal muscles, which increases with activity and later improves with rest. The other group presents with ocular complaints of diplopia or ptosis. Patients in this group may or may not have generalized symptoms as well. Fatigue is the hallmark of the disease; symptoms seem to wax and wane. Patients frequently see a number of physicians before a correct diagnosis is made.

Myasthenia gravis results from an immune-mediated decrease of postsynaptic acetylcholine (ACh) receptors, with variable failure of neuromuscular transmission.  ACh is the transmitter at the neuromuscular junction. When the nerve terminal is stimulated, ACh is released in a quantity far in excess of that needed for effective activation of the ACh receptor. The ACh diffuses across the synaptic cleft to transiently interact with the ACh receptor, and an electrical potential is generated at the myoneural end plate. If it is of sufficient magnitude, the end plate potential initiates an action potential that is propagated along the muscle membrane and leads to muscle fiber contraction. The ACh is rapidly hydrolized by acetylcholinesterase in the synaptic cleft. Figure 66-7 (Figure Not Available) summarizes neurotransmitter action at the neuromuscular junction. Of the millions of receptors at each myoneural junction, only a fraction must depolarize to stimulate muscle fiber contraction. Any factor that decreases the chance of interaction of ACh with ACh receptor decreases the probability of an action potential being generated. Acetylcholinesterase inhibitors (anticholinesterases) have been the mainstay of therapy for myasthenia gravis for years, but they have been supplemented by aggressive immunosuppressive regimens and thymectomy. Failure of neuromuscular transmission may also occur with *excessive* acetylcholinesterase inhibition; the persistence of ACh in the synaptic cleft leads to continuous depolarization of the receptor.

A patient with myasthenia gravis may present to the ED in one of two ways that might necessitate bedside diagnostic testing. The first presentation involves a known myasthenic patient on cholinesterase inhibitor therapy with increased weakness; pharmacologic testing in this setting is controversial and is discussed in the following section. The second presentation involves a previously undiagnosed patient in whom the diagnosis of myasthenia gravis is suspected with ptosis, diplopia, or fluctuating muscular weakness.

Various tests are available to help with the assessment of this second presentation.

ACh receptor antibody assay is now readily available and positive in more than 80% of patients. Repetitive nerve stimulation and single-fiber electromyography tests are available in the electrophysiology laboratory. [40] Several pharmacologic tests have been used, including parenteral administration of edrophonium chloride (Tensilon), neostigmine, and curare. Curare has been used systemically but can also be administered into an ischemic arm; this modification is known as the *regional curare test.* [40] Many of these tests are seldom used, or their performance falls in the realm of the neurologist's expertise. Edrophonium administration for diagnosis of myasthenia gravis (Tensilon test) is described in detail because of the drug's rapid onset, short duration of action, and widespread acceptance for this diagnostic challenge. The "ice pack" test also is included because of favorable reports of its utility and the noninvasive nature of the test.

## Edrophonium (Tensilon) Test

### Background

Edrophonium chloride (Tensilon) is an acetylcholinesterase inhibitor that has been used in the diagnosis of myasthenia gravis for more than 30 years. The short duration of action of edrophonium that made it unsatisfactory as a therapeutic agent makes it useful as a diagnostic agent. The drug's onset of action is rapid, and the duration of maximal effect is short, usually less than 2 minutes. Any effect resolves within 5 to 10 minutes. [43]

Edrophonium administration has been recommended in the past to monitor acetylcholinesterase inhibitor therapy. However, edrophonium is not sufficiently reliable for titrating the effect of anticholinesterase medication. [46]

Myasthenic crisis may be loosely defined as respiratory distress in a patient with myasthenia gravis. Earlier concepts included "myasthenic" crisis from insufficient drug administration, "cholinergic" crisis from cholinesterase inhibitor overdosage, and the "brittle" patient with rapidly changing drug requirements, but terminology in this setting remains controversial. [49] Even the clinical existence of the different types of crisis has been debated. [46] Certainly, in the myasthenic patient with respiratory distress, airway management and assisted ventilation are top priorities. Edrophonium administration should not be viewed as a possible alternative to intubation and ventilation. Several authorities advocate withdrawing all cholinesterase inhibitors in this setting because most patients show increased responsiveness to cholinesterase inhibitors after several days without taking the drug.Others advocate a trial of edrophonium chloride at reduced dosage (1 to 2 mg) only after respiratory support is achieved.

### Indications and Contraindications

The bedside Tensilon test is indicated for diagnosis in patients suspected of having myasthenia gravis. A muscle that is clearly weak must be identified. Ptosis is a commonly monitored sign. A clinically evident extraocular muscle weakness is another clinical example that might allow direct observation of a single weak muscle becoming stronger in response to the drug. Simple grip dynamometry does not aid in evaluation; a

repetitive measure of grip strength (ergogram) is necessary. If a specific muscle cannot be isolated for objective testing, edrophonium administration should be deferred and other approaches to diagnosis pursued, as discussed previously.

A history of asthma or cardiac dysrhythmias is a relative contraindication to administration of cholinesterase inhibitors. Administration of edrophonium to a patient with myasthenia gravis being treated with cholinesterase inhibitors is controversial, and many investigators consider myasthenic crisis to be a contraindication to edrophonium administration, though series of patients with myasthenic crisis continue to report its use.

### Equipment

The following materials are needed for testing an adult. Intravenous access should be secured with $D_5 W$ at a keep-open rate or with a saline lock. Ten milligrams of edrophonium chloride (Tensilon) should be drawn up in a tuberculin syringe. Edrophonium chloride is supplied in 1- and 10-mL vials at a concentration of 10 mg/mL. A second syringe of normal saline should be available to administer as a placebo, although some clinicians have recommended nicotine, calcium chloride, or atropine for this purpose. [45] Atropine and other cardiovascular drugs and resuscitative equipment should be readily available. Cardiac monitoring is generally recommended. Photographic recording equipment is desirable to objectively document improvement in motor function.

### Procedure

Ideally, one person is available to administer the edrophonium or placebo and a second person is free to observe the effect of medication on the patient. It is best if both the person evaluating the response and the patient do not know which syringe contains edrophonium and which contains saline, thus creating a double-blind testing situation.

The paretic muscle to be tested is identified. Ptosis is an easily testable sign and is generally used when present. The principles involved with assessing the effect of edrophonium on ptosis may be extended to testing other muscles. The patient is instructed to look upward for several moments in an attempt to fatigue the levator muscles. The degree of ptosis is noted and documented by measurements or photographs. After a moment's rest, with the patient looking straight ahead, 0.2 mL of the test substance in one syringe is injected (2 mg of edrophonium if active substance). If there is no response within 1 minute, another 0.3 mL is injected (3 mg of edrophonium if active substance). If there is still no response, the remainder of the syringe is injected 3 minutes later. Any increase in strength reflected by an increase in palpebral fissure size is noted. The procedure may then be repeated with the other test substance.

### Complications

A small percentage of individuals are hypersensitive to even the initial small dose of edrophonium and show cholinergic side effects of salivation, lacrimation, and miosis.

These effects are transient. Atropine, 0.5 mg, may be given IV if necessary to counteract these symptoms. A smaller number of patients may experience symptomatic bradycardia that responds to atropine.

**Interpretation**

The key to the procedure is in its interpretation. If a clearly paretic muscle has been identified, objective signs of improvement in the strength of that muscle within a moment of administration of edrophonium and the fading of that improvement over the next 5 minutes are criteria for a positive test result. Up to 90% of patients with myasthenia have a positive test result under ideal circumstances. False-negative results do occur consistently.

For evaluating the effect of edrophonium on ptosis, a positive test consists of the patient having increased ability to elevate the eyelids after administration of 5 to 10 mg of edrophonium. The ptosis returns within 5 minutes. Subjective increases in general strength or relief of fatigue do *not* constitute a positive test. Fasciculations, brief twitches of muscles, are not usually observed in the patient with myasthenia who has received edrophonium, in contrast to normal subjects. The test may be repeated in 30 minutes if necessary.

Normal subjects have no change in muscle strength. They may transiently experience the side effects of salivation, lacrimation, and diaphoresis. Perioral, periocular, or lingual fasciculations are almost always noted in the normal patient following edrophonium administration.

The reproducible and unequivocal reversal of weakness in a specific muscle is extremely specific for myasthenia. False-positive test results have been reported in patients with Eaton-Lambert syndrome and rarely in patients with intracranial lesions. Other rare reports of positive test results involve patients with amyotrophic lateral sclerosis. A "perverse" reaction has been noted rarely, in which a paretic extraocular muscle weak from other causes becomes even weaker with edrophonium administration. [56]

## Ice Pack Test

**Background**

It has been observed clinically that myasthenic patients have exacerbations of weakness with environmental heat and improvement in strength with cold temperatures. A simple bedside test uses these observations to evaluate ptosis. [41] [42] Ice placed in a surgical glove or wrapped in a towel is placed lightly over the eyelid of a patient. Cooling of the eyelid below 29° C is accomplished within 2 minutes. The ptosis has been noted to improve in 80% or more of patients tested and may be more sensitive than the edrophonium test in detecting ocular myasthenia gravis. Although the reported number of patients evaluated by this method continues to be small, the test is included here because of its potential application in the ED, its lack of side effects, and its noninvasive

nature.

**Indications**

Unilateral or bilateral ptosis of uncertain etiology in which myasthenia is a diagnostic possibility is the sole indication for this test.

**Procedure**

Ice and a surgical glove or towel are the only materials required. A camera to record any response is optional.

The degree of ptosis of the patient is noted and, ideally, photographed. Momentary upward gaze may be used to provoke the ptosis. If bilateral ptosis is present, the more affected eye should be tested. Eyelid cooling is accomplished by lightly holding the wrapped ice to the patient's eyelid for 2 minutes or until patient discomfort limits the application. The width of the palpebral fissure is immediately compared with the pretest width (Fig. 66-8) (Figure Not Available) .

**Complications**

Patient discomfort from the ice pack application may limit the cold exposure time to less than 2 minutes but still may allow a successful test.

**Interpretation**

A clear improvement of ptosis in the cooled eye is the criterion for a positive test. The effect should be reproducible. In small clinical studies, the ice pack test is at least as sensitive as edrophonium administration in improving ptosis in patients with ocular myasthenia. False-negative results do occur, probably at about the same frequency as those in Tensilon testing. One individual has been reported to have had a negative ice pack test result with a positive Tensilon test result. Negative or equivocal Tensilon tests have been reported in other individuals who had clearly positive ice pack test results. Normal individuals showed no change in palpebral fissure width after the cold exposure. There are no reports of false-positive results.

## Summary

The bedside Tensilon test has a long history of utility in diagnosing myasthenia gravis, but it has been largely replaced by acetylcholinesterase receptor assay and electrodiagnostic studies in the ambulatory setting. On occasion when rapid diagnosis is desired or myasthenia is suspected in the presence of a normal ACh receptor titer, a carefully performed Tensilon test is still clinically valuable. The use of the Tensilon test in the setting of myasthenic crisis is controversial and is discouraged.

The ice pack test is so simple and noninvasive that it should become the initial

procedure of choice in the ED for evaluating the possibility of ocular myasthenia. A positive ice pack test result strongly suggests ocular myasthenia gravis and alleviates any need for the Tensilon test. False-negative results do occur, and additional testing should be performed if the clinical suspicion of myasthenia gravis is strong.

It is often the case with neuromuscular diseases, given the broad diagnostic possibilities, that the emergency physician is unlikely to establish a confident diagnosis at a single patient encounter. [58] Appropriate consultation and referral are necessary.

# Chapter 67 - Ophthalmologic Procedures

**John R. Samples, Jerris R. Hedges**

The following discussion focuses on procedures commonly performed by emergency physicians during the evaluation and treatment of many eye injuries and diseases. The emphasis is on the practical application of the techniques, and cautions to be heeded by the emergency physician are included.

## DILATING THE EYE

Dilating the eye is an essential step in the management of common eye emergencies. It is useful for both diagnostic and therapeutic purposes. Angle-closure glaucoma may be precipitated by dilating the pupil of the patient with a narrow angle. This is not a concern in patients with open-angle glaucoma, the most common form of glaucoma. However, some patients may have a "mixed-mechanism" glaucoma with both open-angle and narrow-angle components. Systemic reactions can be produced by mucosal absorption of dilating medications. To minimize complications, the correct agent must be chosen and the proper technique used.

There are 2 types of dilators: sympathomimetic agents, which stimulate the dilator muscle of the iris, and cycloplegic agents, which block the parasympathetic stimulus that constricts the iris sphincter. Cycloplegic agents also block the contraction of the ciliary muscles, which control the focusing of the lens of the eye. This second effect of cycloplegic agents is of great importance in the therapeutic use of dilators for iritis.

Cycloplegic agents were used cosmetically as early as Galen's time. Beginning in the early 1800s, extracts from the plants *Hyoscyamus* and belladonna were used in ophthalmology. Atropine was first isolated in 1833. Epinephrine was used on eyes in 1900 as the first sympathomimetic agent. [1]

**Indications and Contraindications**

There are diagnostic and therapeutic indications for dilating the pupil. Dilation is indicated for *diagnosis* when the fundus cannot be examined adequately through an undilated pupil. Such a situation is presented by the elderly patient with miotic pupils and cataracts. Dilation is *therapeutically* useful for many ophthalmic conditions, including inflammation in the eye. In the emergency setting, corneal injury with a secondary traumatic iritis is a common example. Dilation helps the inflamed eye in 2 ways. First, it keeps adhesions (synechiae) from forming between the iris and other ocular structures. Such adhesions eventually limit the movement of the pupil and may precipitate glaucoma. Second, cycloplegic dilating agents relax the ciliary muscle spasm that often accompanies an inflamed eye and thus reduce the pain associated with inflammation.

Whenever dilation is performed, it is important to note on the patient's chart the dose

and time that agents have been given to avoid confusion during subsequent neurologic evaluation. Dilation is contraindicated in the presence of narrow anterior chamber angles. Patients who are predisposed to having narrow angles are usually unaware of this condition. Patients who are using drops such as a beta-blocker, a miotic, or an epinephrine compound are often those who have open-angle glaucoma and are not likely to have an intraocular pressure rise with dilation.

The depth of the anterior chamber can be estimated with a penlight by shining a light in from the side and seeing if the nasal side of the iris lights up. Normally, a uniform illumination of the iris is seen. However, when there is a forward convexity of the iris, only a sector of iris is illuminated with a penlight held tangential to the eye (Fig. 67-1) (Figure Not Available) . With a slit lamp, the depth of the anterior chamber angle can be assessed directly. The definitive test for assessing the anterior chamber angle is gonioscopy, in which the anterior chamber angle structures are viewed directly by means of a special mirrored contact lens and the slit lamp.

Systemic effects can develop after the application of eyedrops.  The reader should review the following sections on agents and complications before using these drugs in patients with compromised cardiovascular function.

## Agents

Only 2 dilators are really needed in the emergency department (ED). Phenylephrine (Neo-Synephrine) 2.5% is used for diagnostic dilation of the pupil for visualization of the fundus. The drug is short acting, and because accommodation is not affected, the patient's vision is not altered. Phenylephrine 10% should not be used routinely because it can seriously elevate the blood pressure in susceptible adults.   For therapeutic cycloplegia in iritis, homatropine 5% works well. Although Table 67-1 indicates a maximum duration of 3 days, 24 hours is a more common duration. Therefore, homatropine 5% is a useful agent for traumatic iritis.

Individuals with lightly pigmented irides tend to have a greater sensitivity to the cycloplegic agents than do individuals with greater pigmentation; the cycloplegic effect may therefore be more prolonged in people with light eyes. Atropine should not be used for traumatic iritis because the undesirable effects of pupillary dilation and blurred vision persist for a week or longer after healing of associated corneal abrasions. Atropine drops may be prescribed as part of the therapy for nontraumatic iritis after appropriate ophthalmologic consultation.

The physician should be aware that malingerers may use mydriatic agents to dilate a pupil unilaterally for the purpose of feigning neurologic disease. Normally, a pupillary dilation caused by intracranial third cranial nerve compression will constrict with 2% pilocarpine eyedrops. The mydriatic-treated eye can be identified by full motor function of the third cranial nerve and the absence of miosis after pilocarpine instillation. It should be noted that legitimate patients may not recall the name of an eye medicine that they used but will usually recall whether the bottle had a red cap, as is found on all cycloplegic solutions. An unexpected mydriasis in a trusted patient may be the result of such an agent. Medications that constrict the pupil, such as pilocarpine, have a green

cap. Pressure-lowering drops for glaucoma may be yellow- or blue-topped (beta-blockers), purple-topped (adrenegic agents) or orange-topped (topical carbonic anhydrase inhibitors).

**Procedure**

The instillation of mydriatic agents is similar to the administration of other eye solutions. For medicolegal purposes, visual acuity should be noted before the instillation of the medicine. This documents that any decreased vision is not the result of the mydriatic agent.

The patient is placed in a supine or a comfortable semirecumbent position. The patient is then instructed to gaze at an object (such as a fixture on the ceiling) in the upper visual field. The physician gently depresses the lower lid using a finger on the epidermis (Fig. 67-2) (Figure Not Available) . A *single* drop of the solution is instilled into the lower lid fornix, and the patient is permitted to blink and to spread the medication. More than a single drop is not recommended, because it produces reflex tearing and reduces the concentration in

| TABLE 67-1 -- Mydriatic Agents | | | |
| --- | --- | --- | --- |
| **Agent** | **Maximum Mydriasis** | **Duration of Mydriasis** § | **Common Trade Name** |
| **Sympathomimetics** * | | | |
| Phenylephrine, 2.5% or 10% | 20 min | 3 hr | NeoSynephrine |
| Cocaine, 5% or 4% | 20 min | 2 hr | -- |
| **Parasympatholytics (Cycloplegics)** | | | |
| Atropine, 1% | 40 min | 12 days | -- |

| | | | |
|---|---|---|---|
| Scopolamine, 0.25% | 30 min | 7 days | -- |
| Homatropine, 5% | 30 min | 1-3 days | -- |
| Cyclopentolate, 1% | 30 min | 6-24 hr | Cyclogyl |
| Tropicamide, 1% | 30 min | 4 hr | Mydriacyl |

§ The duration of effect shows considerable individual variation. These are general estimates.
* Preferred for funduscopic examination.
A 10% solution may produce cardiovascular reaction and hence should not be used.
Preferred for iritis or corneal abrasion therapy.

contact with the conjunctiva. The patient should be forewarned that the medication is uncomfortable and that the closed eye can be blotted but *not* rubbed with a tissue after the medicine is used. If the desired effect is not noted in 15 to 20 minutes, a repeat dose may be used, but this is seldom required.

**Complications**

As mentioned in the section on contraindications, any dilator can precipitate an attack of angle-closure glaucoma in susceptible patients. [6] In a case of angle-closure glaucoma, it may take several hours before symptoms become evident. The patient often complains of smoky vision with "halos" around lights as well as an aching pain that is sometimes severe. There may be nausea and vomiting. The affected eye becomes injected in association with a hazy cornea, elevated pressure on tonometry, and an oval, fixed pupil. Immediate consultation with an ophthalmologist should be obtained. The treatment usually includes osmotic agents, carbonic anhydrase inhibitors, pilocarpine, and, later, definitive laser or surgical procedures (Table 67-2) (Table Not Available) .

The practitioner should be aware that the use of eye medications may introduce infections. Most solutions contain bactericidal ingredients, although contamination of the tips of the droppers can still occur. [7] Only newly opened bottles of eye medication should be used if the practitioner suspects a deep corneal injury or if the patient has recently had eye surgery. Out-of-date drops and drops in which crust or other material is found around the nozzle should be discarded promptly.

Any cycloplegic (in contrast to a sympathomimetic) blurs a patient's near vision. Patients should be forewarned of this effect. Vision will be less blurred in adults older than 45 years of age, who generally have a reduced ability to focus for near vision. Although

most adults will be able to drive safely, even with both eyes affected, it is advisable to have someone else drive whenever feasible. Light sensitivity caused by pupillary dilation may also be bothersome; sunglasses are sufficient for this problem.

Systemic reactions can be produced by sympathomimetic and cycloplegic eyedrops. In one report of 33 cases of adverse reactions associated with 10% phenylephrine, there were 15 myocardial infarctions (11 deaths), 7 cases of precipitation of angle-closure glaucoma, and a variety of systemic cardiovascular or neurologic reactions. [5]

After instillation of eyedrops into the conjunctival sac, systemic absorption can occur through the conjunctival capillaries as well as by way of the nasal mucosa, the oral pharynx, and the gastrointestinal tract after passage through the lacrimal drainage system. Mucosal hyperemia enhances absorption. Symptoms can often be avoided by maintaining digital pressure on the nasal canthus, thus occluding the puncta, for several minutes after administration.

## THE FLUORESCEIN EXAMINATION

Fluorescein staining of the eye should be part of the evaluation of all cases of eye trauma and infection. It is a quick and easy technique that is crucial for the proper diagnosis and management of common eye emergencies. The fluorescein-stained cornea and conjunctiva should be viewed under a "blue" light and ideally in conjunction with slit lamp magnification (see Slit Lamp Examination).

Sodium fluorescein is a water-soluble chemical that fluoresces--it absorbs light in the blue wavelengths and emits the energy in the longer green wavelengths. It fluoresces in an alkaline environment (such as in the Bowman membrane, which is located below the corneal epithelium) but not in an acidic environment (such as in the tear film over an intact corneal epithelium). [19] Thus, it is useful in revealing even minute abrasions on the cornea.

Fluorescein was first used in ophthalmology in the 1880s. [20] It was first used as a drop, but when the danger of contamination by bacteria (especially *Pseudomonas*) was recognized in the 1950s, [21] paper strips were impregnated with fluorescein. These now are supplied in individual sterile wrappers and should be used instead of the premixed solution.

### Indications and Contraindications

Fluorescein staining is indicated for evaluation of all suspected abrasions, foreign bodies, and infections of the eye. [22] This includes "simple" cases of conjunctivitis, which may actually be herpetic keratitis. In actuality, any red eye should be stained.

Fluorescein permanently stains soft contact lenses. Therefore, when fluorescein is used, soft contact lenses should first be removed and the patient cautioned not to put the lenses back into the eye for several hours. Topically administered fluorescein is considered nontoxic, [23] although reactions to a fluorescein-containing solution have

been described. These reports of vagal reactions [24] and generalized convulsions [25] are rare and are believed to be caused by agents other than fluorescein in the solution. The practitioner who chooses to use one of the fluorescein-containing solutions rather than the fluorescein-impregnated strips should be aware of these potential idiosyncratic reactions.

The physician should also be aware that fluorescein dye may enter the anterior chamber of the eye in the presence of deep corneal defects. This form of intraocular fluorescein accumulation is also nontoxic. When the anterior chamber is viewed under the blue filter of the slit lamp, a fluorescein "flare" is visible. This flare reaction should not be confused with the flare reaction noted with iritis.

## Procedure

Ideally one should not use topical anesthetics before fluorescein staining, because some patients will develop a superficial punctate keratitis from the anesthetic, [26] which can confuse the diagnosis. However, with patients who are tearing profusely and who are squeezing their eyes shut from an abrasion or a foreign body, the examination often is impossible if a topical anesthetic is not first used.

The fluorescein strip is grasped by the non-orange end, and the orange end is wetted with 1 drop of saline (most conveniently available as a small bottle of artificial tears) or dextrose solution. The wetted strip is then placed gently onto the inside of the patient's lower lid. The strip is withdrawn, and the patient is instructed to blink. The patient's blinking spreads the fluorescein over the eye. The key to a good examination is a *thin* layer of fluorescein over the corneal and conjunctival surfaces. If the strip is heavily wetted before application in the lower fornix, the eye may become flooded with the solution, thus making evaluation difficult. On the other hand, use of a dry strip in the unanesthetized eye may be irritating. If too much dye accumulates, the patient can remove the excess by blotting the closed eye with a tissue. The physician uses a Wood lamp, the blue filter of a slit lamp, or simply a penlight with a blue filter to examine the eye in a darkened room, checking for areas of bright green fluorescence on the corneal and conjunctival surfaces. Small defects may not be seen with the naked eye; ideally, a slit lamp should be used to examine the stained cornea before ruling out a pathologic process. After completion of the fluorescein examination, excess dye should be irrigated from the eye to minimize damage to the patient's clothing from dye-stained tears.

A special use for fluorescein is in the *Seidel* test [27] for detection of perforation of the eye. To perform this test, the physician instills a large amount of fluorescein onto the eye by profusely wetting the strip. The eye is then examined for a small stream of fluid leaking from the globe. This stream will fluoresce blue or green in contrast to the orange appearance of the rest of the globe flooded with fluorescein.

## Interpretation

Fluorescein is mainly used for evaluation of corneal injuries. Although conjunctival abrasions pick up the stain, most of the staining on the conjunctiva represents patches of mucus rather than a real pathologic condition. Corneal staining is more specific for

injury, and the pattern of injury often reflects the original insult.

The corneal staining shows patterns as illustrated in [Figure 67-3](). Abrasions usually occur in the central cornea because of the limited protection of the patient's closing eyelids. The margins of the abrasions are usually sharp and linear if seen in the first 24 hours. Circular defects are seen about embedded foreign bodies and may persist for up to 48 hours after removal of a superficial foreign object. Deeply embedded objects may be associated with defects persisting for longer than 48 hours. Objects under the upper lid (including some chalazions) often produce *vertical linear lesions* on the upper surface of the cornea. When vertical lesions are noted, a *diligent search for a retained foreign body under the upper lid should be made*. Hard contact lens overuse diminishes the nutrient supply to the cornea. The central cornea receives the most injury and thus fluoresces brightly when stained. Ultraviolet light exposure from sunlamp abuse, snow blindness, or welding flash produces a superficial punctate keratitis, which in its mildest form may not be visible without a slit lamp. The central cornea is the least protected by the lids, and a central horizontal band-like keratitis can result. Herpetic lesions may develop anywhere on the cornea. Classically, these lesions are dendritic, although ulcers may also be punctate or stellate.

Any area of corneal staining with an infiltrate or opacification beneath or around the lesion should alert the practitioner to the possibility of a viral, [29] bacterial, [31] or fungal [32] *keratitis*. Urgent ophthalmologic consultation should be obtained so that cultures of the possible etiologic agents can be procured and treatment initiated.

Frequently, practitioners are unaware that many *Pseudomonas* organisms fluoresce when exposed to ultraviolet light. [33] The presence of fluorescence before the instillation of fluorescein in the red eye should suggest the possibility of a pseudomonal infection.

**Summary**

Fluorescein staining is a quick, easy diagnostic procedure that should be part of every eye evaluation. The extra minute that the examination takes provides a wealth of diagnostic information for patients with eye trauma or infection. With the exception of the reactions noted with fluorescein solution, the potential discoloration of soft contact lenses, and the potential for infection when the solutions rather than fluorescein-impregnated paper strips are used, no complications are associated with the procedure.

# EYE IRRIGATION

Eye irrigation is the crucial first step in the treatment of chemical injuries to the eye. Irrigation should be clinically appropriate to the exposure and severity of the injury. Serious chemical injury to the eye requires irrigation at the site of injury, *before* the patient is brought to the ED. [34] Corneal injury can occur within seconds of contact with an alkaline substance. Eye irrigation must often be continued in the ED.

This section discusses methods of irrigation. Although it is best to irrigate liberally,

copious irrigation is not needed when the patient has gotten a small amount of a noncaustic, nonalkaline compound in the eye.

## Indications and Contraindications

Irrigation is indicated for all acute chemical injuries to the eyes. Irrigation may also be therapeutic for patients having a foreign body sensation with no visible foreign body. Small, unseen foreign material in the conjunctival tissues may be flushed out with irrigation. There is no contraindication to eye irrigation, but if there is also a possible perforating injury to the eye, the irrigation must be performed especially gently and carefully.

## Equipment

The following equipment is necessary for eye irrigation:

Topical anesthetic, such as proparacaine 0.5%

Sterile irrigating solution (usually, intravenous saline in a bag with tubing is the easiest to obtain)

A basin to catch the fluid

Cotton-tipped applicators

Gauze pads to help hold the patient's lids open

Lid retractors

Irrigating device (e.g., Morgan Therapeutic Lens or modified central venous catheter) for prolonged irrigation

## Procedure

### Basic Technique

Topical anesthetic is instilled. Any particulate matter should be swept out of the conjunctival fornices with moistened cotton-tipped applicators. [34] This requires eversion of the upper lid (see the section on foreign body removal and Fig. 67-4) (Figure Not Available) . During actual irrigation, the lids must be held open. It is easiest to use the gauze pads to get a grip on the wet, slippery lids. At times the patient has blepharospasm to such a degree that lid retractors (Desmarres or paper clip retractors; Fig. 67-5) (Figure Not Available) may be necessary (Fig. 67-6) (Figure Not Available) . When lid retractors are used, the practitioner must be certain that the eye is well anesthetized, that the retractors do not injure the globe or the lids, and that chemicals are not harbored under the retractors. Retractors fashioned from metal paper clips (especially those that are nickel-plated and shiny) may have surface chipping which can create an ocular foreign body. [34A] Caution is needed to avert ocular injury when using

such a makeshift retractor.

Deutsch and Feller recommend an ipsilateral facial nerve block for severe blepharospasm (Fig. 67-7) (Figure Not Available) . [34] To avoid swelling of the periorbital tissue, the facial nerve is blocked just anterior to the condyloid process of the ipsilateral mandible. A line of anesthesia (2% lidocaine) is placed subcutaneously to temporarily paralyze the orbicularis muscle.

The saline exiting from the intravenous tubing is directed over the globe and into the upper and lower fornices. The choice of fluid is less important than the rapidity of irrigation initiation. Tap water should be readily available at the scene of the injury, and copious immediate irrigation should be encouraged before patient transport to the hospital. Out-of-hospital care providers should be taught to irrigate all acid injuries of the eye for at least 5 minutes at the scene and to irrigate all alkali injuries for at least 15 minutes. [35] [36] Normal saline solution is preferred for eye irrigation, because it is isotonic without dextrose. Dextrose can be quite sticky if spilled and may serve as a nutrient for an opportunistic bacterial infection. Balanced saline solution is tolerated better than normal saline, lactated Ringer's solution, or normal saline buffered with sodium bicarbonate. [37] It should be considered when normal saline is poorly tolerated during eye irrigation.

Care should be taken to direct the irrigating stream onto the conjunctiva and then across the cornea without letting the stream splash directly onto the cornea, because the mechanical injury of the solution striking the eye can of itself be harmful. Direct irrigation of the cornea can result in the development of a superficial punctate epithelial keratopathy.

**Duration of Irrigation**

Although Deutsch and Feller recommend that a full liter of irrigating solution be used in every case of caustic injury, [34] the duration of the irrigation is best determined by the extent of exposure and the causative agent. Acids are quickly neutralized by the proteins of the eye surface tissues and, once irrigated out, cause no further damage. The only exceptions are hydrofluoric and heavy metal acids, which can penetrate through the cornea. Alkalis can penetrate rapidly and if not removed (because of the slow dissociation of the cation from combination with proteins) will continue to produce damage for days. [34] Therefore, prolonged irrigation is indicated; at least 2 L of solution should be used over 20 minutes for any significant alkaline injury. [38] If the nature of the offending agent is in doubt, prolonged irrigation should be used.

Ophthalmologic consultation should be obtained for all alkaline, hydrofluoric acid, and heavy metal acid injuries. Irrigation on an inpatient basis may be required for a period of 24 hours or more. This is especially likely when the cornea is hazy or obviously thickened. It should be noted that the magnesium contained in sparklers combines with water from tears to produce magnesium hydroxide. [39] Such fireworks accidents should be treated as alkaline injuries rather than thermal injuries. Eye damage from hair straighteners, [40] phosphate-free detergents, [41] and automobile airbags [42] must also be

treated as alkaline injuries.

A good method of checking the effectiveness of irrigation is to measure the pH of the conjunctival fornices with a pH paper strip. The pH indicator on urine multi-indicator sticks can also be used. The pH indicator on urine dipsticks is conveniently closest to the handle; all the distal indicator squares can be cut off with scissors. The normal tear film pH is 7.4. If the pH measured in the conjunctival fornices after the initial irrigation is still abnormal, irrigation is to be continued. If the pH is normal after irrigation, one should check it again in 20 minutes to make sure that it remains normal.

Delayed pH changes are usually the result of incomplete irrigation and inadequate swabbing of the fornices. In anticipation of this deficiency, one should measure the pH deep in the fornices. Often, double-lid eversion with a lid elevator is required to expose the upper fornix for swabbing, irrigation, and pH testing (see Fig. 67-4) (Figure Not Available) .

**Prolonged Irrigation**

Prolonged irrigation may be required with alkali burns. Ophthalmologic consultation is essential in such situations. One technique for prolonged irrigation uses a contact lens-type irrigation device (e.g., Morgan therapeutic lens), which allows for continuous irrigation once the more vigorous irrigation described earlier has been used. During use, the device is set on the anesthetized eye, and the lids are allowed to close around the intravenous tubing adaptor (Fig. 67-8) (Figure Not Available) . Continuous flow through the device onto the cornea and into the fornices occurs. This contact lens device can become uncomfortable, because local anesthetic agents are washed out during the irrigation process; the anesthetic agent must be reapplied frequently during irrigation for patient comfort. The repeated use of the anesthetic agent may itself inhibit corneal healing.

Another irrigation device can be made for the prolonged irrigation of the fornices by modification of a small-caliber central venous catheter. Multiple perforations are made with a scissors or a scalpel in the distal portion of the catheter. This perforated section is placed in the inferior fornix of the anesthetized eye beginning at the lateral canthus and is then looped back in the upper lid fornix (Fig. 67-9) . The patient is instructed to tilt the head toward the side of the tubing to permit drainage into a laterally placed basin. After loosely applying an eye patch, one attaches the catheter to an intravenous setup for continuous irrigation. An anesthetic agent is also needed with this technique, albeit less frequently than with the contact lens-type device.

Lippas described the successful treatment of 2 alkaline burns using a similar but more invasive continuous irrigation technique for prolonged irrigation after immediate copious irrigation. [43] Continuous irrigation of an alkaline injury with a 10 or 20% acetylcysteine (Mucomyst) solution to inhibit collagenase [44] and topical (1%) calcium gluconate eyedrops [45] have been recommended, although one should await ophthalmologic consultation before using these methods.

**Complications**

The only complication from irrigation is abrasion of the cornea or the conjunctiva. This can be a mechanical injury from trying to keep the lids open in an uncooperative patient, a small corneal defect from a Morgan irrigating lens, or a fine punctate keratitis from the irrigation itself. [46] It is for this reason that the stream should not be directed onto the cornea. If a superficial corneal defect occurs, it is treated in the usual manner by topical antibiotics and consideration of patching. Deep or penetrating corneal injuries are likely to be the result of the caustic chemical and require emergency ophthalmologic consultation. In general, the emergency physician should not patch these deeper injuries. They should receive continuous irrigation pending ophthalmologic consultation. There is some experimental evidence that massive parenteral or oral ascorbic acid supplementation may prevent the development of deep corneal injury, [47] although this treatment has not gained universal acceptance.

**Summary**

Eye irrigation is easy, and complications associated with the technique are minimal. At times, the physician may be unsure whether a chemical injury is toxic enough to warrant irrigation. One should irrigate the eye if any doubt exists rather than omit this vital procedure and permit the progression of eye injury.

# OCULAR FOREIGN BODY REMOVAL

Patients with an external foreign body in the eye are frequently seen in EDs. They are often in pain and desperate for help. A high degree of suspicion for foreign body injuries and perforations of the eye is necessary, because such injuries may be occult and not readily detected. Not all foreign body injuries are associated with pain; glass embedded in the cornea may be particularly difficult to detect. This section is a review of the procedures for locating and removing extraocular foreign bodies. A brief discussion covering evaluation for perforation of the globe and for an intraocular foreign body is provided. Care of the patient after removal of an extraocular foreign body is presented.

**Indications and Contraindications**

Removal of extraocular foreign bodies is always indicated. The timing of removal and the technique that is required vary according to the patient's clinical status and the type of injury. For the most part, the emergency physician can proceed directly to removal of the object using the techniques described in this section. When the patient is extremely uncooperative (e.g., a mentally deficient individual or a young child) or when the injury is complicated (e.g., deeply embedded object, multiple foreign objects from a blast injury, or possible globe penetration), immediate ophthalmologic consultation is indicated. A penetrating injury to the cornea is particularly troublesome in that iris tissue may prolapse and may appear to represent a corneal foreign body (Fig. 67-10) (Figure Not Available) . Hence, an irregular pupil, especially a pear-shaped pupil, should alert the

clinician that a penetrating injury may have occurred.

**Equipment**

The following equipment is necessary for extraocular foreign body removal:

> Topical anesthetic, such as proparacaine 0.5%
>
> Sterile cotton-tipped applicators
>
> Fluorescein strips
>
> Magnification: loupes plus a Wood lamp or a slit lamp
>
> Eye spud or 25-ga needle attached to a 1- or 3-mL syringe or to the tip of a cotton-tipped applicator
>
> Dilator drops, such as homatropine 5% or cyclopentolate 1%
>
> Antibiotic ointment, such as erythromycin
>
> Eye patches
>
> Tape (nonallergenic paper tape is preferable)

**Consideration of Intraocular Foreign Body**

The emergency physician should always remain cognizant of the potential for an *intraorbital* or *intraocular* foreign body when examining the patient with a "foreign body" sensation. Penetrating injuries represent a greater threat of visual loss than extraocular foreign bodies and can be disastrous if overlooked.

The clinical presentation is helpful in the determination of which patients are at risk for a penetrating injury to the globe. An individual who complains of a foreign body sensation in the *absence* of trauma or one whose history is simply that something "fell" or "blew" into the eye is at *low* risk for a globe perforation. On the other hand, there is a greater probability of globe penetration in the individual who has sustained a high-velocity wound to the eye (e.g., drilling or grinding metal, blasting rock). The presence of any of the following physical findings should alert the physician to a probable *intra*ocular foreign body: irregular pupil, shallow anterior chamber on slit lamp examination, prolapsed iris, positive Seidel test (see Fluorescein Examination), focal conjunctival swelling and hemorrhage, hyphema, lens opacification, and reduced intraocular pressure. (Tonometry should not be performed in the presence of other physical findings suggesting penetration of the globe.) One should be aware that a penetrating injury may *not* be associated with eye pain. Strong historical evidence and physical findings supporting a diagnosis of penetration of the globe should prompt emergent ophthalmologic consultation.

Often, *intra*ocular foreign bodies are not visible on direct ophthalmoscopy. Although orbital radiography for radiopaque objects and ultrasonography of the globe have been used for indirect foreign body localization, [48] computed tomography of the orbit is now considered the most useful technique. [49] [50] Patients with a suspected metallic foreign body should *not* undergo magnetic resonance imaging if the foreign body may be *intra*ocular. Therapy for intraocular and intraorbital foreign bodies must be individualized. Often, an ophthalmologist can localize an intraocular foreign body (if the vitreous is clear) using indirect ophthalmoscopy. The role of the emergency physician is to suspect the diagnosis, to protect the eye from further harm, and to obtain ophthalmologic consultation. The remainder of this section addresses the problem of extraocular foreign bodies.

## Procedure

### Foreign Body Location

The first step is to locate the foreign body. A drop of both topical anesthetic and fluorescein is applied to the inside of the lower lid (see Fluorescein Examination). Vertical corneal abrasions from foreign bodies under the lids are helpful for localizing these hidden foreign objects (see Fig. 67-3 *C*). One should use a penlight and loupes or a slit lamp to examine the bulbar conjunctiva by having the patient look in all directions. The physician examines the inside of the lower lid by pulling it down with the thumb while the patient looks up. One everts the upper lid by having the patient look down as the end of an applicator stick is pressed against the superior edge of the tarsal plate of the upper lid. Meanwhile, the physician grasps the lashes and pulls down and then up to flip the lid over (Fig. 67-11) (Figure Not Available) .

Minute foreign bodies under the lid will be missed with simple visual inspection. Ideally, the everted lid should be examined under magnification with loupes or a slit lamp. With simple lid eversion it is still not possible to see the far recesses of the upper conjunctival fornix. Although double eversion of the upper lid (see Fig. 67-4) (Figure Not Available) is helpful, the best way to rule out a foreign body in the upper fornix is to sweep the anesthetized fornix with a wetted applicator as the upper lid is held everted. The applicator tip should be examined for removed foreign material. Small conjunctival foreign bodies not hidden by the lids are often best removed with a wetted nasopharyngeal swab (e.g., nasopharyngeal Calgiswab).

The cornea is then reexamined. Most corneal foreign bodies have an area of fluorescein staining around them. A slit lamp makes the examination easy. If the physician is limited to loupes and a penlight, the light is shined diagonally on the cornea. One then finds a foreign body directly or indirectly by noting a shadow on the cornea or the iris (Fig. 67-12) (Figure Not Available) .

With a history of a high-speed projectile hitting the eye, one must rule out an intraocular foreign body. Except in the case of a blast injury, if one foreign body is found on the surface of the globe, it is highly unlikely that there is a second foreign body inside the eye. If a foreign body cannot be found on the surface despite a suggestive history, the

eye should be examined for physical evidence of penetration as discussed earlier. The pupil is dilated, and the fundus is examined. If in doubt regarding an intraocular foreign body, one should consider computed tomography and ophthalmologic consultation.

**Foreign Body Removal**

Once an extraocular foreign body is located, the technique of removal depends on whether it is embedded. If the foreign body is lying on the surface, a stream of water ejected from a syringe through a plastic catheter usually washes the object onto the bulbar conjunctiva. Once the foreign body is on the inner lid or bulbar conjunctiva, a wetted cotton-tipped applicator can be gently touched to the conjunctiva and the object will adhere to the applicator tip. Overzealous use of an applicator for corneal foreign body removal can lead to extensive corneal epithelial injury. A spud device is required for removal of objects that cannot be irrigated off the cornea.

Embedded corneal foreign bodies are best removed with a commercial spud device or a short 25- or 27-ga needle on a small-diameter syringe (e.g., insulin or "TB" syringe) or a cotton-tipped applicator (Fig. 67-13 (Figure Not Available) *A* and *B*). The applicator or the syringe serves as a handle for the attached needle. Contrary to what one might expect, it is difficult to penetrate the sclera or the cornea with a needle. [51] As with removal of conjunctival foreign bodies, the eye must be well anesthetized. The patient should be positioned such that the head is well secured (preferably in a slit lamp frame). When removing a foreign body, it is useful to have an assistant present to help the patient keep the head properly positioned and the eye opened. The patient must be instructed to gaze at an object in the distance (e.g., the practitioner's ear when a slit lamp is used) to stabilize the eye further. The needle or spud device is brought close to the eye *under direct vision*, then while it is in focus, the device is manipulated under the slit lamp to remove the foreign body. The device is held *tangentially* to the globe, and the foreign object is picked or scooped out (Fig. 67-13 (Figure Not Available) *C*).

During removal, the physician should brace his or her hand against the patient's face. It also may be helpful to brace the elbow with a pad or half-full tissue box to provide further support to the arm as the foreign body is removed. The right-handed physician should place the lower hand against the left maxillary bone when removing a foreign object from the patient's left eye and against the bridge of the patient's nose or infranasal area when removing an object from the right eye. These positions should be reversed by the left-handed physician. The use of loupes or a slit lamp for magnification is highly recommended to minimize further injury during removal. In particular, corneal contact with the spud device is more readily discerned when magnification is used. Only topical anesthesia is required to remove foreign bodies from the cornea. Although the patient may feel pressure during foreign body removal, pain should not be felt after the eye is anesthetized.

**Rust Rings**

A common problem with metallic foreign bodies is rust rings. These can develop within hours because of oxidation of the iron in the foreign body. There are 2 preferred techniques for removal of a rust ring. The most direct method is to remove it at the

same time as the foreign body, either with repeated picking away with a spud device or with a rotating burr. The second approach is to let the iron of the rust ring poison and kill the surrounding epithelial cells during a 24- to 48-hour patching period. At that time, the rust ring will be soft and often comes out in one solid plug. [52] After rust ring removal, the eye must be patched again for another 24 hours to allow the residual corneal defect time to heal. Generally, a small rust ring produces little visual difficulty unless it is directly in the line of sight. The rust ring, if large, may delay corneal healing.

### Multiple Foreign Bodies

The patient with multiple foreign bodies in the eye, such as from an explosion, should be referred to an ophthalmologist. A technique that may be chosen by the ophthalmologist is to denude the entire epithelium with alcohol and remove the superficial foreign bodies. The deeper ones gradually work their way to the surface, sometimes years later.

## Aftercare

After removing the foreign body, one should patch the eye if there is any large corneal abrasion (see the section on patching). An antibiotic ointment is frequently instilled before patching, but the value of the ointment for very *superficial* corneal defects after foreign body removal is unknown. *Conjunctival* abrasions do not need patching. If the eye is patched, the patient should be reexamined in 24 hours. An antibiotic ointment is applied before placement of the patch. If the patient sustains a *superficial* injury from the foreign body and an eye patch is applied primarily for comfort, the physician may wish to instruct the patient to return only if the eye does *not* feel completely normal *or* if there is any blurred vision. The majority of superficial injuries heal without difficulty. The patient should be warned that the foreign body sensation may return temporarily before patch removal when the anesthetic agent has worn off. One animal study with direct ocular exposure to *Clostridium tetani* organisms suggests that nonpenetrating ocular injuries are unlikely to lead to tetanus. [53] However, tetanus prophylaxis appears essential for injuries that penetrate through the cornea or sclera.

## Use of Ophthalmic Anesthetic Agents

Application of topical anesthetic agents can be both diagnostic and therapeutic. Relief of discomfort with topical anesthetic use suggests, but does not ensure, a conjunctival or corneal injury. An ocular irritant may also be masked by the use of these agents. Classic teaching is that the anesthetic preparations should not be self-administered by patients. The absence of protective reflexes while the patient is under the effect of the medicine may encourage the patient to use the eye while a foreign body or a corneal infection inflicts further corneal injury.

Bartfield and coworkers found that the pain of instillation of 0.5% proparacaine was significantly less than that of 0.5% tetracaine. [54] As evident from Table 67-3 , the anesthetic solutions that are commonly used have a duration of action <20 minutes. The patient requiring patching may need a more extended period of pain relief. The discomfort associated with a large healing corneal lesion is usually made tolerable by a pressure patch, bedrest, opioid analgesics, and sedatives (e.g., secobarbital or

triazolam). Even in the absence of infection or a retained foreign body, the *repeated* use of ophthalmic anesthetic ointments may be detrimental to corneal healing. [55] Also, frequent removal of a pressure patch to anesthetize the cornea may lead to disruption of migrating corneal epithelial cells and may thus prolong corneal repair.

A final word of caution should be added regarding the use of ophthalmic solutions. Guaiac solutions are commonly supplied in dropper bottles similar in size and appearance to those containing ophthalmic solutions. Well-intentioned ED personnel may store the guaiac reagent bottles with the ophthalmic bottles. One should encourage both color coding of the bottles and examination of the bottle and its label before each use to avoid corneal injury from the guaiac reagent.

### Complications

Complications associated with ocular foreign body removal are rare. The most frequent problem is incomplete removal of the foreign body. In such cases the epithelium has difficulty healing over the affected area, and thus the eye stays inflamed. Eventually, the diseased epithelium either sloughs off and heals or heals over the foreign body remnants, which are gradually absorbed. In either case, the adverse effects on the eye are minimal; a minute scar on the cornea, even directly in the center, will rarely affect the vision. Nonetheless, incomplete removal of a corneal foreign object warrants ophthalmologic follow-up.

Conjunctivitis may develop after removal of an extraocular foreign body. In most cases, the bacteria producing the infection are introduced by the patient through rubbing of the irritated eye.

Although perforation of the globe by the physician's spud device is theoretically possible, this complication is exceedingly rare. Treatment of such a minor corneal puncture wound consists of antibiotics, eye shield placement, and ophthalmologic consultation. In the absence of resultant endophthalmitis, permanent sequelae are unlikely to develop.

Much epithelial injury can occur when cotton-tipped applicators are vigorously used to remove corneal foreign bodies. Indeed, the use of cotton-tipped applicators for *corneal* foreign body removal is condemned.

### Summary

Ocular foreign bodies are one of the most common eye emergencies. Searching for and removing the foreign body is usually straightforward. The only real trap is missing an intraocular foreign body. This must be ruled out if there is a history of a high-speed projectile hitting the eye or if physical findings suggestive of globe penetration are present.

## EYE PATCHING

Patching the lids shut has traditionally been the last step in treatment of a number of common eye emergencies. Many physicians, however, have only a vague idea of what the purpose of patching is, how to do it, and how to follow-up. Even this simple procedure can be performed incorrectly.

Patching may be used for several goals. A simple patch may be used to protect the dilated eye from bright light. Pressure patching as discussed in this section is the use of a patch to hold the eyelid closed to facilitate healing of a corneal defect, by limiting eyelid movement over an injured area.

It has been suggested in some studies that small, simple corneal abrasions do not require patching to aid healing or for reduction of pain. Unfortunately, these small studies have a number of limitations, including minimal description of the patching technique and patient compliance. In one study, 29% of eye pads "fell off during sleep." [57] However, collectively these studies support the treatment of small corneal defects (i.e., <2 mm width) without a pressure patch.

### Indications and Contraindications

Patching is indicated whenever the surface of the cornea has been injured. This can occur after a mechanical abrasion,

| **TABLE 67-3** -- Ophthalmic Anesthetic Agents | | | |
|---|---|---|---|
| Generic name | Tetracaine | Proparacaine | Benoxinate |
| Trade name | Pontocaine | Ophthaine, Ophthetic | Dorsacaine |
| Concentration | 0.5 to 1.0% | 0.5% | 0.4% |
| Onset of anesthesia | Less than 1 min | Less than 20 sec | 1-2 min |
| Duration of anesthesia | 15-20 min | 10-15 min | 10-15 min |

| | | | |
|---|---|---|---|
| Comments | Marked stinging; also available in ointment | Least irritating; no cross-sensitization with other agents | Only anesthetic compatible with fluorescein in solution |

such as a fingernail scratch, or after the removal of a foreign body. Chemical damage, damage from prolonged *hard* contact lens use, and ultraviolet light injuries, which are commonly seen in the ED, also may benefit from patching. With each of these forms of injury, the purpose of the patch is to keep the lids from moving over the cornea and to keep light out. After patching, the patient should immediately experience less pain and tearing.

Patching is contraindicated when the corneal epithelial loss results from an active infection--such as a corneal ulcer--rather than from an abrasion. The first consideration in the differentiation between ulcer and abrasion is the history of the injury (how recently it occurred and how clean the offending object was). The second determining factor is the appearance of the cornea (i.e., an ulcer will have an infiltrate of white cells beneath the area of epithelial loss and will be accompanied by a purulent discharge). Generally, abrasions associated with *soft* contact lens use should be considered infected and patching avoided until ophthalmologic evaluation. [59] Of particular concern is the rapid development of *Pseudomonas* keratitis, an unusual, but serious infection associated with corneal abrasions from soft contact lenses. A pressure patch should never be applied to an eye with a penetrating injury. A protective cup is the preferred covering in such cases pending ophthalmologic consultation.

**Equipment**

The following equipment is necessary for patching the eye:

> Gauze eye patches (2)
>
> Tape (e.g., 1-in. paper tape, preferably nonallergenic)
>
> Antibiotic ointment (e.g., sulfacetamide 10%)
>
> Dilator eyedrops (cyclopentolate [Cyclogyl] 1% or homatropine 5%)

The tape should be precut into 4-in. strips and should be kept within reach. Commercial patching products are also available and are discussed later.

**Procedure**

Before patching a corneal abrasion, one should apply both a dilator drop and an antibiotic ointment. The dilator must be a cycloplegic to relax the ciliary muscle spasm that accompanies corneal abrasions. Both cyclopentolate 1% and homatropine 5% last approximately 24 hours. The patient should be checked for a narrow anterior chamber

before the drop is instilled (see Fig. 67-1) (Figure Not Available) . Antibiotic ointment is used prophylactically, although it is rare for abrasions to become infected. [60] In the past it was thought that the ointment vehicle would slow epithelial healing, but the vehicles that are currently used do not have this effect.

An effective patch must be put on tightly enough to keep the lids shut. The physician should have the patient shut both eyes and should remind him or her to keep them shut throughout the entire procedure. Two patches are used. The vertically positioned first patch is doubly folded and placed over the closed lids. The unfolded second patch is then put horizontally over the first (Fig. 67-14) . The tape strips are stretched diagonally from the center of the forehead to the cheekbone. The physician can pull up the skin of the cheek; when he or she lets go, the tape will be even tighter. If the tape completely covers the patch, slippage of the patch and resultant eye movement are avoided (Fig. 67-15) (Figure Not Available) . The tape should not extend onto the angle of the mandible, because mastication loosens the tape in such a situation. Some physicians paint the skin about the eye with tincture of benzoin to help secure the tape. Care must be taken not to introduce the benzoin into the eye. The presence of extensive facial hairs may prevent tight taping of the patch. An adjustable elastic strap pressure patch (e.g., Presspatch II, Precision Therapeutics, Inc, Las Vegas) may be useful in this setting (Fig. 67-16) (Figure Not Available) . Anecdotally, the patient must be carefully instructed in the use of this commercial patch and avoidance of excessive globe pressure from the elastic straps must be confirmed.

It is imprudent to leave a pressure patch on for >24 hours. When repatching to promote healing is required, a clean patch should be applied within each 24 hours. Repatching prevents a patch from becoming moist and serving as a nidus for the development of infection.

The Donaldson Eyepatch (Keeler Instruments, Inc, Broomall, Pa) is a commercial product used for enforced eyelid closure that avoids the bulkiness of pressure patching (Fig. 67-17) (Figure Not Available) . The device has 2 components: one adheres to the upper lid, and the other is a circle that adheres to the ipsilateral cheek. A tab on the upper component connects by Velcro to the lower component. This patch may be advantageous in some settings, but its design may encourage some individuals to release the lid closure prematurely.

Because of conjugate eye movement, patching only one eye does not totally immobilize the globe. When all movement must be stopped, bilateral eye patches should be applied. The use of bilateral patches may not be accepted by all patients, and one must seek both patient understanding and family support for patient home activities if dual eye patching is to be successful.

## Complications

There are few complications involved in patching. It is possible to patch the patient's lashes in between the lids so that they abrade the cornea. This can occur if the patient partially opens the eye during the procedure. This can be avoided by insisting that both

eyes stay closed during the entire patching.

Most problems develop when the eye is not securely patched and excessive lid motion occurs. In this situation the corneal epithelial cells are not permitted to migrate over and close the epithelial defect. This leads to increased pain and delayed healing. Some corneal defects are extensive and may require 3 to 5 days for healing. Patients with extensive injuries should be observed frequently and treated with cycloplegic agents, pain medication, and (if clinically indicated) sedatives for sleep. The practitioner should document the size of the corneal defect at each visit. If healing does not occur in a progressive fashion, ophthalmologic consultation should be obtained. As mentioned earlier, patching is contraindicated when a corneal ulcer is present. A corneal "abrasion" that does not heal could very well be a herpetic ulcer.

All patients whose eyes have been patched for a corneal abrasion should be warned about possible *recurrent corneal erosions* that might occur in the future as a complication of the original injury. The original abrasion may appear to have healed perfectly, but days, weeks, or even months later a small area of corneal epithelium can come off, re-creating the symptoms of the original abrasion in the absence of new trauma. This usually occurs in the mornings as the patient opens the eyes, presumably due to adherence of the weaker, recently healed corneal epithelium to the upper lid during sleep. These erosions may heal before the patient is reexamined and can be very puzzling. The cause is a failure of bonding of the corneal epithelium to its basement membrane. [63] Patients who develop this syndrome are given 5% sodium chloride ointment to use nightly to prevent the erosions; some require bandage soft contact lenses. [64]

**Summary**

Patching should be an easy, straightforward procedure. A common problem is the lack of follow-up instructions given to the patient after the patching. Too often, the patient is given a bottle of antibiotic drops to be used every 4 hours and only vague recommendations for a follow-up check. When patients remove the patch to put in an antibiotic drop, they are never able to replace the patch properly again. Instead, they should be told to keep the patch on for 24 hours and to return to have their abrasion checked by a physician after that time. This is the only safe way of ensuring that the abrasion has healed properly. As discussed earlier, trustworthy patients with very *superficial* corneal injuries may be given more responsibility (and instruction) for their own subsequent care, and many such cases do not require a patch at all. The physician should, however, make a follow-up telephone call to these individuals if they choose not to return to ascertain their compliance with the instructions.

The patient with an eye patch should be instructed to rest the uninjured eye. Reading should be discouraged, because involuntary movement of the patched eye will result. Watching television from a distance of 10 feet or more promotes eye fixation and is acceptable. Distant vision is unaffected by patching, although a small degree of peripheral vision is lost on the patched side. Although the visual field is minimally affected, driving after a patch is placed is not advisable. Preferably, the patient should be driven home from the hospital to minimize the potential for patient injury. An elderly

patient may require assistance with routine ambulation after eye patching. Use of collagen shields applied directly to the cornea may offer an attractive alternative to eye patches. [65] These devices have yet to be introduced into ED practice.

# CONTACT LENS PROCEDURES

An estimated 24 million Americans wear a form of contact lenses. [66] Removal of these lenses in the ED may be required to permit further evaluation of the eye or to prevent injury from prolonged wear. Emergency physicians also evaluate patients for "lost" contact lenses, which may be trapped under the upper lid. At times, the patient may request that the physician remove a lens that he or she has failed to extract from the cornea. Corneal ulcers may occur in patients who wear contact lenses and may require prompt treatment. This section on contact lens procedures addresses these concerns and discusses injuries associated with removal attempts, the mechanism of injury from prolonged wear, and instructions to be given to patients at discharge.

The first contact lenses were scleral lenses made of glass. These lenses, covering the cornea as well as much of the surrounding sclera, are reported to have been in use from 1888 to 1948. [67] Glass corneal lenses (sitting entirely on the cornea) made by the Carl Zeiss Optical Works of Jena were first described in 1912. A practical synthetic scleral lens using methyl methacrylate rather than glass was discussed by Obrig and Mullen in 1938. [68] [69] In 1947, Tuohy redeveloped the corneal lens using methyl methacrylate. This was the forerunner of the current hard contact lens. [70] The development in Czechoslovakia of lenses made of soft gas-permeable polymers was reported in 1960. [71] These hydrogel (hydrophilic gelatinous-like) lenses have evolved into today's soft contact lenses. Soft contact lenses now come in a variety of types including extended and daily wear. Some extended-wear lenses are disposable. All lenses should be removed at least once a week.

## Mechanism of Corneal Injury from Contact Lens Wear

### Hard Contact Lenses

The oxygenation of the cornea is dependent on movement of oxygen-rich tears under the hard contact lens during blinking. During the "adaptation" phase of early wear, the wearer of hard contact lenses produces hypotonic tears as a result of mechanical irritation from the lens. [72] This results in corneal edema, which reduces subsequent tear flow under the lens during blinking. Overwearing a lens at this time leads to corneal ischemia, with superficial epithelial defects predominantly in the central corneal area (see Fig. 67-3 *D*), where the least tear flow occurs. With adaptation, the tears become isotonic and the blinking rate normalizes, permitting increased wear time. During early adaptation blinking is more rapid than normal and then slows to a subnormal rate during late adaptation. Mucus delivery to the cornea in the tear film may also play an important role in maintaining corneal lubrication. Tight-fitting contact lenses may never permit good tear flow despite an adaptation phase; individuals with tightly fitted lenses may never be able to wear their original contact lenses for longer than 6 to 8 hours. Lenses that are excessively loose can also cause irritation by moving during blinking. Rough or

cracked edges can cause corneal abrasions.

In the ED, the patient who presents with irritation caused by prolonged wear may be either a new or an adapted wearer. The adapted wearer may have been exposed to chemical irritants (e.g., smoke), which reduce the tonicity of tears and lead to corneal edema and decreased tear flow. Alternatively, the adapted wearer with irritation may have ingested sedatives (e.g., alcohol) or may have fallen asleep wearing the contact lenses, thus decreasing blinking and tear flow. Another possibility is that the patient may actually be wearing tight-fitting contact lenses that have never allowed true adaptation despite many months of wear.

The patient with the overwear syndrome usually awakens a few hours after removing the lenses. The patient experiences intense pain and tearing similar to that caused by a foreign body. The delay in the onset of symptoms until after removal of the lenses is caused by a temporary corneal anesthesia produced by the anoxic metabolic by-products that build up during extended lens wear. [73] A second factor is the slow passage of microcysts of edema, which are pushed up to the corneal surface by mitosis of the underlying cells. When the cysts break open on the surface, the corneal nerve endings are exposed. [74]

Most patients with the overwear syndrome can be managed with reassurance, frequent administration of artificial tears, oral analgesics, and advice to "wait it out" in a darkened room. Some patients require patching for comfort. A patient who has experienced no problems with contact lenses before an overwear episode can return to using the lenses after 2 or 3 days of wearing glasses but should be advised to build up wearing time gradually. A patient who was having chronic problems with lens comfort before the episode should check with an ophthalmologist before using the contact lenses again.

**Soft Contact Lenses**

Although there is also oxygenation of the cornea by way of the tear film with soft contact lenses, only approximately one tenth of the flow behind the lens that occurs with a hard lens is present during soft contact wear. [75] The high degree of lens gas permeability permits the majority of oxygenation to occur directly through the lens. The hydrogel lens is more comfortable than the hard contact lens because lid motion over the lens is smooth. The minimization of lid and corneal irritation allows a more rapid adaptation phase because the initial reflex-induced tearing and blinking changes are reduced. Nonetheless, the lenses may still lead to corneal edema and secondary hypoxic epithelial changes if worn for an excessive period when blinking is inhibited. Some individuals can tolerate the lenses for extended periods and may on occasion sleep with the contact lenses in place, although this practice is not encouraged. Newer extended-wear hydrogel lenses (e.g., Permalens) permit wear for several days without injury. These lenses are not discernible from standard soft lenses on examination.

Although the acute overwear syndrome that occurs with hard contact lenses can also occur with soft lenses, it is infrequent. More commonly, ocular damage from soft contact lenses falls into one of the 3 following categories:

1. *Corneal neovascularization.* Often the patient is asymptomatic, but on slit lamp examination fine vessels are seen invading the peripheral cornea. The treatment is to have an ophthalmologist refit the patient with looser or thinner lenses or with contact lenses that are more gas permeable.
2. *Giant papillary conjunctivitis.*[76] The patient notes decreased lens tolerance and increased mucus production. On examination of the tarsal conjunctiva (best seen on eversion of the upper lid), large papillae are seen. These grossly appear as a cobblestoned surface. The treatment is to discontinue wearing the lenses until the process reverses and then have the lenses refitted.
3. *A sensitivity reaction to the contact lens solutions* (usually thimerosol or chlorhexidine). There is diffuse conjunctival injection and sometimes a superficial keratitis. The treatment is to switch to preservative-free saline with the use of heat sterilization. Often, the contact lenses will need to be replaced before lens wear can be resumed.

All of these problems with soft lenses have bilateral, subacute onsets and do not require emergency treatment. The only form of ocular damage associated with soft contact lenses that is a true emergency is a bacterial (often *Pseudomonas* or *Acanthamoeba* with soft contact lenses) or fungal corneal ulcer. Because the nature of soft contact lenses is to absorb water, they also can absorb pathogens, which then can invade the cornea. This is especially true if the soft lens is worn continuously for extended periods of time. The patient presents with a painful, red eye associated with discharge and a white infiltrate on the cornea. Immediate ophthalmologic consultation is required for appropriate culturing and antimicrobial treatment because these infections can permanently affect the patient's visual acuity.

**Indications for Removal**

Removal of a contact lens is recommended in the following situations:

1. *Contact lens wearer with an altered state of consciousness.* The emergency physician should always be aware that the patient with a depressed or acutely agitated sensorium may be unable to express the need to have his or her contact lenses removed. Furthermore, it is likely that patients with a depressed sensorium will have decreased lid motion. During the secondary survey of these patients, the emergency physician should identify the presence of the lenses and should arrange for their removal and storage to prevent harm from excessive wear or possible accidental dislodgment at a later time. Without magnification, soft contact lenses may be difficult to see. Examination with an obliquely directed penlight should reveal the edge of the soft lens 1 to 2 mm from the limbus on the bulbar conjunctiva.
2. *Eye trauma with lens in place.* After measurement ofvisual acuity with the patient's lenses in place, the lenses should be removed to permit more detailed examination of the cornea. Fluorescein may discolor hydrogel lenses; when possible, extended-wear lenses should be removed before the use of this chemical. After the dye is instilled, the eyes should be flushed with normal saline; at least 1 hour should pass before reinsertion. [82] The availability of single-use

droppers of 0.35% fluorexon (Fluresoft) has permitted the safe staining of eyes when soft lenses are to be worn immediately after the examination. A limited eye irrigation after the use of fluorexon drops is still recommended before the reinsertion of soft contact lenses.

3. *Inability of the patient to remove the contact lens.* A patient may present with a hard contact lens that cannot be removed because of corneal edema from prolonged wear. Alternatively, the patient may present with a "lost" contact lens that is believed to be behind the upper lid. There is no urgency for contact removal in the out-of-hospital setting; hence, removal can wait until the patient has been evaluated by a physician.

## Contraindication to Removal

The only major problem with contact lens removal occurs when the cornea may have been perforated. In this case, the suction cup technique of removal described later is preferred.

## Procedure

### Hard Contact Lens Removal

A number of maneuvers have been devised for removal of the corneal lens. One technique is to first lean the patient's face over a table or a collecting cloth. The physician pulls the lids temporally from the lateral palpebral margin to lock the lids against the contact lens edges. The patient should look toward the nose and then downward toward the chin. This movement works the lower eyelid under the lower lens edge and flips the lens off the eye. The technique requires a cooperative patient, because the physician must pull the patient's lids tightly against the edge of the contact lens. The movement of the patient's eye then flips the contact free.

In the unresponsive patient, a modification of the technique can be used while the patient is supine. The physician takes a more active role in lid movement using the following procedure: One thumb is placed on the upper eyelid and the other on the lower eyelid near the margin of each lid. With the lens centered over the cornea, the eyelids are opened until the lid margins are beyond the edges of the lens (Fig. 67-18 (Figure Not Available) *A*). The physician then presses both eyelids gently but firmly on the globe of the eye and moves the lids so that they are barely touching the edges of the lens (Fig. 67-18 (Figure Not Available) *B*). One presses slightly harder on the lower lid to move it under the bottom edge of the lens. As the lower edge of the lens begins to tip away from the eye, the lids are moved together, allowing the lens to slide out to where it can be grasped (Fig. 67-18 (Figure Not Available) *C*). The physician should remember to use clean hands (and preferably wear examination gloves that have been rinsed in tap water or saline) when removing the lens.

Alternatively, one can move the lens gently off the cornea using a cotton-tipped applicator to guide the lens onto the sclera, where the applicator tip can be forced under an edge of the lens to flip the contact loose. Topical anesthesia is indicated when using an applicator and the patient is awake. Care must be taken with this technique to avoid

contact of the applicator with the cornea when the lens is moved off the eye. Perhaps the easiest technique is to use a moistened suction-tipped device and simply lift the lens off the cornea (Fig. 67-19) (Figure Not Available) .

Several lenses (those hard contact lenses that cover both the cornea and an amount of the sclera) can be removed by an exaggeration of the manual technique described earlier (Fig. 67-20) (Figure Not Available) . Elevation of the lens with a cotton-tipped applicator or a suction-tipped device is also an effective technique. Soft contact lenses should not be removed with a suction-tipped device because tearing or splitting of the lens may occur.

### Soft Contact Lens Removal

With clean hands (preferably using gloves rinsed in saline or tap water), the physician pulls down the lower eyelid using the middle finger. The tip of the index finger is placed on the lower edge of the lens. The lens is slid down onto the sclera and is compressed slightly between the thumb and the index finger. This pinching motion folds the lens and allows its removal from the eye (Fig. 67-21) (Figure Not Available) .

## Lens Storage

After a contact lens has been removed, it should be stored in sterile normal saline solution. It is best to use the patient's own storage container and, if available, the patient's lens solution. A variety of alternative sterile containers are available for use in the ED. One should be certain that right and left lenses are kept separate and in appropriately labeled containers. The containers should be kept with the patient until a friend or family member can procure them or should be locked with the patient's valuables.

## Evaluation of the "Lost Contact"

A patient may present with a request to be examined for a "lost" contact lens. The patient may be unsure if the lens is hidden under a lid, remains on the cornea, or is truly outside the eye.

The evaluation of the patient with a "lost" contact should begin, as should all eye examinations, with the measurement of visual acuity. Visual acuity is preferably measured using a 20-foot eye chart. A diminished visual acuity in the eye in which a patient just cannot seem to "take out" a soft contact lens may be the most convincing evidence that the lens is missing. Although transparent, soft contact lenses in proper position are usually seen easily when viewed closely with loupes or on slit lamp examination. The lens forms a fine line where it ends on the sclera several millimeters peripherally to the limbus. Hard contact lenses are even more evident as they change in position on the cornea.

If the contact is not evident on initial inspection, the lids are everted as discussed in the section on foreign body removal (double eversion of the upper lid). If the lens is still not

visible, a drop of topical anesthetic is placed in the eye. The upper fornix is gently swept with a wetted cotton-tipped applicator while the patient looks toward the chin. If the lens is still not evident although the patient remains insistent that it is in the eye, one may perform a fluorescein examination after explaining that the dye will color the lens (permanently). The upper lid should again be doubly everted and visualized using an ultraviolet light source.

If the lens remains elusive, the patient should be reassured that a thorough examination has been performed and that no object has been located under the eyelids or on the cornea. The cornea should then be examined for defects that warrant antibiotic ointment and a pressure patch placed (as discussed in the section on patching). Follow-up with the patient's eye specialist for a replacement lens and further reassurance is encouraged. One also should ask the patient to retrace movements at the time the contact began to give trouble or was missed and to check the clothing being worn for the presence of the lens. A final possibility is that the patient may have accidentally placed the 2 lenses together in the same side of the carrying case, causing them to stick together. In fact, patients have inadvertently placed one contact lens over the other--both in the same eye! One should note that hard contact lenses have been found embedded in conjunctival tissue under the upper lid (Fig. 67-22) (Figure Not Available) , at times for more than a year. [83] [84] Hence, a methodical approach as outlined earlier should be taken to ensure that no lens remains hidden in the eye.

**Complications of Lens Removal**

Unless care is used during lens removal, a corneal abrasion can occur. It may be difficult at times to determine whether the injury was produced by the patient or was a result of the physician's technique. Fortunately, the corneal injury is usually of a superficial nature and responds well to eye patching.

**Summary**

Contact lens removal is seldom a difficult task. More challenging situations are the identification of emergency patients at risk for corneal injury due to overuse, the evaluation of patients who cannot locate a soft lens, and the instruction of patients with contact lens-related problems concerning aftercare.

# INFECTIOUS KERATITIS

Infectious keratitis with corneal ulceration can have a variety of causes, including the overwear of contact lenses. Diagnosis of a corneal ulcer requires the use of a slit lamp and an accurate determination of the patient's history. Infectious keratitis is a frequent problem in ophthalmic practice. Herpes simplex is a common corneal pathogen. *Acanthamoeba* is another pathogen that has been particularly associated with contact lens use and exposure to organism-tainted environments. Patients presenting with a corneal ulcer require prompt referral to an ophthalmologist. When immediate referral to an ophthalmologist is not possible, bacterial and viral cultures need to be obtained and

therapy needs to be initiated.

Patients with herpes simplex keratitis will often give a history of prior episodes of the disease. Patients who undergo almost any form of corneal stress may sustain an activation of preexisting corneal disease. Herpes simplex keratitis is classically recognized by its dendritic pattern on fluorescein staining.

*Acanthamoeba* keratitis is a recently described disease with potentially devastating consequence. Its frequency seems to be increasing particularly in contact lens wearers, and its pathophysiology is not completely understood. Patients will often present with a red eye in which initial bacterial culture results are negative.

Bacterial keratitis can occur in a variety of settings. Organisms range from the relatively common *Staphylococcus* or *Streptococcus* to *Mycobacterium,* which can be difficult to identify. A variety of antibiotics are used against bacterial agents. Ciprofloxacin is a quinolone that has demonstrated efficacy against most of the common causative agents. Bacterial organisms in the cornea can develop resistance to any antibiotic and resistance to fluoroquinolones has been observed. [85] Ideally, treatment should follow culturing of the ulcer.

In instances in which a cellular infiltrate is seen on slit lamp examination and in which there will be a delay of hours before an ophthalmologic consultant can culture the patient, it may be prudent to initiate therapy with topical ciprofloxacin. In such circumstances, the emergency physician may obtain corneal cultures under the telephone guidance of the consultant before administering the antibiotic. One approach is to lightly touch a culture-moistened cotton-tipped swab against the ulcer and then streak standard culture media. If the ulcer has been chronic or the patient is immunocompromised, fungal cultures also should be obtained. Finally, a saline-moistened cotton-tipped swab can be used to obtain a Gram stain of the ulcer. Initiation of therapy before obtaining specimens for culture will make the subsequent identification of an organism difficult, and for this reason the immediate initiation of treatment must depend on the circumstances of the individual case.

## TONOMETRY

Tonometry is the estimation of intraocular pressure obtained by measurement of the resistance of the eyeball to indentation of an applied force. Elevated intraocular pressure is associated with visual field loss and blindness. Sudden elevation of intraocular pressure can follow trauma or occur with primary angle-closure glaucoma. Often, patients with primary angle-closure glaucoma come to the ED with systemic complaints that include nausea, vomiting, and headache; the emergency physician must determine the intraocular pressure and its relationship to the systemic symptoms. Occasionally, such patients are surprisingly free of pain in or about the eye.

Patients with an elevated intraocular pressure are at risk for retinal hypoperfusion when their systemic blood pressure is suddenly lowered. The emergency physician who treats patients in shock or uses potent antihypertensive agents needs to remain vigilant for the development of decreased retinal perfusion. Monitoring such patients for retinal artery

perfusion with the direct ophthalmoscope is helpful.

Ophthalmologists depended on tactile estimation of eye pressure until the 1860s, when von Graefe developed the first mechanical tonometer. [86] Applanation tonometry was introduced in 1885 by Maklakoff [87] but was not popularized until Goldmann improved the instrument in the 1930s. [88] Schiotz developed an impression tonometer in 1905 and modified it in the 1920s; this form is still in use today. [89] Aside from modifications in configuration, current tonometers closely resemble the devices popularized by Schiotz and Goldmann. The most dramatic variations are the Mackay-Marg tonometer, [90] which permits a continuous tonographic recording, and the noncontact tonometer, which is a pneumatic applanation tonometer. [91] Pocket-sized tonometers using the MacKay-Marg tonometer principle are available. One such device is the Tono-Pen XL (Mentor O & O, Inc, Norwell, Mass). [92] These devices are portable, lightweight, and relatively accurate, with built-in provisions for calibration. They have the advantage of using a one-time-use cover that eliminates concern about the possible transmission of an infectious agent.

**Tonometric Techniques**

Three tonometric techniques are reliable and clinically useful for estimating intraocular pressure:

1. The impression method uses a plunger 3 mm in diameter to deform the cornea and then the "indentation" is measured. This technique was popularized by Schiotz and commonly bears his name.
2. The MacKay-Marg method is a refined version of the impression technique in which smaller amounts of cornea are indented.
3. In the applanation method a planar surface is pressed against the cornea.

One can either measure the pressure necessary to flatten a defined area or determine the size of a flattened area produced by the defined pressure. These tonometric techniques are based on the Imbert-Fick law, which states that if a plane surface is applied with force (F) to a thin, spheric membrane within which a pressure ($P_t$) exists, at equilibrium the expression $P_t = F/A$ is valid if A is the area of the applied surface (Fig. 67-23) (Figure Not Available) . The Schiotz tonometer (Fig. 67-24) (Figure Not Available) actually measures the total intraocular pressure (initial pressure plus the pressure added by the weight of the tonometer and the plunger). Friedenwald [93] empirically found that a "rigidity coefficient" could be introduced to allow an estimation of the true intraocular eye pressure. One must be aware, however, that calculated conversion tables for Schiotz tonometers use an average estimate of the rigidity coefficient and hence are not accurate when eye rigidity is altered (e.g., after scleral buckle procedures for retinal detachment or with extreme myopia). Although the applanation tonometer (Fig. 67-25) (Figure Not Available) also increases the intraocular pressure during measurement, the applied pressure is much smaller and is partially countered by the surface tension of the eye tear film. Studies have shown the applanation tonometer measurements to be within 2% of the true intraocular pressure. [94]

The noncontact tonometer is a pneumatic applanation tonometer that permits

intraocular pressure measurement without eye contact. A pulsed air jet is used to deform the cornea. The technique is also dependent on ocular rigidity. Although readings taken by different examiners correlate well, the measurements are altered by the use of local anesthetics and show a wide standard deviation of measurement in patients with pathologic elevation of ocular pressure (when standard applanation tonometry is used as a reference). [95] Furthermore, the technique is not useful with corneal surface irregularities (e.g., corneal edema, keratoconus, corneal perforation) or when medications in viscous preparations have been used. The use of this type of tonometer is not recommended when accurate determination of the intraocular pressure is required. This type of device is primarily useful for glaucoma screening.

**Indications for Tonometry**

Measurement of intraocular pressure in the ED is part of any complete eye examination. Special situations in which tonometry is required are as follows:

1. *Confirmation of a clinical diagnosis of acute angle-closure glaucoma.* The middle-aged or elderly patient who presents with acute aching pain in one eye, blurred vision (including "halos" around lights), and a red eye with a smoky cornea and a fixed midposition pupil obviously needs a pressure reading. Sometimes the findings are less dramatic, and sometimes the patient complains mostly of nausea and vomiting that suggest a "flu" rather than an eye disorder.
2. *Determination of a baseline ocular pressure after blunt ocular injury.* Patients with hyphema often have acute rises in intraocular pressure because of blood obstructing the trabecular meshwork. [96] Later, angle recession can cause a permanent form of open-angle glaucoma. Arts and coworkers suggest that an intraocular pressure >22 mm Hg or a difference of 3 mm Hg or greater between eyes is a good marker for "ocular injury" in the setting of an orbital fracture. [97]

3. *Determination of a baseline ocular pressure in a patient with iritis.* Patients with iritis can develop both open- and closed-angle glaucoma as well as corticosteroid-induced glaucoma.
4. *Documentation of ocular pressure in the patient at risk for open-angle glaucoma.* All patients older than 40 with a familial history of open-angle glaucoma, optic disc changes, visual field defects, and pressures over 21 mm Hg should be referred to an ophthalmologist for further work-up. Referral should also be made for those patients with suspiciously cupped discs who have normal pressures; some of these patients may have "low pressure" glaucoma associated with visual field defects.
5. *Measurement of ocular pressure in patients with glaucoma and hypertension.* There is conflicting evidence  concerning the relationship between acute reductions in systemic blood pressure and further visual field loss in glaucoma patients. Progressive or rapid visual field loss is a rare but reported phenomenon in association with systemic blood pressure reduction. The prudent physician measures the intraocular pressure and consults with the glaucoma patient's ophthalmologist before instigating treatment for systemic hypertension. Consideration should also be given to the use of a beta-blocker to lower

intraocular and systemic pressures simultaneously.

## Contraindications to Tonometry

Tonometry is relatively contraindicated in eyes that are infected unless one is using a device such as the Tono-Pen XL, which uses a sterilized cover. [106] One should sterilize a tonometer before and after applying it to a potentially infected eye. Infected eyes are preferably measured with either a noncontact tonometer or a device with a covered tip (e.g., Tono-Pen). The contact portions of any device should be swabbed with alcohol and allowed to dry before use on another eye. Not all viruses may be destroyed by alcohol cleansing. Hydrogen peroxide is effective for deactivating the human immunodeficiency virus responsible for the acquired immunodeficiency syndrome (AIDS). Ultraviolet sterilization, cold-sterilizer bathing of the footplate and plunger, and ethylene oxide sterilization have been advocated as alternatives to sterilize the Schiotz tonometer tip. The Schiotz tonometer may also be used with sterile disposable coverings (marketed as Tonofilm). Nonetheless, measurement of intraocular pressure in an obviously infected eye can be deferred until a subsequent visit to the ED or private physician unless the red eye demands an immediate determination of intraocular pressure.

Examples of a need for immediate tonometry in the setting of a red eye are suspected angle-closure glaucoma (acute onset of redness and pain in the eye with smoky vision, a cloudy cornea, and a fixed pupil in mid-dilation) and iritis (ciliary injection with photophobia), in which secondary angle-closure or corticosteroid-induced pressure changes may occur. Reported cases of conjunctivitis spread by tonometry predominantly tend to be viral infections. Particular efforts should be made to avoid use of the instrument on patients with active facial or ocular herpetic lesions or on patients who may have AIDS.

The presence of corneal defects also represents a relative contraindication to tonometry. [20] The use of a tonometer on an abraded cornea may lead to further injury and is commonly deferred until a subsequent visit. Patients who cannot maintain a relaxed position (e.g., because of significant apprehension, blepharospasm, uncontrolled coughing, nystagmus, or uncontrolled singultus) are unlikely to permit an adequate examination and can receive corneal injury when sudden movements occur during an examination. Furthermore, tonometric examination, with the exception of the palpation technique (through the lids) and the noncontact method, should not be performed on a cornea without complete anesthesia.

Tonometry should *not* be performed with a suspected penetrating ocular injury. [106] Globe perforation may be exacerbated by pressure on the globe with resultant extrusion of intraocular contents. Slit lamp examination can be used for detection of a possible perforation.

## Procedure

**Palpation Technique**

All forms of tonometry are essentially ways of determining the ease of deforming the eye; an eye that can easily be deformed has a low pressure. The most direct way to do this is simply to press on the sclera through the lids and grossly compare one eye with the other. One can easily distinguish the rock-hard eye of acute glaucoma from the normal opposite eye by this method. Another method is to anesthetize the eyes topically and press a wetted applicator on the sclera of each eye. Again, eye deformation is inversely related to ocular pressure. Rigidity of the globe also is a factor in this crude method of tonometry.

**Impression (Schiotz) Technique**

Use of the Schiotz tonometer requires relaxation on the part of the patient and steadiness on the part of the physician. The patient is placed in either a supine or a semirecumbent position and is instructed to gaze at a spot directly above the eyes. A spot on the ceiling should suffice; alternatively, the patient can stretch the arm up over the head and gaze at the thumb. A drop of topical anesthetic is placed in each eye. After the irritation of the drop passes, the patient is allowed to blink while the physician blots the tears away with a tissue. Rubbing the eyes lowers intraocular pressure. The patient is reassured that further discomfort during the procedure will not occur.

The patient *keeps both eyes wide open and fixed on an object*, and the physician separates the eyelids on the side to which he or she is standing. Care must be taken to direct pressure onto the orbital rims rather than into the orbit, because pressure directed into the orbit falsely raises the reading (Fig. 67-26) (Figure Not Available) . The tonometer is momentarily held over the open eye, and the patient is informed that the instrument will block vision in the one eye. The patient is instructed to continue to gaze at the fixation point as though the instrument were not there. After the patient relaxes the involuntary muscle contraction that occurs when the instrument is first placed in the line of sight, the instrument is gently lowered onto the middle portion of the cornea. The instrument should be vertically aligned with the footplate resting on the cornea; the reading should be in midscale. Should the reading be on the low end of the scale (<5 units), additional weight should be added to the plunger after the instrument has been removed. The process should be repeated as before with the additional weight.

The opposite eye should be measured in the same fashion. A converted scale reading giving an intraocular pressure of >21 mm Hg requires ophthalmologic referral (Table 67-4) . Patients with elevations of intraocular pressure >30 mm Hg require more urgent consultation and initiation of therapy.

| **TABLE 67-4** -- Schiotz Tonometry |
| --- |
|  |

| Tonometer Scale Reading (Units) | Tonometer Weights (g) | | |
|---|---|---|---|
| | 5.5 (mm Hg) | 7.5 (mm Hg) | 10 (mm Hg) |
| 2.50 | 27 | 39 | 55 |
| 3.00 | 24 | 36 | 51 |
| 3.50 | 22 | 33 | 47 |
| 4.00 | 21 | 30 | 43 |
| 4.50 | 19 | 28 | 40 |
| 5.00 | 17 | 26 | 37 |
| 5.50 | 16 | 24 | 34 |
| 6.00 | 15 | 22 | 32 |
| 6.50 | 13 | 20 | 29 |
| 7.00 | 12 | 18 | 27 |

| | | | |
|---|---|---|---|
| 7.50 | 11 | 17 | 25 |
| 8.00 | 10 | 16 | 23 |
| 8.50 | 9 | 14 | 21 |
| 9.00 | 8 | 13 | 20 |
| 9.50 | 8 | 12 | 18 |
| 10.00 | 7 | 11 | 16 |

The table provides estimates of the intraocular pressure to the nearest mm Hg for the different weight of the Schiotz tonometer. Accuracy is most dependable with scale readings larger than 5. If the scale reading is less than 5, use the next highest weight that will give a reading of 5 or more.

Associated symptoms or signs of angle-closure glaucoma (primary or secondary) represent an ophthalmologic emergency. [107]

**Errors with impression tonometry.**

Inaccurate readings can occur with the Schiotz tonometer for a variety of reasons. If the plunger is sticky, falsely low readings may be obtained. Plunger motion and the zero point of the tonometer should be checked on a firm test button before use. A sticky plunger can be cleaned with isopropyl alcohol and dried with a tissue. When the lids are held open, pressure directed into the orbit elevates the intraocular pressure and provides a falsely elevated reading. The following eye movements have been found to elevate the intraocular pressure: closure of the lids (increase by 5 mm Hg), blinking (increase by 5 to 10 mm Hg), accommodation (increase by 2 mm Hg), and looking toward the nose (increase by 5 to 10 mm Hg). [108] Repeated measurements or prolonged measurements have been found to lower the intraocular pressure approximately 2 mm Hg and may also lower the pressure in the opposite eye. [109] As mentioned in the introduction to this section, the calibration of the Schiotz tonometer is

based on a mean rigidity coefficient. Factors that produce a reduction in ocular rigidity falsely lower the measured pressure. These factors include high myopia, anticholinesterase drugs, overhydration (e.g., 4 large cups of coffee or 6 cans of beer), and scleral buckle operations. [108] [110]

Ocular pressure measurements can vary with ocular perfusion. When measured after a premature ventricular contraction, the intraocular pressure may be reduced as much as 8 mm Hg. [111] Similarly, decreased venous return as produced by breath holding, the Valsalva maneuver, or a tight collar can increase the intraocular pressure. [108]

**Impression (Tono-Pen XL) Technique**

When using this device, the preparations for testing are similar to those for the Schiotz device. The patient is encouraged to relax, and a topical anesthetic is used to numb the corneas. The patient should be encouraged to stare with both eyes at a distant object during testing. As noted previously, the operator can help separate the eyelids, but any pressure on the globe itself should be avoided. One major advantage to the use of the Tono-Pen XL is that the patient may be evaluated in any position as long as the device is applied perpendicular to the corneal surface. Another advantage is that the device can be used in cases of irregular or high corneal astigmatism.

Although ideally the complete instructions that are provided with the device kit should be consulted prior to each use, the following synposis is provided to help in circumstances in which instructions are unavailable. It is assumed that the previously mentioned preparations for testing have been made before the device is used.

The probe tip is first sprayed with compressed gas to clean the mechanism and ensure its free movement. An Ocu-Film (latex) cover is placed snugly (but without tension) over the probe tip.

Calibration is performed before use at least once each day. The *activation* switch is momentarily depressed and released. The liquid crystal display (LCD) should show "----". If the device beeps and "====" appears on the LCD, the activation switch should be pushed again so that the "----" reappears. If the prior calibration was "bAd" (on LCD), a long beep sounds, followed by "CAL" (on LCD). A short beep follows and then the desired "----" is displayed.

Once the "----" is displayed, the probe is held vertically with the tip pointing straight down. The activation switch is then pressed and released twice in rapid succession. Two beeps will then sound, and "CAL" will appear (on LCD). The probe is held in this position (up to 20 seconds) until a beep sounds and "-UP-" appears (on LCD). The probe is then immediately turned 180° so that the tip points straight up. In a few seconds, another beep occurs, and the LCD changes. If the LCD reads "Good", the calibration was successful. If the LCD reads "bAd", the calibration was unsuccessful.

With an unsuccessful calibration, repeat the calibration steps above until two consecutive "Good" readings are obtained. If further attempts are unsuccessful, loosen

the Ocu-Film tip cover and repeat the calibration process. If attempts are still unsuccessful, press the RESET button and repeat the process. If still unsuccessful, use compressed air to clean the probe tip and repeat the process. If still unsuccessful, the battery should be replaced and the process repeated. Continued failure warrants a call to the MENTOR Technical Service Group at 800-992-7557 or 617-871-6950.

Measurement occurs once the device is calibrated and the patient is prepared as outlined earlier. The activation switch is again depressed and released to obtain "====" (on LCD). A beep will occur with this change. If the switch is not depressed long enough, the LCD will be blank. If a blank screen is seen, again use the activation switch to obtain "====" (on LCD). The probe is held like a pen and briefly and lightly touched to the cornea. This is done four times. A click will sound and a reading will appear on the LCD each time a valid reading is obtained. After four valid readings, a final beep will sound and the averaged measurement will appear on the LCD. The number represents the intraocular pressure in millimeters of mercury. The associated bar reflects the statistical reliability (a reading of >20% reflects an unreliable measurement, which should be repeated).

If four dashes ("----") appear on the LCD after the final beep, too few valid readings were obtained. In such a case, the probe can be reactivated (without recalibration) and the measurement procedure repeated. If the probe is not reactivated within 20 seconds, the LCD will clear, but the device can be activated as noted previously without recalibration.

The values are interpreted as outlined earlier for the Schiotz device. Readings may be affected by the same features noted as causes of errors with impression tonometry via the Schiotz device. The device should be stored with a unused Ocu-Film cover protecting the probe tip.

**Applanation Technique**

One can perform this technique using a slit lamp attachment for an applanation tonometer with the patient's head stabilized in the headrest of the slit lamp (see the following section on the slit lamp examination) (Fig. 67-27) (Figure Not Available) . [112] [113] A portable device also is available and is similar in principle. The portable device is not discussed specifically.

The patient must be comfortable and relaxed. The physician should anesthetize the eye as discussed previously, avoiding ocular pressure, which can lower the subsequent measurements. Fluorescein should be applied to each eye. Excess fluorescein should be blotted from the eye. The patient's head should be in the slit lamp with the forehead firmly against the headrest, and the physician should direct the patient to gaze straight ahead. One can use a light for fixation or can ask the patient to focus on the physician's ear on the side opposite the eye being examined.

The cobalt blue light filter is placed in the light beam, and the slit diaphragm is opened fully. The light arm is angulated to shine on the applanation prism in the region of the encircling black line near the anterior prism tip at an angle of 45° to 60° to the line of observation. The voltage is turned to the maximum setting, and the low-power

microscopic system is focused through the plastic prism so that the front face is clearly seen through the chosen eyepiece. The pressure knob of the tonometer is turned to 1 g (10 mm Hg), bringing the prism arm to its forward stop. Thus, when corneal contact is made, the prism will be exerting only light pressure. The room lights are dimmed.

The patient's eye that is being examined and the applanation prism are watched from the side (or with the operator's eye not sighting through the microscope) as the instrument is brought forward by the joystick control until gentle contact is made between the prism face and the corneal center. Contact is evidenced by an immediate bluish glow throughout the limbus. The patient's lids must be wide open and unblinking. Contact with the lid margins produces reflex blinking, and the lids may require separation by the physician's fingers. Pressure during lid separation must be exerted only against the orbital rims. Through the microscope, the physician sees 2 blue semicircles (surrounding the flattened area of cornea). Each semicircle is bordered by an arc of green light and pulses synchronously with the cardiac rate (Fig. 67-28) (Figure Not Available) .

The semicircles should be of equal size; their width should be approximately one tenth the diameter of the flattened surface contained within each arc. If the semicircles are grossly widened, either excessive tears are present or the prism was probably wet before contact. A wet prism must be withdrawn, dried, and reapplied. If the semicircles are grossly narrowed, the tear film has dried excessively. In this case, the prism must be withdrawn and the patient instructed to blink several times before contact with the cornea is attempted again. If the semicircles are so broad that they extend beyond the illuminate field, there is excessive flattening and the slit lamp must be drawn back. If the semicircles suddenly shrink, either the patient has moved back or the instrument has been backed away from the eye. The semicircles should be of equal extent above and below a horizontal dividing line. If the dividing line is not horizontal, the applanation prism assembly should be rotated on its holder until the line is horizontal. If the semicircles are not equally divided above and below the line, vertical adjustments of the slit lamp should be made.

Readings should be taken at approximately the midpoint between systole and diastole, when the inner (concave) boundaries of each semicircle rhythmically glide past each other through excursions of equal distance (see Fig. 67-28 (Figure Not Available) C). One finalizes adjustments to the end point of properly located and sized semicircles by rotating the pressure knob back and forth. When applanation pressure exceeds intraocular pressure, the semicircles are too small to intersect.

At the end point is a flattened disc area 3.06 mm in diameter within the 7-mm diameter of the prism face. Here the attractive surface tension of the tears toward the prism is counterbalanced by the elasticity, or springiness, of the cornea; at this point the grams of force applied through the prism (indicated on the pressure knob) are directly convertible (when multiplied by 10 into millimeters of mercury) to express intraocular pressure. With an applanation tonometer, the average intraocular pressure in a seated adult is 14 to 17 mm Hg.

After use, the tonometer should be wiped dry and removed for storage if used

infrequently in the ED. One should verify the pressure adjustment periodically using the test weight or metal balance bar supplied with the instrument.

Potential sources of error with the applanation tonometer are similar to those mentioned for the impression tonometer, with the exception that ocular rigidity is not a factor. Inaccuracies primarily result from ocular motion or tensing of the lids.

### Complications

When tonometric instruments are used properly and reasonable precautions are taken, complications are unusual. The eye with preexisting corneal injury should be spared the additional trauma of tonometer placement. Corneal abrasions can be produced by ocular movement during testing. In particular, patients with uncontrollable nystagmus, singultus, or coughing or those who are extremely apprehensive should not be subjected to tonometry. Infection can be transmitted by the use of the instrument. Careful cleansing of the device and avoidance of tonometry in patients with obvious conjunctivitis, corneal ulcers, or active herpetic lesions should minimize the risk of spreading the infection to the unaffected eye or to subsequent patients. Although protective coverings can be placed over the tonometer contact, tonometry can usually be postponed in the aforementioned individuals until the risk of infection is minimal. Extrusion of ocular contents with penetrating injuries is a potential but rare complication.

### Summary

Tonometry is an easily learned technique that should be used by the emergency physician for the detection of elevated intraocular pressure. An elevated intraocular pressure in conjunction with physical findings suggestive of acute angle-closure glaucoma is an indication for therapy and consultation with an ophthalmologist. The baseline measurement of intraocular pressure will aid the ophthalmologist in subsequent evaluation of a referred patient. In addition, the emergency physician can serve as a referral source for patients with elevated intraocular pressure who are suspected of having open-angle glaucoma. In particular, future drug therapy for systemic hypertension may be altered by the presence of concomitant intraocular hypertension. The emergency physician who aggressively manages patients with hypertensive crises must also be aware of potential visual field defects when systemic blood pressure is vigorously lowered without concurrent lowering of intraocular pressure.

## SLIT LAMP EXAMINATION

The slit lamp is an extremely useful instrument; it makes the examination of the anterior segment of the eye a pleasure. The instrument can reveal pathologic conditions that would otherwise be invisible. The slit lamp permits detailed evaluation of external eye injury and is the definitive tool for diagnosing anterior chamber hemorrhage and inflammation. The emergency physician should not attempt to diagnose any but the simplest of eye problems without the aid of a slit lamp.

Since the 1800s, physicians have searched for a better way both to magnify and to illuminate the anterior segment of the eye. In 1891, Aubert developed the first true binocular stereoscopic microscope. Then, in 1911, Gullstrand introduced a slit illuminator device. The microscope and the illuminator were combined by Henker in 1916; the result was the first true slit lamp. Goldmann improved the mechanical supports for the microscope and the illuminator and in 1937 marketed a slit lamp that resembles closely the device that is used today. [114]

## Indications and Contraindications

The slit lamp can be used in every eye examination. It is especially useful in the ED for the diagnosis of corneal abrasions, foreign bodies, and iritis. [115] The slit lamp facilitates foreign body removal and is also used in conjunction with most applanation tonometers. Although portable slit lamp instruments exist, emergency physicians generally have access only to a stationary, upright device. Therefore, in the absence of a portable device, a slit lamp examination is contraindicated in patients who cannot tolerate an upright sitting position (e.g., those with orthostatic syncope).

## Equipment

The slit lamp has 3 essential components: a binocular microscope mounted horizontally, a light source that can create a beam of variable width, and a mechanical assembly to immobilize the patient's head and to manipulate the microscope and the light source. The location and arrangement of the knobs that control these components vary in devices made by different manufacturers. Usually, by simply turning each knob and watching the results, one can quickly master a new machine. Figure 67-29 (Figure Not Available) illustrates the location of the functional controls on one particular instrument.

The first knob that one should locate is the on/off switch for the entire machine. Often this switch incorporates or is adjacent to a rheostat that provides 2 or 3 different power settings. The lowest setting is adequate for routine examination and will preserve bulb life. One can use a high-intensity setting when examining the anterior chamber with a narrow slit beam. Often, these controls are located on a transformer placed beneath the table to which the slit lamp has been attached. The second knob that one should find is the locking nut for the mechanical assembly. This must be loosened for the assembly to be moved.

The patient should be comfortable while sitting with the head in the device. The patient's forehead should be firmly against the headrest, and the chin should be in the chin rest. By varying the table height and height of the chin rest, one should be able to maximize the comfort of the patient's neck and back. The chin rest should be adjusted to align the patient's eye level with the mark on the headrest support rods.

The binocular microscope has a control for varying the magnification. Usually low powers, such as 10× or 16×, are the most useful. A higher power is helpful when the anterior chamber is examined for cells and flare and when the cornea is examined in

minute detail. The binocular interpupillary distance should be adjusted to match that of the examiner. One can focus the eyepieces by moving the instrument forward and backward until the narrowed vertical beam is sharpest on the patient's cornea when viewed with the unaided eye. Then, while viewing through each eyepiece individually, the physician adjusts the focus of each to produce a sharp image of the anterior cornea.

The light source is mounted on a swinging arm. There are knobs to vary the width and the height of the light beam. There are also filters that can be "clicked" in; only white and blue filters are usually needed. The angle of the slit beam can be varied from vertical to horizontal. The vertical alignment is preferred for routine examinations in the ED.

Both the microscope and the light source are mounted on swivel arms, linked at their base to a movable table. One can change the position of this table by pushing on any part of it. For finer movements, the physician uses a joystick. One can vary the height of the microscope and the light source by twisting either the joystick or a separate knob at the base, depending on the design of the instrument.

**Procedure**

There are 3 setups that every slit lamp operator must know. [116] [117] The first is for an overall screening of the anterior segment of the eye. For examination of the patient's right eye, the light source is swung to the examiner's left at a 45° angle while the microscope is directly in front of the eye. The slit beam is set at the maximum height and the minimum width using the white light. To scan across the patient's cornea, one first focuses the beam on the cornea by moving the entire base of the slit lamp forward and backward. One then moves the whole base left and right to scan across. The 45° angle between the microscope and the light source should not be varied. The most common mistake is to try to scan by swinging the arm of the light source in an arc; this does not work because the light beam will remain centered on the same point of the patient's eye. The examiner scans across at the level of the conjunctiva and the cornea and then pushes slightly forward on the base and scans at the level of the iris. The depth of the anterior chamber is determined by this low magnification setup. When the depth of the anterior chamber is reduced, one should suspect a corneal perforation or a predisposition to angle-closure glaucoma.

This basic setup can also be used to examine the conjunctiva for traumatic lesions, inflammation, and foreign bodies. The lids can be examined for hordeolum, blepharitis, or trichiasis. Complete lid eversion (as described in the section on foreign body removal) can be performed in conjunction with the slit lamp examination to permit evaluation of the undersurface of the upper lid for foreign body retention.

Corneal foreign body removal can be enhanced by use of the slit lamp. In particular, the instrument allows stabilization of the patient's head. Magnification also minimizes corneal injury during foreign body or rust ring removal. The upper eyelid may be immobilized by a cotton-tipped applicator, as discussed previously. The physician's hand can be steadied against the patient's nose, cheek, or forehead or against the support rods of the headrest. The patient should be instructed to stare straight ahead at

a fixed light or at the physician's ear during removal of the foreign body.

The second setup is essentially the same as the first but uses the blue filter. The purpose is to identify any areas of fluorescein staining. After fluorescein is applied, the blue filter is "clicked" into position, and the beam is widened to 3 or 4 mm. A patient can tolerate a wider beam without photophobia if it is blue. Corneal defects (as discussed in the section on the fluorescein examination) are sought with this setup. The blue filter is also used with applanation tonometry, as discussed in the section on tonometry.

The purpose of the third setup is to search for cells in the anterior chamber--either the white cells of iritis or the red cells of a microscopic hyphema. The height of the beam should be shortened to 3 or 4 mm and should be as narrow as possible. The microscope should be switched to high power. The beam is first focused on the center of the cornea and is then pushed forward slightly so that it is focused on the anterior surface of the lens. When the joystick is again pulled back to a focus point midway between the cornea and the lens, it will be focused on the anterior chamber (Fig. 67-30) . One should keep the beam centered over the pupil so that there is a black background. Normally, the aqueous humor of the anterior chamber is totally clear. If small particles are visible floating up or down through the beam, these are usually circulating cells. If the beam lights up the aqueous like a searchlight in the fog, then the examiner has found the protein flare that accompanies iritis. Note should be made of the fact that fluorescein can penetrate an abraded cornea, producing a fluorescein flare on slit-lamp evaluation. To avoid confusion, some physicians prefer to examine for anterior chamber flare before the stain is used.

**Summary**

In practice, the 3 setups described here take only 1 minute per eye. Experience with the instrument enhances the ability of the user. The device is helpful for the evaluation of ocular infections and corneal lesions, the removal of corneal foreign bodies, the measurement of intraocular pressure by applanation tonometry, and the diagnosis of iritis.

# UNILATERAL LOSS OF VISION

There are a variety of reasons that an individual may sustain a complete loss of vision in one eye, but most commonly such loss may be related to occlusion of the central retinal vein or the central retinal artery or to optic nerve damage. Less commonly, pressure in the orbit from a retro-orbital hemorrhage may compromise the ophthalmic artery.

Although discussion of all the potential causes of unilateral loss of vision is beyond the scope of this text, amaurosis fugax desires special mention. Amaurosis fugax is a transient loss of vision that is most commonly due to cholesterol or platelet emboli from atherosclerotic carotid occlusive disease. When plaques are visualized in the retinal vasculature, it is prudent to auscultate for carotid bruits and to refer the patient for ultrasound examination of the carotid artery.

**Central Retinal Artery Occlusion**

The patient with central retinal artery occlusion generally presents with a recent sudden (complete or nearly complete) unilateral vision loss. On examination there is an afferent pupillary defect (i.e., sluggish or nonreactive pupil in the affected eye with direct illumination with a normal consensual response) and reduced visual acuity. Immediately after the event, the fundus may appear nearly normal; however, it soon becomes pale and a classic "cherry-red spot" in the macula may be evident as a result of patent choroidal vessels showing through the transparent fovea.

**Therapy**

If the patient presents within 2 hours after the initial onset of symptoms, immediate attempts at improving retinal flow are indicated. [120] Studies in laboratory animals have demonstrated visual recovery after central retinal artery occlusion for over 1½ hours. [121] Ophthalmologic consultation should be made while initiating therapy.

Although of unknown value, slow *rebreathing* into a paper bag is believed to increase the arterial $CO_2$ level, thus aiding vasodilation and permitting the occlusion to move more peripherally, possibly reducing the ischemic area. The physician should concurrently initiate *digital globe massage*. With the patient lying supine, firm steady pressure is applied to the affected globe through the patient's closed lids using the physician's thumb. The pressure is applied for 5 seconds and then abruptly released. The procedure is immediately repeated several more times. The technique is intended to help break up the occlusion and to encourage its movement more peripherally.

A more aggressive therapy is *anterior chamber paracentesis*. This technique should be considered when central retinal occlusion is recent and unresponsive to the above therapeutic approaches. For this procedure, the patient is kept supine with the head and eyelids secured. The cornea is anesthetized with topical anesthetic drops (e.g., 0.5% proparacaine drops) and the conjunctiva is anesthetized. The conjunctiva is injected adjacent to the limbus using a 27- or 30-ga needle until the entire perilimbal area is infiltrated, giving the appearance of chemosis in all quadrants. During the remainder of the procedure, an assistant must firmly grasp the conjunctiva with toothless forceps at the 3 and 9 o'clock positions to stabilize the eye. A 30-ga needle on a tuberculin syringe is then inserted obliquely just adjacent to the limbus, at either the 4:30 or 7:30 o'clock position and directed toward the 6 o'clock position to avoid the lens (Fig. 67-31) (Figure Not Available) . After 1 to 2 drops of aqueous are expressed with gentle pressure on the globe, the needle is withdrawn.

**Complications**

Overzealous globe massage has the potential to produce intraocular trauma including retinal detachment and intraocular hemorrhage. Anterior chamber paracentesis may produce hemorrhage, infection, or mechanical injury to the cornea, iris, or lens. [124] Although these complications are rare, ophthalmologic consultation for assistance with

the underlying central retinal artery occlusion and surveillance for these potential complications should be initiated on an emergent basis.

**Retrobulbar Hemorrhage**

Acute facial trauma may produce retrobulbar hemorrhage with sufficient pressure to compromise the ophthalmic artery. A form of post-traumatic glaucoma may also occur when the retrobulbar hematoma forces the globe against the eyelids and intraocular pressure rises precipitously, because the globe is in a relatively closed space due to the firm attachment of the eyelids to the orbital rim by the medial and lateral canthal ligaments. In this situation an emergency canthotomy should be considered for relief of the pressure on the eye.

Ophthalmoscopic evaluation should reveal a blanched ophthalmic artery in the presence of obvious retrobulbar pressure and ecchymosis around the eye. The patient exhibits *decreased visual acuity,* and an afferent pupil defect is often seen. The intraocular pressure is markedly elevated but may be relieved by an emergency lateral canthotomy. Such a procedure needs to be performed quickly because the ischemic retina will not retain function if it is deprived of blood for a long period of time.

### Technique

The goals of the procedure are to release pressure on the globe and to decrease intraocular pressure enough to reinstitute retinal artery blood flow. Because retinal recovery will be unlikely to occur if rapid relief of ischemia is not accomplished, taking time to clean the eye beyond simple saline cleansing of the lids and lateral canthus is ill-advised. While the patient's head and lids are stabilized, the lateral canthus is first anesthetized with injectable 2% or 4% lidocaine with epinephrine. A small hemostat is used to crush the lateral canthus for 1 to 2 minutes to minimize bleeding before incising the lateral canthus. The canthus is incised using iris scissors, with precautions taken to avoid injury to the protruding globe (Fig. 67-32) (Figure Not Available) . The incision begins at the lateral canthus and extends toward the orbital rim. The lateral canthal ligaments are found and released from the orbital rim.

### Complications

Although hemorrhage, infection, and mechanical injury may result from the procedure, these complications generally respond to therapy better than retinal injury from prolonged ischemia. Emergent ophthalmologic consultation should be obtained, although when the procedure is indicated, it should not be delayed pending arrival of the consultant. Lateral canthotomy incisions generally heal without suturing or significant scarring.

# REDUCTION OF GLOBE LUXATION

Although luxation of the globe is uncommon, the emergency physician should be aware of the condition and its mechanisms, know how to reduce the globe, and know when to

prioritize ophthalmologic consultation. With luxation of the globe there is extreme proptosis, which permits the lids to slip behind the globe equator (Fig. 67-33) (Figure Not Available) . Subsequent spasm of the orbicularis oculi muscles sustains the luxation and limits extraocular movements. Traction on the optic nerve and retinal vessels may produce direct or indirect injury to the optic nerve and retina.

Luxation may be spontaneous, voluntary, or traumatic. A variety of conditions (e.g., orbital neoplasms, Graves' disease, histiocytosis X, cerebral gumma, and craniofacial dysostoses) may predispose the patient to luxation. Triggering events include maneuvers increasing intraorbital pressure (e.g., the Valsalva maneuver), trauma to the orbit or forehead, or eyelid manipulation.

### Indications and Contraindications

Early globe reduction is indicated to relieve symptoms and to minimize visual impairment. Attempts at reduction in the ED are relatively contraindicated when there is obvious rupture of the globe.

### Technique

Before globe reduction, it is valuable to perform a rapid eye examination to document visual acuity, range of eye motion, pupillary reactivity, and any evidence of globe rupture (see earlier). [125] The patient is made comfortable in a recumbent position, and a topical ocular anesthetic agent (e.g., 0.5% proparacaine) is administered. When the lashes are visible, an assistant should apply steady outward and upward traction while the globe is gently pushed behind the lids. The globe is manipulated back into the orbit using gloved fingers to apply steady scleral pressure. When the lashes cannot be grasped, a lid retractor may be introduced behind the lid to provide countertraction. Others recommend placement of a suture through the anesthetized skin of each lid to provide countertraction.

After the procedure, a repeat eye examination documenting visual acuity and extraocular movement is warranted. It is not uncommon for return of full visual function to be delayed for several days, occasionally longer.

### Complications

It is common with this procedure for lashes to be retained in the conjunctival fornices. It is important to evaluate for and remove any free lashes to prevent corneal injury. Edema, retrobulbar hemorrhage, or orbital deformity may prevent outpatient reduction. When reduction is not possible in the ED, saline drops should be applied to the globe and a noncontact eye shield applied.

### Aftercare

Patients with spontaneous luxation and no visual impairment in whom the globe is easily reduced warrant follow-up within 24 to 48 hours. Instructions to avoid potential triggering

maneuvers should be given. Recurrent luxation may warrant lateral tarsorrhaphy. Further evaluation of potential precipitating illness can be pursued on an outpatient basis.

Patients with traumatic luxation are at greater risk for underlying ophthalmic injury and warrant emergent consultation. A computed tomographic scan of the orbit is helpful for evaluating both the soft tissue and bony structures about the globe.

# Chapter 68 - Otolaryngologic Procedures

**David E. Manthey, Benjamin P. Harrison**

Examination of the oropharynx, larynx, ear canals, and nasal passages and the management of related acute disorders are most effectively performed using special equipment and techniques. This chapter addresses these techniques from the perspective of the emergency physician who often must assess injuries or illnesses of potential compromise to the airway and to auditory function. Decisions related to definitive treatment in the emergency department (ED) vs timely referral are addressed. Related topics, including airway management, esophageal and laryngeal foreign bodies, and assessment of caloric testing, are discussed elsewhere.

## PHARYNX

### Examination of the Larynx

Several techniques to visualize the larynx are discussed and the clinician should become adept with more than one method of examining the hypopharynx and larynx (Fig. 68-1) . Laryngoscopy is indicated for the evaluation of unexplained hoarseness, dysphagia, odynophagia, or foreign body sensation. The majority of patients will require a repeat examination by an otolaryngologist for verification, but laryngoscopy in the ED may identify pathologic conditions requiring more urgent consultation. Latex gloves should be worn at all times while performing any of the following methods of examination to minimize exchange of body fluids.

Traditionally, laryngoscopy has been discouraged in the patient with a high potential for epiglottitis, as oropharyngeal manipulation may rarely precipitate laryngospasm and acute respiratory arrest. However, some authors report that careful laryngoscopy may be performed in stridulous patients with the presumed diagnosis of croup to rule out epiglottitis when the suspicion for the latter condition is low. [1]

When impending airway obstruction from epiglottitis is suspected, the first priority is to quickly assemble a predesignated team (usually consisting of an anesthesiologist and an otolaryngologist in the operating room). Any attempt at laryngoscopy should follow full preparation for rigid bronchoscopy or a surgical airway. Patients with severe laryngeal trauma or partially obstructing hypopharyngeal foreign bodies should be approached in a similar manner.

#### Illumination

The reflected light from a head mirror or direct illumination from a head lamp can be used not only for indirect laryngoscopy, but also for inspection of the oropharynx, nares, and auditory canal. The advantages of a head mirror are the high degree of brightness it provides into deep recesses and its simplicity of design. Generally, the beam of light

from an electric head lamp is easier to focus than is the head mirror.

**Procedure**

**Head mirror/light source.**

The head mirror is convex with a central hole that allows the examiner to see directly along the reflected light beam. Begin by swinging the mirror down over the dominant eye just touching the skin or glasses. Position the light source (a 150-watt bulb works well) over the patient's shoulder on the same side as the head mirror. Keeping your eyes open, adjust the focus of light by adjusting your distance from the patient. Change the direction of the beam by tilting the mirror or turning your head slightly.

**Head lamp.**

The electric head lamp also attaches by a forehead strap, and the light should be placed nearly between the examiner's eyes to be maximally effective. The intensity of light in this position is nearly as bright as the head mirror. After securing the head lamp, hold your hands in front of you at a distance that is comfortable for working. Focus the beam of light at that point by adjusting the head lamp into position without moving your head or eyes. This allows for the beam to shine on and follow the area on which your eyes are focused.

**Indirect Laryngoscopy**

This traditional method is most commonly used by the otolaryngologist, but it has some application in the emergency setting if the necessary equipment is readily available. The clinician who is unfamiliar with this method should practice frequently, as it requires significant eye-to-hand coordination to reflect the light beam off the angulated mirror onto the larynx. When this procedure is properly performed, most patients are able to tolerate it without anesthesia of the oropharynx. In EDs, where fiberoptic nasopharyngeal scopes are available, the latter technique has largely replaced indirect laryngoscopy.

Begin by establishing a rapport with the patient by explaining how the examination will be performed. Have the patient sit erect in the "sniffing position," with the feet flat on the floor and leaning slightly forward. Warm the mirror with warm water or in a flame to prevent fogging, but check the temperature of the mirror with your hand before placing it into the oropharynx. Alternatively, anti-fogging solutions can be applied to the mirrored side. With your nondominant hand, grasp the patient's tongue (after wrapping it with gauze to prevent it from slipping or being injured by the lower incisors) (Fig. 68-2) . Apply gentle traction to the tongue with your thumb and index finger while lifting the patient's upper lip with your middle finger. Slide the mirror into the oropharynx with the glass surface parallel to the tongue but not touching it. Place the back of the mirror against the uvula and soft palate, smoothly lifting until the larynx is visualized. While this should not induce gagging, try to make only slight changes in mirror position to inspect

the appropriate structures.

In patients who cannot tolerate this procedure without gagging, apply topical anesthetic to aid in the examination. Benzocaine (Hurricaine spray or Cetacaine gargle) or aerosolized tetracaine or lidocaine may be used. One or two quick sprays of benzocaine into the posterior oropharynx is sufficient. Prolonged or repeated spray use may result in methemoglobinemia. Reassure the patient beforehand that although this may make the throat feel as if is swelling or paralyzed, in actuality it is just the numbness that accounts for the sensation. The tendency to gag also can be minimized by having the patient concentrate on his or her breathing efforts and keep the eyes open, with vision fixed on an object in the distance.

Once the patient is anesthetized, repeat the above steps and position the mirror against the soft palate. Rotate the angle of the mirror and systematically inspect the base of the tongue, valleculae, epiglottis, pyriform recess, arytenoids, false and true vocal cords, and, if possible, the superior aspect of the trachea (Fig. 68-3) . Observe for masses, evidence of infection, asymmetry, or foreign bodies. Further evaluate the anterior structure of the larynx and function of the vocal cords by having the patient say "eeee" in a high-pitched voice. This should move the epiglottis away from blocking the view of the larynx and bring the true cords together at the midline.

**Laryngoscopy by Angled Telescopes**

A less frequently used method of examining the larynx in the emergency setting is by rigid angled laryngoscopy (e.g., LarynxVue II, Astralite Corp., Anaheim, Calif). This provides a clear and more continuous view of the larynx during respiration, but its use is limited in patients whose epiglottis blocks the view. The light source is powered by batteries or wall outlet. The degree of mirror angulation is fixed, but it may vary between instruments (usually 70° to 90°). At 70° of mirror angulation, the scope does not need to be placed as far posterior into the oropharynx to visualize the structures.

Position the patient as for indirect laryngoscopy and anesthetize the soft palate if necessary, as previously described. Gently grasp the tongue and slide the scope into the oropharynx. Stabilize the scope on the fingers that are holding the tongue, taking care not to touch the sensitive base of the tongue. Once the scope is touching the soft palate and is near proper position, look into the eyepiece and make final adjustments to bring the laryngeal structures into focus. Observe the anatomy and function as previously described in a systematic fashion.

**Flexible Fiberoptic Laryngoscopy**

Fiberoptic examination of the nasopharynx and larynx can be accomplished with either a flexible nasopharyngoscope or a bronchoscope. The nasopharyngoscope is thinner, shorter, and easier to manipulate (Fig. 68-4) . Fiberoptic visualization is especially useful in patients who are difficult to examine because of persistent gagging or unusual anatomy.  Attach the endoscope to its light source, attach the suction to its port (if available), and ensure that both are functioning properly before beginning. Prior to inserting the scope, the eyepiece is adjusted to the operator's visual acuity; it is helpful

to check the scope focus on newsprint or a small object at this time. Review of the scope directional controls is also recommended.

Examine the nares and choose the more patent one to enter. Anesthetize and vasoconstrict the naris with lidocaine and epinephrine (as described in Epistaxis, later in the chapter). Some clinicians also anesthetize the pharynx to minimize gagging. Warm the end of the scope in warm water to help prevent fogging. The patient should be seated, with the head placed against a headrest, in the "sniffing" position. Insert the tip of the lubricated scope just inside the naris. A series of soft nasal trumpets may be used to dilate the nasal cavity, allowing easier passage of the scope. The movement of the scope against the inside of the nasal passage may be irritating; this discomfort can be minimized by resting the fourth and fifth fingers on the bridge of the patient's nose while stabilizing and guiding the passage of the scope between the thumb and index finger (Fig. 68-5) .

While looking through the eyepiece, slowly advance the endoscope past the inferior turbinate into the nasopharynx or through the lumen of the trumpet. To clear fogging or mucus off the lens, have the patient swallow, wipe the lens against the pharyngeal mucosa, or use the suction. Once it is in the nasopharynx, direct the tip inferiorly, using the thumb control near the eyepiece. *Up-and-down movements of the scope may be accomplished with the thumb control, whereas rotating the scope about its axis and then applying the thumb control provides for lateral movement and visualization.* At this point, the base of the tongue and tonsils will come into view. Slide the scope further caudad to bring the larynx into focus. Once again, systematically view the anatomy and function during both respiration and phonation.

If the nasopharyngeal scope will not pass through either naris, pass it through the oropharynx. Properly anesthetize the oropharynx and avoid the posterior tongue to prevent gagging. Cut a 10-mL syringe (without the plunger) in half. Have the patient hold this in the mouth between the incisors. Pass the fragile endoscope through this tube to prevent accidental biting of the scope.

Complications include traumatic abrasions and bleeding anywhere along the path of the laryngoscope. In patients with head injury, there is always the rare risk of passing the scope intracranially if a basilar skull fracture exists, but use of a soft nasal trumpet significantly reduces that risk. The induction of laryngospasm and acute airway compromise is possible in patients with paraglottic infections.

## Peritonsillar Abscess

### Anatomy

Peritonsillar abscess, also known as *quinsy*, is most common during the second and third decades. It remains the most common head and neck abscess in adults. Currently, treatment of peritonsillar abscess includes tonsillectomy (immediate or delayed), intravenous (IV) antibiotics, incision and drainage, needle aspiration, or any combination

of the above.

The relative anatomy must be understood to treat peritonsillar abscesses (Fig. 68-6) . The palatine tonsils are located between the anterior and posterior pillars of the throat, bound in a capsule and covered by mucosa. The lateral wall of the tonsil is defined by the superior pharyngeal constrictor muscle. Of great importance is the *internal carotid artery,* which lies approximately 2.5 cm posterolateral to the tonsil.

**Pathophysiology and Presentation**

Peritonsillar abscess occurs in patients with inadequately treated tonsillitis and in recurrent tonsillitis. The abscess is usually unilateral and spreads to the peritonsillar space between the tonsillar bed, the capsule, and the superior constrictor muscle in the region of the anterior pillar. The abscess is most commonly initiated from the upper pole of the tonsil. However, it can also spread from the middle or the inferior poles. Complications may include pharyngeal obstruction or extension into the closely approximated neurovascular bundles and parapharyngeal space.

Inadequately treated tonsillitis occurs when a patient fails to follow the prescribed regimen or when the regimen is inadequate. The latter may occur as the result of an improperly chosen antibiotic or because of increasing antibiotic resistance. Anecdotal cases of failure after intramuscular long-acting penicillin also have been noted. Although Group A *Streptococcus* remains the leading cause of peritonsillar abscess, *Staphylococcus aureus*, *Haemophilus influenzae*, and mixed anaerobic infections are common. Recent literature reveals cases of tonsillitis due to beta-lactamase-producing organisms, which confers resistance to beta-lactam antibiotics. [2] Fine-needle aspiration of peritonsillar abscesses may allow identification of organisms and appropriate modification in antibiotic therapy, thus avoiding the need for tonsillectomy.

Patients with peritonsillar abscesses present with sore throat, odynophagia, low-grade fever, and a variable degree of trismus. The trismus develops secondary to pterygoid muscle irritation. The patient may also complain of ipsilateral otalgia or have dysarthria ("hot potato" voice). As the abscess expands, swallowing becomes increasingly painful and may lead to dehydration secondary to poor intake. Tender ipsilateral anterior cervical lymphadenopathy is frequently present. Be cautious of a fever >39.4 °C, as this has been associated with parapharyngeal extension and sepsis. [3]

Examination of the oropharynx may be difficult because of associated trismus. Have the patient sit with the head in the sniffing position. Encourage the patient to open the mouth as wide as possible, and depress the tongue to obtain a better view of the oropharynx. Use a head lamp or head mirror/light source to ensure adequate illumination. Digital palpation for a fluctuant site can be useful, but the patient may gag or bite the examiner by reflex. Generally, the same information can be obtained using a cotton-tipped applicator. Medial displacement of the tonsil and uvula are noted with a fluctuant mass involving the tonsillar pillar. The normally sharply delineated pillar-like structure will be obliterated by swelling. The tonsil will look edematous and erythematous, and it may be covered with a whitish exudate.

The differential diagnosis for this acute process includes unilateral tonsillitis, peritonsillar cellulitis, peritonsillar abscess, and, possibly, carotid artery aneurysm. Chronic conditions include leukemia, carcinoma, and parapharyngeal space tumor.

Differentiation of a peritonsillar abscess from peritonsillar cellulitis may be difficult, especially in the early stages of an abscess. The histories and time course for the two disease processes are quite similar. Trismus is uncommon in peritonsillar cellulitis. Needle aspiration will be diagnostic if purulent material is removed. However, a negative test does not rule out an abscess. The abscess may be located posteriorly, which is not accessible to the aspiration needle. Intraoral sonography has a sensitivity and specificity of 91% and 80%, respectively, in detecting peritonsillar abscesses [4] and may therefore augment diagnostic accuracy.

**Indications for Needle Aspiration**

Treatment of peritonsillar abscess has undergone significant change in the past 100 years and continues to do so at this writing. A myriad of opinions exists on the appropriate treatment method, although most agree that some form of drainage procedure should be performed. Treatment of an initial peritonsillar abscess commonly included an incision and drainage followed by an interval tonsillectomy to prevent recurrence. Emergent tonsillectomies had been traditionally avoided because of fear of septicemia and increased intraoperative bleeding. However, this procedure gained favor in the early 1970s, as these concerns were never substantiated. The emergent tonsillectomy removes the source as well as the abscess itself. Recently, treatment with needle aspiration and IV antibiotics in lieu of traditional incision and drainage has been performed with a recurrence rate from 3 to 23%. Some studies report an average cure rate of 80% with needle aspiration.

Points of discussion concerning needle aspiration include the following:

1. Aspiration may miss the peritonsillar abscess and therefore allow misdiagnosis as peritonsillar cellulitis. Up to

   12 to 24% of abscesses have been missed on initial aspiration. [5] [6] Therefore, some authors propose admission of patients with negative aspirations with the presumed diagnosis of peritonsillar cellulitis for IV antibiotics and observation to prevent further morbidity. [7]

2. Recurrence rates of 3 to 23% have been noted after needle aspiration. This recurrence rate matches that noted for young adults with incision and drainage at a rate of 5.9 to 22.7%. [10] Some suggest immediate (quinsy) tonsillectomy to prevent recurrences.
3. Repeat aspirations are often necessary, which causes further morbidity. Repeat aspirations were required in 6 to 10% of patients. [6]

Benefits of needle aspiration over incision and drainage include decreased pain and

trauma. Although most studies were performed with hospitalization and IV antibiotics, outpatient treatment with oral antibiotics has been successful. [7] [8] This approach adds the benefits of no hospital stay and decreased costs.

Treatment guidelines based on a review of the literature [11] suggest that (1) pediatric patients with peritonsillar abscess be admitted for IV antibiotics and observation with possible quinsy tonsillectomy; (2) adult patients with *recurrent* tonsillitis/peritonsillar abscess be treated with either quinsy tonsillectomy or interval tonsillectomy after initial treatment with incision and drainage or aspiration; and (3) adult patients with an initial episode be managed with either incision and drainage or needle aspiration. This applies especially to those with a low risk of recurrence, such as persons older than 40 years. These procedures can be done in combination with hospital admission and administration of IV antibiotics or as an outpatient with oral antibiotics. The approach depends on the patient's clinical status and medical history. Decisions about the treatment of a peritonsillar abscess in the ED should be made in consultation with an otolaryngologist.

**Needle Aspiration/Incision and Drainage**

The two procedures described here include needle aspiration and incision and drainage. They should be performed in consultation with an otolaryngologist. They should only be performed in the cooperative patient without severe trismus. With the carotid artery located 2.5 cm behind and lateral to the tonsil, there is minimal room for error, patient movement, or poor anesthesia.

Have the patient sit upright, with a support behind the head. This is best done as a 2-person procedure. An assistant can retract the cheek laterally to maximize visibility. A head lamp provides optimal lighting; a double tongue-blade setup aids visualization of the operative area (Fig. 68-7) . Topically anesthetize the area with Cetacaine spray, or 4 to 10% lidocaine. *Stay within the maximal allowed dosage* (see below and Chapter 31) . Determine the fluctuant area of the abscess using a cotton swab. Additionally anesthetize this area with local infiltration of 1 to 2 mL of 2% lidocaine with 1:100,000 epinephrine via a 27-ga needle. Infiltrate the lidocaine intramucosally for the best results. During the injection, be careful not to increase the abscess size by direct injection into the abscess cavity. If the trismus is so pronounced as to prevent adequate anesthesia administration, it will probably be too difficult to incise the abscess properly.

For *aspiration*, prepare a 20-ga needle on a 10- to 20-mL syringe. Smaller syringes limit the operator's view of the pharynx. Fashion a needle guard by cutting off the distal 0.5 cm of the plastic needle cover (or impale a stopper from a test tube with the aspirating needle). Securely attach this guard to the needle and syringe with tape to prevent inadvertent displacement (Fig. 68-8) . Ensure that the needle protrudes only 0.5 cm beyond the cover. This procedure will lessen the risk of entering any major vascular structure. Insert the needle into the most fluctuant (or prominent) area as previously determined, which is most commonly the superior pole of the tonsil. Note that the tonsil itself is not aspirated, because the abscess develops in the peritonsillar space surrounding the tonsil. *The needle is advanced only in the sagittal plane and is not directed laterally, where it may injure the carotid artery.* If the aspirate is positive,

remove as much purulent material as possible. If the aspirate is negative, attempt aspiration again in the middle pole of the tonsil approximately 1 cm caudal to the first aspiration. A third and final attempt should be performed at the inferior pole. It must be stressed that a negative aspirate does *NOT* rule out a peritonsillar abscess. Obtain culture and sensitivity on any aspirated fluid. Some clinicians advise a formal incision and drainage (see below) if frank pus is obtained, whereas others accept needle aspiration (with close follow-up) as the definitive *initial* treatment.

To *incise* a peritonsillar abscess, anesthetize the area as described earlier. Prepare a No. 11 or 15 scalpel blade by taping over all but the distal 0.5 cm of the blade to prevent deeper penetration (Fig. 68-9) . Incise the area of maximal fluctuance or where a preceding aspiration, if one was performed, located pus. Incise the mucosa in an area 1 cm long in a posterior to anterior direction. A stab incision with a No. 11 blade usually suffices. Warn the patient that the pus will flow posteriorly and the patient will have to expectorate this fluid. Expect bleeding, as this is a vascular area. Suction the incised area with a No. 9 or 10 Frazier suction tip or a tonsil suction tip to aid in removal of the purulent material. Place a closed Kelly clamp into the opening and gently open it to break up the loculations. Culture the abscess for both aerobic and anaerobic organisms. Allow the patient to rinse and gargle with a half hydrogen peroxide and half normal saline solution. Packing is *not* used in the drainage of this abscess. *Following aspiration or incision, it is prudent to observe the patient for about an hour to watch for complications (e.g., bleeding) and to ensure the ability to sustain oral fluids.*

Antibiotics are recommended to eradicate the offending organisms. Penicillin or cephalosporins are a reasonable first choice. Reasonable cure rates have been obtained with oral penicillin in modest doses (500 mg PO QID). Many clinicians prefer to administer an IV loading dose of penicillin (2 to 4 million units) or cefazolin (1 g) prior to releasing the patient. Any patient who appears to be having a toxic response, whose immune system is compromised, is unable to take oral antibiotics, or is dehydrated should be admitted for IV fluid hydration and antibiotics administration. Reevaluation of all patients treated with needle aspiration drainage should be performed in 12 to 24 hours to assess the need for repeat aspirations or formal incision and drainage. Warm saline gargles and mild opioid analgesics also are recommended with outpatient care. Patients treated with incision and drainage also benefit from a short-term follow-up visit, but are less likely to require intervention than patients treated with aspiration. All patients should immediately return for recurrence of symptoms, fevers, or continued bleeding from the incision.

### Complications

Aspiration or incision of a carotid artery or misdiagnosed carotid artery aneurysm may have devastating results. As discussed before, the misdiagnosis of a peritonsillar abscess as cellulitis will lead to further morbidity. A too-large or too-small incision may lead to poor healing or inability to completely evacuate the abscess, respectively.

# EAR

## Anatomy of the External Auditory Canal

The external auditory canal (EAC) extends from the tympanic membrane to the concha and measures approximately 2.5 cm in the adult. It is relatively short and straight in early infancy but begins to take on its adult S-shape and overall anterocaudal orientation beginning at age 2 years. Initially, the EAC is almost entirely cartilaginous, but by adulthood its medial two-thirds is comprised of bony support with an overlying thin, stratified, squamous epithelium. The lateral third has a less sensitive, thicker, hairy epithelium that produces cerumen and retains its cartilage as support. The arterial supply to the EAC originates from the external carotid artery via the posterior auricular, maxillary, and superficial temporal branches. The mandibular branch of the fifth cranial nerve ($V_3$ ) and the vagus nerve innervate the ear.

Other important anatomic considerations include the following: (1) Two natural narrowings of the EAC exist, which are important when considering foreign bodies. One is located at the junction of bone and cartilage and the other lies just lateral to the tympanic membrane. (2) A blind spot may occur in the tympanic sulcus (inferior and anterior to the tympanic membrane) due to the oblique orientation of the tympanic membrane. An examiner using a simple otoscope may not visualize a foreign body in this sulcus.

## Anesthesia of the Ear

### External Ear/Auricle

Indications for local anesthesia of the auricle include closure of extensive lacerations or other painful procedures, such as hematoma incisure and drainage. Four nerve branches supply the external ear, and knowledge of their anatomy is required to understand the location for anesthesia injection (Fig. 68-10 *A* and *B*). The *greater auricular nerve* (branch of the cervical plexus) innervates most of the posteromedial, posterolateral, and inferior auricle. A few branches of the *lesser occipital nerve* may contribute to this area. The *auricular branch of the vagus* supplies the concha and most of the area around the auditory meatus. The *auriculotemporal nerve* (from the mandibular branch of the trigeminal nerve) supplies the anterosuperior and anteromedial aspect of the auricle.

#### Procedure.

Fill a 10-mL syringe with either 1% lidocaine or 0.25% bupivacaine (both with epinephrine if a regional block is planned in an area *without* evidence of traumatized vascularity) and attach the syringe to a 25- or 27-ga needle (5 to 7 cm in length). One of several methods may be used to accomplish partial or complete anesthesia, depending on the area of concern. The greater auricular and lesser occipital nerve branches may be anesthetized by injecting between 3 and 4 mL of anesthetic in the posterior sulcus (Fig. 68-11 *A*). Insert the needle behind the inferior pole of the auricle and gradually aspirate and inject toward the superior pole, following the crescent-shaped contour of

the posterior auricle. Anteriorly, the auriculotemporal nerve may be anesthetized by placing 3 to 4 mL of anesthetic just superior and anterior to the cartilaginous tragus. Use the technique shown in Figures 68-12 and 68-13 to provide anesthesia of the auricular branch of the vagus to include more central areas of the auricle.

Another and possibly more effective option is the regional block shown in Figure 68-11 B. Insert the needle subcutaneously (SQ) at a point approximately 1 cm above the superior pole of the auricle and direct it to a point just anterior to the tragus. Be sure to inject the skin of the scalp while avoiding the auricular cartilage. Aspirate, then slowly withdraw the needle, injecting anesthetic until the needle is almost to the puncture site. Redirect the needle posterior and repeat the process while aiming at the skin just behind the mid-auricle. Remove the needle and perform the same procedure, but insert the needle just inferior to the insertion of the ear lobule and anesthetize in a superior direction. Again, block the auricular branch of the vagus as described in Figure 68-13 if additional anesthesia of the concha is required.

The addition of epinephrine to the anesthetic solution should be used with caution when placing regional blocks of the ear, especially if the blood supply has already been traumatically reduced. Do not include epinephrine when directly infiltrating wounds of the auricle, as restriction of blood flow through end arteries here may result in tissue necrosis. Other complications related to local anesthetics and regional blocks may be reviewed in Chapters 31 through 33.

**External Auditory Canal and Tympanic Membrane**

The EAC is innervated by the auricular branch of the vagus (inferiorly and posteriorly) and by the auriculotemporal nerve (superiorly, anteriorly, and inferiorly). The primary indication for local anesthesia of the auditory canal is for foreign body removal, including debridement of otitis externa or removal of significant cerumen impaction. Topical anesthetics are inadequate due to their poor absorption through the rather impermeable and keratinized epithelial surface of the EAC. Although effective, instilling local anesthetics in and around the auditory meatus is quite painful and is often difficult to perform in a struggling and uncooperative patient. Certain instances warrant adjunctive use of conscious sedation (see discussion under Foreign Body Removal).

It is quite difficult to obtain anesthesia of the tympanic membrane. The membrane is sensitive and can be stimulated during attempts at removing a foreign body from the ear canal. Topical anesthetics have limited value, but Moller and Grontved demonstrated that 10% aerosolized lidocaine (first sprayed into a syringe and shaken to evaporate the propellant) and 4% lidocaine suspension, when dripped into the ear canal, provided good anesthesia of the membrane. [12] However, it required 30 minutes for this anesthesia to take effect. Both solutions were alkaline and it was noted that the lidocaine hydrochloride, the form usually used for wound infiltration, is acidic and provided no anesthesia when applied topically.

**Procedure.**

Local anesthesia is performed with a 25- or 27-ga needle (3 to 5 cm in length) attached

to a syringe of 1% lidocaine with epinephrine (1:100,000). A 1:10 mixture of 8.4% sodium bicarbonate to lidocaine helps to reduce pain with injection in this sensitive area. Place a speculum just inside the auditory meatus and inject 0.3 to 0.5 mL of the anesthetic into the SQ tissue, stopping after a small bulge in the skin is raised. Inject in this manner in all four quadrants by moving the speculum after each injection (Fig. 68-12) . If additional anesthesia is necessary, 2 more small injections can be made. Inject the same amount slightly further into the canal, once along the anterior wall and again at the posterior wall at the bone-cartilage junction.

Another similar technique involves depositing the anesthetic just lateral or exterior to the external auditory meatus. Using the same size needle and type of anesthetic solution as just described, inject approximately 0.5 to 1.0 mL into each of 5 points around the auditory meatus and tragus (Fig. 68-13) .

## Examination

Several methods are available to examine the EAC and tympanic membrane. In all methods, the superior pinna should be grasped and pulled cephalad and posterior to straighten the slightly tortuous EAC. The most common manner of examination is with a *fiberoptic otoscope* (Fig. 68-14) . The clinician may insufflate the tympanic membrane as well as examine the EAC with the diagnostic head, while the operating head allows for instruments to be passed into the EAC and maneuvered more easily. A plastic or metal speculum may be placed in the auditory meatus for examination, using a head lamp or head mirror/light bulb as a light source.

Although this provides excellent illumination, magnifying loupes are generally needed for adequate visualization during procedures. The ideal setup for cerumen or foreign body removal consists of an operating microscope and a speculum. This provides binocular vision and frees the examiner's hands for instrumentation (unfortunately this equipment is seldom found outside of the otolaryngology clinic setting). The hand holding the otoscope is stabilized against the patient's temporal skull to prevent inadvertent canal injury due to unexpected patient movement.

## Cerumen Impaction

The apocrine and sebaceous glands lining the EAC excrete cerumen, which subsequently migrates out the auditory meatus. Cerumen repels water, has documented antimicrobial activity and forms a protective barrier against infection. Cerumen often becomes impacted, causing complaints of a "blocked" ear, hearing impairment, or dizziness. Symptomatic impaction is an indication for removal, although symptoms are rare until complete obstruction is present. More commonly in the emergency setting, cerumen obstructs visualization of the tympanic membrane and needs to be evacuated as a part of the evaluation of a febrile child or the patient complaining of ear pain.

### Cerumen Removal

Irrigation is an effective approach for cerumen removal and has the advantage of being painless and simple to perform. The patient does not have to remain completely still; thus, it is ideal for the pediatric population. Although usually more time-consuming than manual extraction, irrigation is an appropriate initial method to attempt and can be performed by technicians with guidance from the physician. One contraindication is known or suspected tympanic membrane perforations. Use irrigation judiciously in elderly and immunocompromised patients, as malignant otitis externa is frequently preceded by irrigation of the EAC. [13]

**Ceruminolytics.**

These products may soften obviously hardened or impacted cerumen. Although many products are available to assist in this, a 5 or 10% solution of sodium bicarbonate much more quickly and efficiently disintegrates cerumen compared with commercially prepared ceruminolytics and other products. Cerumenex, Cerumol, Auralgan, Buro-Sol, alcohol, and oils were all tested and took more than 18 hours to disintegrate cerumen vs 90 minutes for the sodium bicarbonate solutions. [14] Place the patient in the supine position with the affected ear up and instill the solution at least 15 minutes prior to attempts at removal. Instillation can be repeated between attempts at manual extraction or irrigation.

**Irrigation.**

This procedure is best achieved by having the patient sit upright and hold an emesis or ear irrigation basin flush against the skin just below the ear lobule (Fig. 68-15) . Insert the irrigation tip into the EAC only as far as the cartilage-bone junction, and direct the stream of water around the cerumen to prevent further impaction. Water should be near body temperature to prevent caloric stimulation. Multiple attempts may be necessary, and intermittent attempts at manual removal of loosened cerumen may help hasten the process. During the irrigation, the operator or an assistant should apply traction to the pinna to straighten the canal for more efficient irrigation.

Irrigation techniques using manual pressure to discharge the water include metal ear syringes and bulb syringes. Improvisational irrigators can be assembled with equipment found in the ED. Attach a 20- or 30-mL syringe to a 19-ga or larger butterfly device, cutting off the needle and wings and leaving the resultant tubing for irrigation. A plastic or Teflon IV catheter (16- or 18-ga with the needle removed) can similarly be affixed to a syringe.

Although most commonly found in an otolaryngologic clinic, automated pressure devices may also be available. The *DeVilbiss irrigator* (Fig. 68-16) uses a pressurized air source to propel the irrigating solution into the ear. *Oral jet irrigators* (Water Pik) are another accepted method. Although the instances have been rare, tympanic membrane rupture has been documented with these apparatuses. [15] Therefore, use the lowest-power setting and guide the stream of the water against the EAC wall, not

directly toward the tympanic membrane.

After irrigation of the EAC, application of several drops of isopropanol in the EAC will facilitate evaporation of residual moisture. The tympanic membrane must be intact if isopropanol is to be used. Further, topical Cortisporin suspension drops may be soothing after prolonged irrigation.

Although more common with jet irrigators, complications may occur with any method of ear irrigation. These include otitis externa, tympanic membrane perforation, or middle ear injury from a preexisting defect in the tympanic membrane. Irrigation should be stopped and the tympanic membrane examined in any patient experiencing sudden pain, tinnitus, hearing loss, nausea, or vertigo. If the membrane is ruptured, prophylactic antibiotics for otitis media should be given along with a referral to an otolaryngologist for an audiogram and possible tympanic membrane grafting.

**Manual instrumentation.**

This procedure is more advantageous as it is usually quicker, and the examiner may more easily remove hardened or larger concretions of cerumen under direct visualization. Either the diagnostic or operating head of the fiberoptic otoscope or a speculum may be placed in the auditory meatus to serve as a protective port through which instruments are passed and manipulated (Fig. 68-17) (Figure Not Available) . An operating microscope works best in this situation but, again, is usually not available. To prevent startling or agitating an already anxious patient, allow the patient to experience the sensation of an instrument in the canal by first placing the instrument softly against the ear canal wall.

Instruments used for cerumen removal include *flexible plastic or wire loops, right-angle hooks, suction-tip catheters, or plastic scoops* (Fig. 68-18) . The spoon-like instruments and irrigation are both more effective in removing softer cerumen. Firm cerumen ordinarily is more easily withdrawn with loops or right-angle hooks. Gently tease the cerumen off of the canal wall using loops and then pass hooks or loops around the cerumen and withdraw the cerumen slowly (Fig. 68-19) . Care should be taken to keep both hands in contact with the patient's head, as any sudden movement may cause trauma to the canal or the tympanic membrane. Complications most commonly occur when inadvertent contact is made with the thin, friable skin of the bony canal. Trauma may cause EAC laceration, hematomas, otitis externa, or tympanic membrane perforations.

## Otitis Externa

Otitis externa, or "swimmer's ear," is an infection of the auditory canal usually due to *Pseudomonas*, *Staphylococcus*, and mixed gram-positive and gram-negative organisms. The disease process involves a continuum of gradually worsening inflammatory changes. The patient may present with symptoms ranging from slight itching and discomfort to severe pain, purulent discharge, or systemic toxicity. Pain with manipulation of the pinna is the hallmark for otitis externa. Otoscopy of the external auditory canal may initially reveal minimal debris and erythema, but as the infection

progresses more edema, exudate, erythema, and possibly even a surrounding cellulitis may become apparent. In severe stages the edema may obstruct the canal, preventing instillation of ear drops.

*Aspergillus* is the most common cause of a fungal otitis externa, which presents as a furry lining of the canal. Herpes zoster affecting the geniculate ganglion may appear as grouped vesicles on an erythematous base within the canal. This condition, known as Ramsay Hunt syndrome, is associated with facial nerve palsies, hearing loss, and other cranial nerve impairment. Diabetics and other immunocompromised patients are susceptible to *malignant otitis externa*, a life-threatening form of otitis externa caused by *Pseudomonas*. Deep tissue necrosis, osteomyelitis, intracranial extension, systemic toxicity, and failure of initial outpatient therapy help differentiate malignant otitis externa from otitis externa.

**Canal Debridement/Wick Placement**

In the early stages of inflammation, remove debris using suctioning, a curette, or a cotton-tipped applicator (Fig. 68-20) . The cotton is soaked with Cortisporin or acetic acid solution. For more advanced cases presenting with significant exudate and edema, this procedure remains necessary but is intensely painful. The authors recommend using a local block of the auditory canal (see Fig. 68-13) as long as the cellulitis has not extended out to the tragus or concha. Administer parenteral analgesics if additional pain control is required.

Ear wicks may be used when edema, debris, and exudate are marked enough to impede antibiotic drops from contacting the canal skin. After debridement, use one of several methods to accomplish this. A 0.25-in. strip of Nu-Gauze dressing is covered with an antibiotic and steroid cream (Cortisporin otic cream) and may be packed into the external acoustic canal in a fashion similar to the technique used for anterior nasal packing. Using an otoscope and alligator forceps, place the leading edge of the gauze deeply in the canal until it is fully packed. Withdraw the otoscope and finish by packing the lateral aspect of the canal as well.

Another choice is to place commercially available ear wicks, such as the Pope Merocel ear wick. Place this wick into an edematous canal and apply antibiotic/hydrocortisone drops onto it. The wick swells and helps to reduce edema by the antimicrobial and anti-inflammatory effects of the solution and through pressure exerted against the walls as it expands. Leave wicks in place until the patient is followed up in 24 to 48 hours for removal and further debridement.

**Antibiotic Therapy and Follow-up**

Antibiotic ear drops most frequently consist of some combination of neomycin and polymyxin, but an acetic acid solution is another acceptable first-line therapy. Ear drops are instilled as 2 to 4 drops 4 times daily for 7 to 10 days. Hydrocortisone may be added to either the antibiotics (Cortisporin suspension or solution) or the acetic acid (VoSol HC). Cortisporin otic *solution* (clear appearing) is harmful to the middle ear if it passes through the tympanic membrane. At times it is difficult to distinguish between a ruptured

tympanic membrane secondary to otitis media and severe otitis externa in a child. Therefore, the cloudy appearing Cortisporin *suspension* is recommended in any case of suspected or known tympanic membrane perforation.

Inform patients to avoid water for the full course of treatment and to apply their ear drops immediately if water does contact the ear canal. Follow up severe cases in 24 to 36 hours and repeat debridement if needed. Generously administer oral opioids for the first 24 to 48 hours, as this condition can be quite painful.

Systemic symptoms or localized cellulitis requires administration of broad-spectrum antibiotics. A cautious approach to treating the mild to moderate case of otitis externa in the immunocompromised patient without toxic reaction is to add oral ciprofloxacin for greater coverage of *Pseudomonas*. Promptly admit suspected cases of malignant otitis externa and begin an IV anti-pseudomonal antibiotic. In addition, immediately consult an otolaryngologist specialist for possible surgical debridement. Patients with Ramsay Hunt syndrome should be admitted for administration of IV acyclovir. Treat otomycosis by swabbing the canal with a cotton-tipped applicator saturated with an antifungal solution (such as clotrimazole or boric acid/alcohol) in an effort to remove debris. Repeat this if necessary on follow-up in 3 to 7 days. Do *not* administer corticosteroids in cases of known fungal otitis externa.

**Foreign Bodies of the Ear Canal**

Despite its small size, the EAC may play host to a number of types of foreign bodies. [16] [17] Living insects account for the majority of foreign bodies found in adults. Children frequently place food (e.g., peas, beans), organic matter (e.g., grass, leaves, flowers), and inorganic objects (e.g., beads, rocks, dirt) into the ear canals during play, and they often fail to admit this to parents. Button batteries may cause significant tissue destruction in a matter of hours, and it is vital to immediately obtain otolaryngologic consultation for removal if the button battery is not easily extracted. Symptoms of foreign body retention are usually ear pain, fullness, or impaired hearing in the adult, whereas the pediatric patient may not present until an associated otitis externa with a purulent discharge has developed. Tinnitus, vertigo, significant hearing loss, or bleeding from behind the object should raise a high suspicion for an associated tympanic membrane rupture.

As previously described, the anatomy of the external auditory canal predisposes to the entrapment of foreign bodies in either a lateral or a deeper position. Removal of more medial objects can be much more painful and anesthesia is usually required. Even the most cooperative patient may become difficult after feeling pain during manipulation of the ear canal. It is probably impossible to adequately immobilize the head of an uncooperative awake child to delicately extract a foreign body. Some authorities claim local anesthesia makes extracting foreign bodies even more difficult because of soft tissue distortion, although swelling should be minimal if proper amounts of anesthetic are used. A more realistic concern is that if the foreign body is deeply or firmly embedded, the patient should be referred early for removal under an operating microscope, before canal trauma and swelling mandate admission for removal under general anesthesia. Conscious sedation (preferably an analgesic-sedative combination

or dissociative anesthetic) can aid in the removal of foreign bodies in the distraught child by preventing further struggling and potential canal trauma. The care provider must weigh the inherent risks of conscious sedation against those of general anesthesia and the cost of hospital admission.

Before initiating removal, the clinician should set realistic limits on the number of attempts to be made. Even the best clinician can become too aggressive as frustration builds with failed attempts to extract the object. Early consultation with an otolaryngologist should not be considered a failure in cases of difficult foreign bodies. Indeed, with the proper equipment and experience, most objects can be atraumatically removed.

**Procedures**

Most approaches to foreign body removal are anecdotal and are found in the literature as case reports or case series rather than as prospective clinical trials. The clinician should be familiar with several techniques, as the most appropriate choice can vary depending on depth of impaction or the size, shape, and consistency of the object. *Irrigation* is the least invasive option and the techniques and related complications are explained in detail earlier in this chapter (see Cerumen Removal). Deeply embedded vegetable matter (such as beans, peas, or seeds) should not be irrigated, as swelling may occur and make extraction more difficult. Irrigation works particularly well with small rocks, dirt, or sand that lies deep in the canal next to the tympanic membrane.

**Suction-tip catheters.**

These devices can also be used, but the patient must be informed of the impending noise to prevent sudden movements from a startle reflex. Place either the blunt or soft plastic tip against the object and slowly withdraw. If using a suction instrument with a thumb-controlled release valve (as with the Frazier suction [Fig. 68-18]) , remember to cover the port to activate the suction.

**Manual instrumentation.**

This approach may be attempted with a variety of instruments (Fig. 68-21) . Illumination and magnification are accomplished by either the diagnostic or operating head of a fiberoptic otoscope. An assistant should hold the pinna back and out so that the examiner may hold the otoscope with one hand and manipulate the instrument with the other. A speculum and either a head lamp or head mirror/light source can also provide illumination, but magnifying loupes are usually required for adequate visualization. Use small alligator forceps to remove objects with edges that can be grasped, but avoid trying to encircle an impacted round foreign body, as this may cause trauma to the canal wall. A small right-angle hook is another choice. Place the tip past the object, rotate it 90°, and then pull the object from the canal. Fine-tissue or Adson forceps, curettes, and skin hooks are other instruments that are used occasionally.

**Small Fogarty catheters.**

Small Fogarty catheters (biliary or vascular) may be used in a manner similar to that described later in the chapter for nasal foreign body removal. [18] Attach the catheter tip to a 2-mL syringe and gradually inflate the balloon once the tip is past the object; then drag the foreign body out along with the balloon. Immediately deflate the balloon if sudden pain occurs, because tympanic membrane rupture is a potential complication.

**Cyanoacrylate (Superglue).**

This compound may be used in small amounts applied to the tip of a thin paintbrush, a straightened paper clip, or the blunt end of a wooden cotton-tipped applicator. [19] Place the tip against the object, allow it to dry, then carefully withdraw the foreign body. Minor complications are possible if the tip dries against the canal wall (abrasion, excoriation) or if the glue spills or drips onto the wall (creating a new foreign body).

**Dissolution of Styrofoam.**

*Styrofoam impaction* in the ear canal can be problematic. Application of acetone in a very small amount (0.1 mL) was reported to effectively dissolve deep-seated Styrofoam in a child's ear canal. [20] The ear was irrigated immediately after carefully dripping the acetone directly onto the Styrofoam, and no significant complications of the canal or tympanic membrane were noted. The potential ototoxic effects of acetone in the middle ear have not been studied; therefore, do not perform this technique if any suggestion or history of tympanic membrane perforation exists.

**Removal of insects.**

Cockroaches are the most commonly found *live insect* as an intra-aural foreign body; treatment consists of instillation of various substances into the ear canal to first immobilize or kill the bug. This helps retrieval by allowing for a stationary target and also halts the disturbing and painful movement of the insect. Controversy exists about which agent most effectively accomplishes this. Mineral oil has traditionally been used, although lidocaine has been reported to paralyze roaches and to allow for easier extraction than the more viscous mineral oil. An in vitro comparative study showed that immersion in mineral oil and 2 or 4% lidocaine solution killed roaches in <60 seconds (27 and 41 seconds, respectively). [21] The roaches struggled less in the viscous oil than in the lidocaine, which did not appear to cause paralysis. Other substances (Auralgan, isopropanol, water, succinylcholine, hydrogen peroxide) were shown to be ineffective in killing the roaches in a reasonable amount of time. Once disabled, insects may be removed with mechanical extraction as previously described, and pieces can be suctioned out if fragmentation occurs. Mineral oil is safe, but lidocaine has been reported to cause vertigo and middle ear symptoms in patients with tympanic membrane perforations. [22]

**Follow-up/Complications**

Hearing should be evaluated before and after foreign body removal, especially in patients with suspected tympanic membrane or middle ear injuries. Examine both the opposite ear and nose of children to search for the rare but possible second foreign body. Minor lacerations or excoriations of the canal usually heal quickly with or without antibiotic ear drops, so long as the canal is kept clean and dry. Document preexisting canal trauma or suspected tympanic membrane rupture before attempts at removal; otherwise, this may falsely be attributed to iatrogenic causes at a later date. Most patients with foreign bodies that cannot be removed in the emergency setting can be safely referred to an otolaryngologist the next day. Exceptions that require immediate consultation include severe pain, suspected tympanic membrane rupture, embedded button batteries, or a concomitant canal infection.

## Auricular Hematoma

Auricular hematomas occur after a shearing force to the ear, most commonly in wrestlers. A subperichondrial hematoma forms, separating the perichondrium from the cartilage. Recurrent or untreated injuries allow the development of new cartilage, which subsequently deforms the auricle (cauliflower ear).

**Procedure**

The treatment of an auricular hematoma involves complete evacuation of the subperichondrial hematoma and reapproximation of the perichondrium to the cartilage.

**Needle aspiration.**

Aspiration of auricular hematomas is performed by perforating the hematoma with a 20-ga needle (Fig. 68-22) . The hematoma is "milked" between the thumb and forefinger until the entire hematoma is evacuated. A pressure dressing is subsequently applied. Frequently reexamine the ear for reaccumulation of the hematoma. Reaccumulation of blood requires reaspiration. Because of the possibility of inadequate evacuation and recurrent hematomas, the authors do not recommend needle aspiration.

**Incision.**

The auricular hematoma, if less than 7 to 10 days old, may be incised along the natural skin folds. Anesthetize the pinna using local infiltration of 1% lidocaine (without epinephrine) or by an auricular block (described earlier). Incise the skin with a No. 15 blade at the edge of the hematoma, following the curvature of the pinna (Fig. 68-23) (Figure Not Available) . Gently peel the skin and perichondrium off the hematoma and underlying cartilage. Completely evacuate the hematoma and irrigate the remaining pocket with normal saline.

**Aftercare.**

After removal of the hematoma, apply antibiotic ointment and reapproximate the perichondrium to the cartilage via a pressure dressing. A compression dressing may be applied to the ear as described in Figure 68-24 . An alternative technique is to suture dental rolls over the area (see Fig. 68-23) (Figure Not Available) . [23] To accomplish this, pass 4-0 nylon suture through the entire thickness of the ear over the hematoma. Wrap the suture around a dental roll on the posterior aspect of the ear and then pass the needle back through the pinna. Wrap and tie the suture around a second dental roll on the anterior aspect of the pinna. A second suture may be placed to secure a third dental roll. The dressing should firmly re-approximate the perichondrium to the cartilage without vasculature compromise. Remove the dressing in 1 week.

Prescribe anti-staphylococcal antibiotics and instruct the patient to inspect the wound frequently for evidence of vascular compromise and/or infection. Reevaluate the wound in 24 hours for recurrence of the hematoma. Infection is treated with bandage removal, surgical drainage, and IV antibiotics. Refer patients with auricular hematomas of >7 days' duration to a surgeon, as the new perichondrial growth must be debrided to prevent auricular deformation.

# NOSE

## Anatomy

The nose consists of the vestibule, nasal septum, lateral wall, and the nasopharynx. The vestibule is the anteriormost portion of the nares, which is composed of skin and contains the hair follicles. The nasal septum is the midline structure, which is composed of cartilage anteriorly and of bone posteriorly. The lateral wall of the nose contains the superior, middle, and inferior turbinates as well as the auditory tube opening.

Three major arteries supply the nose and conjoin via anastomoses. The sphenopalatine artery emerges from the sphenopalatine foramen, which is located at the posterior aspect of the middle turbinate (Fig. 68-25) (Figure Not Available) . This is the most common source of posterior epistaxis. This artery supplies the turbinates laterally and the posterior septum. The anterior and posterior ethmoidal arteries branch off the ophthalmic artery and penetrate the cribriform plate to supply the superior nasal mucosa. The superior labial branch of the facial artery completes the triad, supplying the nasal septum and vestibule. The watershed area on the anterior septum, also known as Kiesselbach's plexus, is the most common source of anterior epistaxis (Fig. 68-26) (Figure Not Available) .

## Anesthesia

Apply local anesthetic and vasoconstrictors on cotton swabs. Placement of these swabs is illustrated in Figure 68-27 (Figure Not Available) . If a larger area of anesthetic is required, use cotton pledgets. Figure 68-28 explains the procedure of making pledgets.

Soak each pledget in anesthetic or vasoconstricting substance and then squeeze the excess fluid out of the pledget. Be aware of the total amount of drug being administered and stay within recommendations for the maximum safe dosage. Place each pledget horizontally on the floor of the nasal cavity, stacking the next pledget on top. Three pledgets are usually required to pack the nasal cavity. These can be replaced with new pledgets in 5 minutes if the desired anesthetic effect is not achieved. Hurricaine spray may also be used as a topical anesthetic. Remind the patient that any excess anesthetic may numb the throat but will not inhibit swallowing.

As with all drugs, review the maximum dose allowed before administering the anesthetic (see Chapter 31) . Always use the smallest amount of anesthetic required to obtain adequate analgesia. The maximum dose of lidocaine without epinephrine is 3-5 mg/kg up to a dose of 300 mg. The maximum dose of lidocaine with epinephrine is 5-7 mg/kg up to a dose of 400 mg. The maximum dose for cocaine is 3 mg/kg for mucosal application up to a dose of 200 mg. Remember that these are guidelines and that any patient can have a severe reaction with less than the total dose. For a 60-kg person, the maximum total dose of lidocaine would equal 300 mg (60 kg × 5 mg/kg = 300 mg). A 1% solution (10 mg/mL) would allow for a total of 30 mL to be administered (30 mL × 10 mg/mL = 300 mg). A 2% solution (20 mg/mL) would allow for only 15 mL to be administered (15 mL × 20 mg/mL = 300 mg). Cocaine is packaged in a 4% solution (40 mg/mL), allowing only 5 mL for a mucosal application to a 70-kg patient (3 mg/kg × 70 kg = 210 mg; 5 mL × 40 mg/mL = 200 mg, which is the maximum total dose). Always be familiar with the side effects and potential complications of the drugs being administered. Cocaine can induce tachycardia, hypertension, irregular respirations, seizures, and hyperpyrexia (especially in children). Lidocaine will manifest its antiarrhythmic and membrane stabilization effects.

## Examination

Examination of the nares is relatively straightforward. When using a nasal speculum, insert it into the naris with the handle parallel to the floor and slowly open the blades in the superior-to-inferior direction. Stabilize your hand on the patient's nose to prevent damage to the mucosa due to unexpected movement (Fig. 68-29) (Figure Not Available) . When attempting to visualize the nasal passageway, remember to have the patient keep the floor of the nose parallel to the ground. Tilting of the head only allows for a view of the anterosuperior area (Fig. 68-30) . A nasopharyngoscope may be used to view the nasal passageway as well, and its use is described in the previous section on examination of the pharynx.

## Epistaxis

Nasal hemorrhage commonly presents to the ED. Identification of the source of bleeding and subsequent control are paramount to the treatment of epistaxis. Although this can be frightening to both physician and patient, a systematic approach with the proper equipment will lessen the anxiety of the situation. The purpose of the procedure is to tamponade the bleeding. If the source is anterior, this may be the final treatment. For posterior bleeds, these are generally temporizing maneuvers until a definitive hemostatic procedure can be completed by a consultant. The procedures can be

performed in the ED with proper lighting and the equipment listed below. Controlling epistaxis may be a time-consuming process without the proper equipment or the cooperation of the patient.

In preparation for any procedure to treat epistaxis, evaluate the patient's hemodynamic status by assessing vital signs and orthostatic symptoms and quantifying blood loss. Also determine if the patient has any underlying medical problem, such as angina or chronic obstructive lung disease, which may be exacerbated due to hypovolemia or anemia. If the patient is symptomatic in any of these areas, or if the blood loss is deemed significant, start a large-bore IV line and administer fluid boluses. Obtain a complete blood count and consider a type and screen. Coagulation studies should be undertaken in patients taking anticoagulant therapy, those with underlying hematologic abnormalities, or those with recurrent epistaxis.

Many patients with epistaxis are hypertensive as well. No direct correlation has been proven between hypertension and epistaxis. Most authors now consider hypertension to be a stress response instead of an inciting event. [24] Therefore, hypertension does not require treatment until the bleeding is controlled and the anxiety of the situation has resolved. However, any patient exhibiting other signs of a hypertensive emergency needs immediate treatment in addition to control of the epistaxis.

**Indications/Contraindications**

Any continuing episode of epistaxis can be treated with the following techniques. Massive facial trauma with the possibility of a basilar skull fracture would preclude the use of a transnasal balloon, as it may travel into the skull cavity.

**Equipment**

- Chair with headrest or gurney with inclinable back

- Headlight with light source, head mirror

- Wall suction with multiple suction catheters

- Gloves, mask, and gown for physician

- Gown or drapes for patient

- Topical anesthetic

- Topical vasoconstrictor

- Nasal speculum

- Tongue depressors

- Small red rubber catheters

Bayonet forceps

Scissors

Kidney basin

Gauze (4 × 4 in., 2 × 2 in.)

Dental rolls or cotton

No. 2 surgical silk ties

1.2-cm wide Vaseline gauze or 0.5-in. wide Nu-Gauze packing

Antibiotic ointment

Silver nitrate sticks or electrocautery

Pediatric Foley catheters (12 Fr)

Rhino Rocket, nasal tampons

Dual balloon pack

**Epistaxis Examination**

As most patients are frightened by continued epistaxis, reassure the patient that you will control the bleeding. Drape the patient with a gown to protect his or her clothing from the bleeding. Have the patient hold an emesis basin to collect any continued bleeding and as a precaution to emesis of swallowed blood. Patients with epistaxis can be quite anxious, and manipulation of the nose can be quite painful. Therefore, sedation/analgesia with appropriate physiologic monitoring should be considered when extensive manipulation is anticipated.

Have the patient sit upright in the sniffing position with the neck flexed and head extended. The base of the nose should remain parallel with the floor. Position yourself in front of the patient, level with the patient's nose after putting on a face shield, gown, and protective gloves. Allow the patient to clear the nose of any blood clots by forceful blowing or, alternatively, you may suction the passageway until it is free of clots. If the bleeding is minimal, attempt to identify the bleeding source. If the bleeding is too profuse for visualization, administer a topical anesthetic and vasoconstrictor. A 4% cocaine solution would be ideal, but it is not commonly stocked in EDs; however, 2 to 4% lidocaine with epinephrine (or lidocaine supplemented by Neo-Synephrine) can be used.

Insert the nasal speculum into the naris and use the suction catheter to evacuate any blood. Do not hesitate to reapply anesthetic or vasoconstrictor if the desired results have not been achieved. As most cases of anterior epistaxis occur in Kiesselbach's plexus, inspect this area closely for areas of bleeding, ulceration, or erosion. If the

patient presents with a history of epistaxis but is no longer bleeding, gentle stroking of the anterior nasal mucosa with a cotton swab or having the patient forcefully blow the nose may reveal the bleeding source. If no source is found and the bleeding has ceased, the authors suggest packing the nose only if the epistaxis is recurrent. If no anterior source is found and bleeding continues down the posterior pharynx, assume a posterior source and pack the nose with an anterior and posterior pack.

**Cautery**

After identifying an anterior source of bleeding, there are several options to obtain hemostasis (Fig. 68-31) . *Silver nitrate sticks* may cauterize the area. Remember that silver nitrate sticks will not cauterize an actively bleeding source; therefore, hemostasis must be achieved first. Start by cauterizing above the bleeding source first to avoid the flow of blood. Cauterize around the bleeding source in a circular fashion. This technique should provide enough hemostasis to allow direct cautery of the source. Wipe away any excess silver nitrate to prevent inadvertent cautery of other areas of the nose. If this restarts the bleeding, the initial cautery was insufficient. *Electrocautery* will work in the same manner, but the cauterization will penetrate more quickly than the silver nitrate sticks. Care must be taken to prevent inadvertent septal perforation due to overaggressive or repeated cautery. Cautery works well for a small circumscribed area of bleeding.

If this is the initial bleed and hemostasis is achieved, no packing is necessary. If this is a recurrent bleed within 72 hours of another, or if cautery does not provide hemostasis, the anterior cavity should be packed. If hemostasis is accomplished, apply Vaseline or antibiotic ointment to the area to prevent desiccation. Do not administer aspirin or nonsteroidal anti-inflammatory drugs for 4 days after epistaxis. If bleeding recurs at home, instruct the patient to pinch the nostrils closed for 20 minutes. Instruct the patient to return to the ED if this maneuver is unsuccessful or the bleeding is profuse.

**Anterior Nasal Packing**

*Anterior gauze packing* is placed to complete hemostasis, prevent desiccation, and protect the area from trauma. However, improperly placed packing may further abrade the area, dislodge prematurely, or migrate into the posterior pharynx. Anterior packing must be placed with adequate analgesia, proper visualization, and deliberate movements. The packing is applied in an "accordion" fashion so that each layer extends the entire length of the nasal cavity.

Coating the packing with antibiotic ointment aids in its placement and helps prevent toxic shock syndrome secondary to nasal packing. Proper placement of the speculum allows visualization of the floor of the nasal cavity. Lay a strip of 1.2-cm wide (0.25- or 0.5-in. Nu-Gauze) gauze across the nasal floor, with the starting end of the gauze at the naris (Fig. 68-32) . Replace the speculum over the layer of gauze, gently packing it to the floor of the nose. Measure the gauze to twice the length of the nasal cavity. Grasp the gauze at the midpoint and insert this point to the posterior aspect of the nasal cavity. This allows placement of the gauze without movement of the underlying layer. Continue

this pattern, replacing the speculum after each layer until the cavity is filled.

An alternative to anterior nasal packing is the *nasal tampon* (Merocel) or the Epistat II nasal catheter (Xomed Surgical Products, Jacksonville, Fla) (Fig. 68-33 *A* and *B*). Place these products into the anterior nasal cavity along the floor of the nose. Contact with moisture will cause them to swell. It is sometimes necessary to place two tampons side by side before inserting them, to fill the nasal cavity. Any packing in the anterior nasal cavity may obstruct drainage of the paranasal sinuses or block the nasolacrimal ducts. In addition, because it is a foreign body, it will stimulate mucus production and act as an impetus for infection. Antibiotics should be prescribed with any packing left in the nose after emergency treatment because of the risk of toxic shock syndrome.

**Posterior Nasal Gauze Packing**

The patient has posterior epistaxis if no source of bleeding is found anteriorly and the patient continues to hemorrhage down the posterior pharynx. Posterior epistaxis may respond to topical vasoconstrictors. However, anterior nasal packing will not provide hemostasis, as it will not cover the source of bleeding. A posterior pack directly compresses the sphenopalatine artery and prevents the passage of blood or anterior packing into the nasopharynx.

The *posterior nasal gauze pack* is the classic method of treating posterior epistaxis. However, because balloon devices are easier to use and are less distressing to the patient, posterior nasal packing is less commonly used. To place a posterior nasal gauze pack, anesthetize the patient's nares and posterior pharynx with topical anesthetic. Prepare a roll of gauze with two No. 2 silk ties secured around the middle and extending in opposite directions. One set of ends will be used to place the posterior pack and the second will remain extruding from the oral cavity to remove the pack. Place a No. 10 red rubber catheter through the bleeding nostril (Fig. 68-34) . When it is seen in the posterior pharynx, grasp it with forceps and guide it out of the mouth. Attach it to one set of ends of the silk ties secured to the gauze pack. Retract the red rubber catheter, thus carrying the No. 2 silk tie through the nasopharynx and out of the nose. Grasp the suture and pull the pack into the nasopharynx. Guide the pack swiftly into the oral cavity and nasopharynx with the other hand. Attach the silk tie that remains in the oropharynx to the patient's cheek to aid in removal or rescue of the posterior pack. Use the silk ties exiting the nostril to maintain the position of the posterior pack. Pack the anterior passage as described for anterior epistaxis. Secure the silk ties over a gauze pad or dental roll. Patients with this type of posterior pack are admitted to the hospital.

**Inflatable Balloon Packs**

Inflatable balloons come in two varieties. The *Foley catheter* is often used as a posterior pack because of its availability, ease of use, and successful tamponading effect. [25] Insert a 12 Fr Foley catheter through the bleeding naris into the posterior pharynx (Fig. 68-35) . Inflate the balloon half way with about 5 to 7 mL of normal saline or water. Slowly pull the Foley into the posterior nasopharynx and secure it against the posterior aspect of the middle turbinate. Finish inflating the balloon with another 5 to 7 mL of normal saline or water. If there is pain or inferior displacement of the soft palate, deflate

the balloon until the pain resolves. Complete inflation of the balloon before proper placement causes the balloon to remain too posterior in the nasopharynx, thus failing to achieve hemostasis. [26] Place a short section of plastic tubing over the catheter and secure it with a nasogastric tube clamp or umbilical clamp. Be careful not to exert undue pressure on the nasal alar, as this may cause necrosis.

The second type of inflatable balloon pack is the pre-made *dual balloon tamponading system*. Goitschach Nasostat (Sparta Surgical Corp, Hayward, Calif) and Xomed Epistat (Xomed Inc, Jacksonville, Fla) are examples of some available devices. The *dual balloon pack* has a posterior balloon that inflates with about 10 mL of air and an anterior balloon that inflates with about 30 mL of air (Fig. 68-36) (Figure Not Available) . Each device may vary slightly (Fig. 68-37) . After appropriately anesthetizing the naris, place the lubricated pack along the floor of the affected naris as far back as possible. Inflate the posterior balloon about half way with air and then pull the balloon into place up against the posterior aspect of the middle turbinate. Complete the inflation of the posterior balloon with air. If the patient complains of pain or if the posterior soft palate deviates downward, deflate the balloon until the symptoms are relieved. Maintain the position of the balloon and inflate the anterior balloon with up to 30 mL of air. Again, halt the inflation if the patient experiences increasing pain or deviation of the nasal septum. Some authors suggest packing the opposite naris to prevent this lateral deviation. A small piece of gauze is placed between the nose and external catheter hub to decrease skin irritation. Generally, healthy patients given this type of pack are treated as outpatients, and the device is removed in 2 to 3 days.

**Complications**

Care of the posterior nasal packing is of particular concern. Posterior nasal packing is uncomfortable and often painful. Complications associated with posterior packing include infection, dysphagia, hypoventilation, tissue necrosis, and dislodgment. Therefore, any patient with a posterior pack should be admitted to the hospital for sedation and monitoring. Rebleeding may also be seen with early pack removal; one series found pack removal within 48 hours to increase the risk of rebleeding. [27]

*Infection* risk with posterior packing includes toxic shock syndrome, nasopharyngitis, and sinusitis. The packing blocks the sinus ostia, preventing proper drainage of the sinuses. In addition to coating the packing with antibiotic ointment, broad-spectrum antibiotics should be administered. *Dysphagia* due to the packing can lead to poor oral intake, and IV fluid hydration may be required.

A significant decrease in $Pa_{O_2}$ (7.5 to 11 torr) and increase in $Pa_{CO_2}$ (7 to 13 torr) is seen in patients with nasal packing who are treated with sedation. [28] This physiologic adaptation is even more worrisome in patients with underlying lung disease. With the risk of *hypercarbia* and *hypoxia*, patients with lung disease with posterior nasal packs should be closely monitored in an intensive care setting.

*Tissue necrosis* of the nasal ala, nasal mucosa, and soft palate has been described secondary to improper placement or padding. The risk of necrosis increases with the

duration of the packing, so all packing should be removed in 3 to 5 days.

If the *posterior pack becomes dislodged*, it will fall into the oropharynx with the risk of asphyxiation, vomiting, and aspiration. The patient, as well as nursing personnel, needs to be familiar with the technique for removing the pack. To accomplish this, cut the anterior sutures that exit the naris from the gauze roll if they have not already broken. Grasp the sutures exiting the mouth and guide the packing out of the nasopharynx. It may be necessary to manually extract the packing with forceps or digits.

*Toxic shock syndrome* has been described with nasal packing. [29] The syndrome is caused by a toxin released by *Staphylococcus aureus*. Sudden onset of vomiting and diarrhea with high fever as well as development of an erythrodermic rash heralds the onset of the disease. Untreated, the disease can advance into shock and multisystem organ dysfunction. Therefore, in addition to coating the gauze with antibiotics to decrease the local bacterial concentration, broad-spectrum antibiotics should be given to any patient with nasal packing. Any symptoms of toxic shock syndrome should prompt the removal of the packing and admission to the hospital for fluid hydration and IV nafcillin or vancomycin therapy.

## Septal Hematoma

Trauma to the anterior portion of the nasal septum may cause a hematoma to form between the perichondrium and cartilage. If untreated, the hematoma may cause complications including septal perforation, septal abscess, and cartilage destruction, with resultant saddle-nose deformity.

Diagnose a septal hematoma by inspecting the nasal septum for swelling, pain, and a fluctuant area. Direct palpation may be necessary, as newly formed hematomas may not yet be ecchymotic. Inspect both sides, as bilateral hematomas are possible. The absence of nasal obstruction does not rule out a septal hematoma.

### Drainage

Treatment of a septal hematoma consists of evacuation of the clot with subsequent reapproximation of the perichondrium to the cartilage. To drain the hematoma, *incise* the mucosa horizontally over the hematoma after adequate anesthesia is achieved (Fig. 68-38) . Suction out all clot and then irrigate with normal saline. Excise a small amount of mucosa to prevent premature closure of the incision and place a section of a sterile rubber band to act as a drain. The nostril should then be packed, as in anterior epistaxis, to reapproximate the perichondrium to the cartilage.

Give the patient broad-spectrum antibiotic therapy. Inspect the septum daily for signs of infection, recurrent hematoma, or necrosis. Recurrent hematomas should be evacuated. When there is no further hematoma for a 24-hour period, remove the drain. Pack the affected naris for 1 more day to complete the apposition of the perichondrium to cartilage where the drain has been. Any evidence of infection should prompt admission

for IV antibiotics and surgical debridement.

## Nasal Fracture

Nasal fractures present with symptoms ranging from mild swelling to epistaxis to periorbital ecchymosis with obvious deformity. As with any trauma to the head, coexistent intracranial injury or neck injury must be evaluated first. In the evaluation of nasal trauma, rule out the existence of a septal hematoma or cerebrospinal fluid rhinorrhea. In most cases, the swelling and soft tissue deformation prevent adequate evaluation and/or treatment. Radiographs are not routinely recommended as they will not alter the course of treatment or injury. [30] Have the patient apply ice to the area and keep the head elevated to treat the soft tissue swelling. Refer the patient to an otolaryngologist for reexamination and definitive treatment in 3 to 5 days. Stress to the patient the importance of being evaluated again in less than 10 days so that the bones do not set in a malaligned state.

### Nasal Fracture Reduction

Simple fractures with minimal local swelling can be treated with closed reduction. To minimize potential litigation, obtain written consent and take prereduction and postreduction photographs. Inform the patient that the outcome is not guaranteed, as impacted fractures may not reduce and greenstick fractures may deform again after reduction. Any swelling may obscure the extent of the deformity or the degree of reduction.

Anesthetize the mucosa as described above (see Figs. 68-27 (Figure Not Available) and 68-28) . Topical anesthetic is usually adequate; however, infiltration with 1% lidocaine without epinephrine may be used adjunctively. For infiltrative anesthesia, inject the lidocaine either deep to the nasal fracture by entering intranasally or externally into the fracture site for a hematoma block. The latter approach is usually quite uncomfortable for the patient.

Asch forceps or a scalpel handle is advanced into the naris with the dominant hand (Fig. 68-39) (Figure Not Available) . Apply pressure in an anterosuperior direction at a right angle to the ridge of the nasal bone. The other hand manipulates the nasal fracture in an anteroinferior direction. Firm, constant pressure is maintained until the bone shifts back into its original position. Assess the reduction for malalignment or subsequent displacement secondary to a greenstick fracture. If either occurs, refer the patient to an otolaryngologist.

Exterior splint dressings can be applied to maintain reduction. Some authors believe this will mask an incomplete reduction or adversely manipulate the reduction during placement. Remove the splint in 7 to 14 days. [31] As in most closed fractures, antibiotics are not indicated. Epistaxis or direct evidence of an open fracture should prompt referral to an otolaryngologist and initiation of broad-spectrum antibiotics.

**Nasal Foreign Bodies**

Nasal foreign bodies frequently present in the pediatric patient, but it is not uncommon to find them in psychiatric or mentally retarded populations as well. Usually a family member has witnessed the event or the patient actually complains of discomfort from the foreign body. Maintain a high level of suspicion, however, in patients who present with unilateral purulent nasal discharge, unilateral sinusitis, or recurrent unilateral epistaxis, as retained foreign bodies can often initially fail to cause pain or other symptoms.

Types of nasal foreign bodies vary widely and often include food (e.g., meat, nuts, beans), beads, jewelry, hardware (e.g., nuts, screws), and even certain living larvae or worms. Alkaline button batteries may cause significant mucosal burns, ulcerations, necrosis, and even septal perforation in hours to days. [32] It is imperative to promptly remove these batteries before tissue damage occurs from leakage of battery contents, electrical currents, and direct pressure.

Nasal foreign bodies often come to rest on the floor of the anterior or middle third of the nose. Metallic or calcified objects may show up on x-ray, but physical examination remains the most reliable means for diagnosis. Maxillary, ethmoid, or sphenoid sinusitis may also accompany foreign body retention. Plain radiographs or facial computed tomographic scanning may be of value in detecting sinusitis, although they are usually not necessary in the acutely retained object.

Failure to remove nasal foreign bodies results in admission for removal under anesthesia; therefore, it behooves emergency medicine physicians to be skilled in this procedure. Admission incurs increased cost, inherent procedural risks, and psychological stressors for both the parents and patients. Like auricular foreign bodies, the removal of nasal foreign bodies can be both frustrating and time consuming. Irrigation cannot be used, as fluid will pass through the nasopharynx.

**Nasal Foreign Body Removal**

A cooperative patient is essential; therefore, young children often require general anesthesia for more posterior foreign bodies. While conscious sedation may aid in removal and preclude the need for admission and general anesthesia, the possible increase in aspiration risk must be considered with use of agents that blunt protective airway reflexes. Before attempting removal, anesthetize and vasoconstrict the affected naris, as previously described in the chapter. Obtain assistance to stabilize the patient's head, and immobilize younger patients as required. The more cooperative patients should be placed in the "sniffing" position, and a head lamp should be used for proper illumination.

Use *alligator forceps* or *bayonet forceps* to retrieve more anteriorly lodged foreign bodies with edges that are amenable to grasping. *Wire loops, right-angle hooks,* or even a properly bent *paper clip* may be carefully passed by the object and rotated, allowing it

to be pulled from the naris (see Fig. 68-18) . Direct mucosal trauma and epistaxis may occur with any of these methods. Foreign bodies with smooth, rounded edges that are difficult to grasp or to pass instruments around may be extracted with a *suction-tip catheter* in a similar manner as that described earlier for EAC foreign bodies.

Objects that cannot be removed with anterior instrumentation may be removed with *balloon catheters*. Several case series have depicted Fogarty catheters as being highly effective in removing nasal foreign bodies (Fig. 68-40) . [33] [34] A No. 4 or 5 vascular Fogarty catheter, a 12 Fr Foley catheter, and a No. 6 biliary Fogarty catheter have all been described for this use in the literature (the biliary catheter reportedly is less apt to rupture). The patient lies supine after proper vasoconstriction and anesthesia of the affected naris. With a 5-mL syringe attached, and the catheter lubricated with lidocaine gel, the tip is passed above the object and into the nasopharynx. The balloon is then inflated with air or water (approximately 2 mL in small children and 3 mL in older children) with the thumb controlling the syringe plunger and, thus, balloon size. The catheter is slowly withdrawn until resistance is felt, and then the object is removed. Complications mentioned in the literature are limited to occasional mild posttraumatic bleeding only, but the theoretical risk of airway obstruction by the balloon or aspiration from further displacement of the object should be considered.

Another approach to the posteriorly placed nasal foreign body is blowing the object out with *air pressure*. One technique requiring no instrumentation is performed by lying the child supine and occluding the unaffected nostril (Fig. 68-41). As in mouth-to-mouth resuscitation, air is then exhaled by the examiner until resistance (from the patient's epiglottis shutting) is felt.  Air is then briskly blown into the mouth which produces an outward pressure behind the object. This either moves it within grasping reach or pops it completely out of the naris. The patient's parent may also be talked through this procedure, which can provide for a less frightened and more cooperative patient. A variation on this method includes using a bag-valve mask instead of the examiner's mouth to provide the air pressure, and application of the Sellick maneuver to prevent air from passing down into the esophagus. [37] Although theoretical complications would include possible barotrauma to the tympanic membrane or those seen with positive-pressure ventilation (pneumothorax, mediastinal emphysema), no reported complications have been published in regard to these two techniques.

# Chapter 69 - Emergency Dental Procedures

**James T. Amsterdam**

Patients with a variety of general dental, oral, and maxillofacial emergencies may present to any emergency unit. Emergencies may range from an agonizing toothache to massive maxillofacial trauma or infection. Most general dental emergencies can be evaluated and managed initially by the emergency physician; however, pediatric dental emergencies and dentoalveolar trauma may require immediate dental consultation and early follow-up. In addition, consultation is essential in maxillofacial trauma or in certain dental infections in which a seemingly minor problem may have potential life-threatening implications, including airway compromise, septicemia, and dehydration. Management of oral and facial pain, dentoalveolar trauma, dental infection, and maxillofacial trauma requires an understanding of the anatomy of the stomatognathic system and the relevant anatomy will be discussed. Intraoral and extraoral local anesthesia can be very useful in the diagnosis and management of dental emergencies; this topic is covered in Chapter 33 . This chapter concludes with an overview of the oral manifestations of systemic disease that are of particular importance to the emergency physician.

## ANATOMY

### Stomatognathic System

The muscles of mastication are divided into 2 groups: the supramandibular muscles, or elevators of the mandible, and the inframandibular muscles, or depressors. The most important elevating muscles are the masseters, the medial pterygoids, and the temporalis. The bilateral simultaneous function of this group is to move the condyle of the mandible superiorly and posteriorly. The muscles involved in the depressor function of the mandible are the lateral pterygoids, the digastric muscles, the geniohyoid, and the mylohyoid. Unilateral contraction of the lateral pterygoid muscle causes movement of the mandible to the opposite side. If both lateral pterygoids contract simultaneously, the mandible is depressed, causing the jaw to open in a downward and forward movement.

The mandible is essentially formed bilaterally by 2 rami: the horizontal portion and the ascending portion (Fig. 69-1) (Figure Not Available) . The ascending ramus of the mandible extends up to form 2 processes: the coronoid process, which extends anteriorly, and the more important condylar process, which extends posteriorly. The temporomandibular articulation is a diarthrosis joining the mandibular fossa and the articular tubercle of the temporal bone with the condyle of the mandible. A fibrous connective tissue articular disk or meniscus intervenes between the articulating bones. A joint capsule surrounds the temporomandibular joint (TMJ). The capsule consists of an outer fibrous layer, which is strengthened on its lateral surface to form the TMJ and the capsular ligaments. The capsular ligaments reinforce the capsule and function to limit mandibular movement. A small amount of synovial fluid may be found in the articular spaces. Frequently,trauma to the mandibular condyle produces pain resulting from extension or torquing of these ligaments, which should be distinguished from pain

caused by a fracture in this area.

**Teeth**

A tooth has been described as a homogeneous body of dentin surrounding a central pulp--the neurovascular supply--from which the microporous dentin is nourished and was initially derived. The pulp continuously lays down additional dentin throughout life. The tooth may also be divided into coronal and root portions. The enamel-covered coronal portion is the part that is normally seen in the mouth. The root portion of the tooth, which serves to anchor it, is covered with cementum, a substance that is much softer than enamel (Fig. 69-2) .

There are numerous classifications for the teeth. The permanent dentition generally consists of 32 teeth, which comprise 4 types--incisors, canines, premolars, and molars (Fig. 69-3) (Figure Not Available) . If one begins from the midline and counts backward, one will find 1 central incisor, 1 lateral incisor, 1 canine, 2 premolars, and 3 permanent molars in the normal dental anatomy. The third molar is commonly referred to as the wisdom tooth. Agenesis, or absence, of any of these teeth can occur occasionally. In addition, a patient can have extra, or supernumerary, teeth, which are somewhat small and unusually shaped. There are many methods of notation in the literature for numbering or classifying teeth. Although some systems are more universal than others, it is perhaps best for the emergency physician simply to describe the type of tooth and the location involved in a particular emergency (e.g., an upper right second premolar or a lower left canine). The following dental nomenclature may be of use to the emergency physician:

> *Facial:* That part of a tooth that faces the oral vestibule, or the cheek and the lips. In the area of incisors to canines, this surface is called the *labial* surface; for premolars and molars, it is referred to as the *buccal* surface.

> *Oral:* That part of a tooth that faces the tongue or the palate, usually referred to as the *lingual* surface of the tooth.

> *Approximal:* The contacting areas of adjacent teeth. The area closest to the midline is called the *mesial* surface, and the area toward the posterior aspect of the mouth is referred to as the *distal* surface.

> *Occlusal:* Biting surfaces of the premolars and the molars.

> *Incisal:* Biting surface of the canines and incisors.

> *Apical:* The tip of the root.

> *Coronal:* Toward the biting surface of the tooth.

### Periodontium

The normal periodontium can be divided into 2 major components: the gingival unit and the attachment apparatus. The *gingival unit* is composed of the soft tissues investing the teeth and the alveolar bone. The *gingiva* is covered by a keratinized, stratified squamous epithelium. It extends from the free gingival margin to the mucogingival junction. Apical to the mucogingival junction is the *alveolar mucosa,* which is covered by a nonkeratinized, stratified squamous epithelium and is continuous with the mucosa of the lip and the cheek.

In healthy individuals, the gingiva is attached tightly to the tooth. From a level that is coronal to the margin of the alveolar bone and extending to the level of the cementoenamel junction, connective tissue fibers from the gingiva insert into the cementum of the root.

Coronally to the epithelial attachment is a space bounded on 1 side by enamel and on the other by a continuation of the gingival epithelium. This space is called the *gingival sulcus.* This is the cuff that is formed around the necks of the teeth by the gingival tissues. The gingiva lining this space is not attached to the tooth and is therefore called *free gingiva.* The gingiva apical to the base of the gingival sulcus is called *attached gingiva.* In the healthy periodontium, the gingival sulcus is rarely >3 mm in depth.

The *attachment apparatus* is, as the name implies, the group of structures that attach the teeth to the jaws. It consists of the cementum covering the root, the alveolar bone surrounding the root, and the periodontal ligament. The periodontal ligament is composed of collagen fibers that insert on 1 end in the alveolar bone and on the other end in the cementum. It is important to note that the union of the tooth to the alveolar bone is not a direct calcific union, but a fibrous attachment. The anatomy of the dental unit (crown and root) and the periodontium is illustrated in cross section in Figure 69-2 .

## DENTAL ALVEOLAR TRAUMA

### Fractures of Teeth

The simplest type of dental trauma involves the fracture of anterior teeth. The management of dental fractures is based on (1) the extent of the fracture in relation to the pulp of the tooth and (2) the age of the patient. A classification system, the Ellis system, was developed to describe the anatomy of fractures of teeth. The emergency physician may  alternatively use a descriptive classification of traumatic injuries to teeth and supporting structures.

#### Ellis class I fractures.

The Ellis class I fracture involves only the enamel portion of the tooth (Fig. 69-4) . This is generally a minor problem and requires immediate intervention only if a sharp piece of tooth is causing trauma to soft tissues. In such situations, the rough edge may be

smoothed with something as simple as an emery board, or the patient may be referred at his or her convenience to a general dentist for more definitive management. What is perhaps most important is for the emergency physician to reassure anxious parents that with plastic enamel bonding materials, a cosmetic restoration of the tooth is possible. It would be inappropriate for the emergency physician to attempt immediate restoration of these teeth; however, no irreversible damage will occur from the smoothing of rough edges. These fractures are not painful and do not result in sensitivity to heat or cold.

**Ellis class II fractures.**

The Ellis class II fracture is a more complicated fracture in that it involves not only the enamel, but also the exposure of dentin. On inspection, dentin is identified by its pinkish or yellow appearance, as opposed to the white hue of enamel. The patient with exposed dentin may frequently complain of sensitivity to hot or cold or even air. The immediate treatment of the Ellis class II fracture is dictated by the age of the patient (Table 69-1) (Table Not Available) . Because, as the tooth matures, the pulp continues to produce a larger amount of dentin while the pulp itself shrinks in size, the dentin that is exposed in the Ellis class II fractures in patients <12 years of age is closer to pulpal tissue.

Dentin is a microtubular structure that can permit the passage of microorganisms from the oral environment directly to the pulp. Hence, contamination and resulting inflammation of the pulp can be anticipated in affected patients if dentin is exposed to the oral environment for >24 hours. Therefore, because of the possibility of damage to the pulp, the management of Ellis class II fractures in patients <12 years of age requires immediate placement of a dressing on the exposed dentin. The dressing not only provides pain relief, but also helps prevent infection.

A simple dressing that the emergency physician can apply consists of a calcium hydroxide resin paste (Dycal), which is available from dental supply companies (Fig. 69-5) . Calcium hydroxide paste is prepared by mixing small (equal) amounts of base and accelerator from 2 tubes with an applicator. The exposed dentin is dried with a piece of gauze,
and a small amount of calcium hydroxide is placed on the exposed area with a cleansed applicator instrument; the tooth surface must be perfectly dry, or the Dycal will not adhere. The tooth is then covered with a small piece of aluminum foil or, preferably, "dental-adhesive dry" foil. Dycal will set in approximately 2 minutes, more quickly if exposed to humidity. Dycal is easily removed by the dentist. Patients are advised not to eat solid foods to prevent dislodging of the Dycal dressing.

More sophisticated techniques involve covering the tooth with the plastic bonding materials mentioned previously; an aesthetic restoration will then result. The patient treated by the emergency physician requires referral within 24 hours. Patients with simple class II fractures (i.e., patients >12 years old who have a greater dentin-to-pulp ratio) may be advised to avoid extremes in temperature and to seek dental care the following day. A protective dressing need not be routinely applied. Patients with severe Ellis class II fractures (which may usually be recognized because of their larger exposed areas of pinkish- or yellowish-tinged dentin) should be treated with a dressing in a manner similar to that for younger patients. Analgesics may also be required, depending

on the degree of sensitivity of the patient. It should be noted that correct management of Ellis class II fractures may obviate the need for root canal therapy. It may be advisable to warn any patient who has sustained trauma to the anterior teeth, no matter how minor, that disruption of the neurovascular supply to the tooth may have occurred. The long-term complication of the initial trauma may be necrosis of the pulp or resorption (dissolving) of the root.

**Ellis class III fractures.**

Ellis class III fractures of the teeth involve, in addition to fracture of enamel and exposure of dentin, actual exposure of the pulp. One may differentiate the Ellis class III fracture from the Ellis class II fracture by gently wiping a tooth clean with a piece of gauze to remove any blood that may be present from soft tissue trauma. The tooth is then examined for any red blush of dentin or frank drops of blood that may extrude from the pulp. A patient may complain of exquisite pain; however, on occasion the tooth may be in shock from disruption of the neurovascular supply, in which case the patient feels little sensitivity in the tooth.

Ellis class III fractures are true dental emergencies and require immediate consultation with a general dentist or endodontist (root canal specialist) (Fig. 69-6) (Figure Not Available) . Ultimate treatment will consist of the total removal of pulpal tissue (pulpectomy). Alternatively, in the case of primary teeth, partial amputation of pulpal tissue (pulpotomy) may be performed. A delay in either of these procedures will result in significant pain and probable abscess formation. Some reports have recommended the use of the endodontic barbed broach for the removal of exposed pulp in the emergency department. [3] A barbed broach, when twisted in the root canal, engages the remaining pulp; when the broach is removed, the pulp is generally attached. However, even in the hands of an endodontist, the use of this instrument is difficult, and it is prone to breakage in the root canal. A broken instrument in a root canal is difficult to remove and may necessitate extraction of the tooth. Therefore, emergency physicians and other nondentists generally should not attempt to use this instrument. If a dentist is not immediately available, the tooth may be temporarily covered with aluminum or, preferably, dental dry foil so as to minimize pulpal irritation and pain.

Because of bleeding or other sources of moisture, it may be difficult to apply Dycal to these fractures effectively. Analgesics should be prescribed, and the patient should be told to see a dentist as soon as possible. It is preferred to neither prescribe nor apply any of the over-the-counter topical dental analgesic preparations. Although these agents may give the patient temporary relief from pulpal pain, they often cause soft tissue damage because of their irritant effects, and they are caustic to the tooth. Use of these agents has been known to result in sterile abscesses. In all cases of tooth fracture, the soft tissue should be palpated for tooth fragments and radiographed if swelling limits the examination and if one has not accounted for tooth fragments.

## Subluxation and Avulsed Teeth

The same force that may have resulted in the fracture of anterior teeth may also result in actual loosening of the tooth in its socket. This is called *subluxation*. Traumatized

teeth should always be examined for subluxation by applying pressure with the fingers or with 2 tongue blades on each side of the tooth. The tooth is wiggled in a back-and-forth motion. A more subtle indication that teeth have been traumatized is the appearance of blood in the gingival crevice of the tooth. Teeth that are minimally mobile usually heal well if the patient is kept on a soft diet for 1 to 2 weeks. Teeth that appear grossly mobile to the eye require stabilization as soon as possible. The techniques for stabilization are described later. Although stabilization procedures are usually performed by a general dentist or an oral and maxillofacial surgeon, the techniques that are described can be used by the emergency physician who is trained in such procedures. As a temporary measure for teeth that are very loose, it is often useful to have the patient bite gently on a piece of gauze to keep the tooth in place, pending examination by a dentist or an oral surgeon.

**Avulsed and Intruded Teeth**

Teeth that have been completely avulsed from the socket constitute a true dental emergency. If the patient is unaware of the location of the missing tooth, a complete *intrusion* of the tooth below the level of the gingiva must be ruled out with a radiograph (Fig. 69-7) (Figure Not Available) . An intruded tooth has been forced back into the alveolar bone, implying disruption of the supporting structures and possible fracture of the alveolar bone. Intrusion may be missed by a superficial examination, and one should not automatically conclude that all spaces in dentition following trauma represent avulsed teeth. Dice and coworkers have reported a case in which an intruded tooth was initially thought to be a fractured tooth; facial cellulitis and subsequent periodontal infection developed as a result of the misdiagnosis. [4] Intruded primary teeth ("baby teeth") in the absence of infection are allowed to erupt for 6 weeks before repositioning is considered. Intruded permanent teeth are surgically repositioned with a forceps within 24 to 48 hours and are then stabilized. Failure to diagnose intruded teeth may also result in cosmetic deformity.

As in other cases of dental alveolar trauma, the management of an avulsed tooth depends on the age of the patient and the length of time that the tooth has been absent from the oral cavity. Fully avulsed primary anterior teeth in the pediatric patient age 6 months to 5 years *are not replaced into their sockets.* Loss of these primary anterior teeth poses no threat to normal development and alignment of permanent teeth. Reimplanted primary teeth have a high tendency to ankylose, or fuse to the bone itself. The most serious consequence of an ankylosed primary tooth is facial deformity in the child. As growth continues, it may hinder eruption of the permanent tooth, and as time progresses, ankylosed teeth are more difficult to surgically remove. Temporary prosthetic replacement of avulsed primary teeth is easily accomplished if a cosmetic effect is desired.

In general, *permanent* teeth should be replaced in their sockets as soon as possible. Care must be taken to ensure that the tooth does not become dehydrated and that the remaining periodontal ligament fibers are disturbed as little as possible. The transport medium is also important in terms of pH and osmolarity to preserve the cells of the periodontal ligament fibers. [5] A percentage point for successful reimplantation is lost every minute that the tooth is absent from the oral cavity. This time interval can be

extended from 4 to 24 hours if the tooth is placed in a medium consisting of a pH-balanced cell culture fluid, such as Hank's solution. A system called the *Save-A-Tooth system* (Biological Rescue Producers, Inc., Pottstown, Pa) contains the cell-preserving fluid in a break-resistant, sterilized container that does not need to be refrigerated. [6] [7] An avulsed tooth is simply dropped in the system's basket, which sits in the fluid, and the cap is replaced. When the tooth is needed, the basket is lifted out of the container. Teeth that have been absent from the oral cavity for >20 minutes should also be placed in this fluid for at least 30 minutes to rehydrate the periodontal ligament cells prior to replacement.

Therefore, when a call is received about an avulsed tooth, the first question that should be asked is the age of the patient. If it is determined that the tooth is permanent, the parent or patient should be instructed to replace the tooth immediately, if possible. If there is gross debris on the tooth, the tooth can be rinsed under cool, running tap water prior to being reimplanted in its socket. If actual reimplantation is not possible, the patient should be advised to bring the tooth to the emergency unit as quickly as possible, preferably in a "Save-A-Tooth" container (Fig. 69-8) (Figure Not Available) or a cup of milk. Alternatively, the alert, older patient may be allowed to place the tooth in his or her own mouth to bathe in saliva, but care should be taken that the tooth is not swallowed or aspirated. In all cases, one should attempt to prevent dehydration of the avulsed tooth, because teeth that become dehydrated have the poorest prognosis for healing.

An avulsed tooth should be reimplanted as soon as possible; the initial procedure is relatively simple. [7] The avulsed tooth is held by the crown at all times. It is rinsed under saline or under running water to remove gross debris but is *not* scrubbed. Scrubbing damages remaining periodontal ligament fibers and adversely affects reattachment. Ideally, if the "Save-A-Tooth" system is available in the emergency department, the avulsed tooth can be cleansed by soaking in this system. Indeed, if the tooth has been avulsed for >20 minutes, it should be soaked in the preserving solution for 30 minutes. If the patient has other, more serious injuries, the tooth can remain in Hank's solution until reimplantation can be accomplished. The socket is then inspected. Blood clots or bone fragments, which may prevent reimplantation, should be irrigated or removed by gentle suction. When reimplantation is delayed (>30 minutes), local anesthesia is recommended (see below).

The socket is suctioned and debrided of all blood and foreign matter, and the tooth is immediately reimplanted. Generally, the tooth can be forced into its original position with the operator's thumb. The socket should not be sharply scraped, because this may damage the periodontal ligament or the attachment fibers. If the tooth does not fully sit in the socket as compared with the alignment of the adjacent teeth, it is often because the socket contains a blood clot or the injured gingiva prohibits implantation. These conditions can often be remedied. If there is confusion as to the position of the tooth after reimplantation, the procedure should stop at this point. The patient should then gently bite on a piece of gauze until seen by a general dentist or an oral or maxillofacial surgeon, or the tooth can be transported in the Hanks solution to the dentist. Large gingival lacerations may be sutured at this juncture.

**Prognosis for Patients with Avulsed Teeth**

When a tooth is avulsed, the neurovascular supply to the tooth is completely disrupted. If the tooth is reimplanted within a few minutes, there may be some restoration of the neurovascular supply, but most avulsions result in hypoxia and ultimate necrosis of the pulp. Therefore, almost all reimplanted teeth require subsequent root canal therapy within a short period (i.e., 7 days). The purpose of root canal therapy is to (1) debride the pulp, (2) render the tooth insensitive to pain, and (3) fill and seal the pulp chamber with an inert material. This inert material prevents infection or chronic inflammation, which may discolor the tooth or interfere with stabilization of the tooth by the periodontal ligament.

The object of immediate reimplantation is not to keep the tooth alive, but to keep the periodontal ligament alive, thus ensuring a retained functional tooth. Healing of the periodontal ligament is variable following reimplantation. In addition, some resorption of the root surface always follows replantation. The degree of resorption varies and may even result in ankylosis of the tooth with surrounding bone. Although the long-term prognosis favors retaining an avulsed tooth if timely treatment is available, the patient should always be advised of the possibility of losing the tooth in a few months to a number of years following reimplantation. In general, immature permanent teeth have a better prognosis for survival than do older teeth.

**Stabilization Techniques**

Avulsed teeth require immediate stabilization so that they do not exfoliate. Although stabilization is normally performed by a general dentist or an oral or maxillofacial surgeon, there are situations in which it may be performed by the emergency physician. Stabilization is indicated in the case of a single avulsed tooth that has been placed back into the socket with satisfactory alignment. Satisfactory alignment of the tooth is judged both by visual inspection and by a report from the patient that there is no prematurity of occlusion when the jaw is closed. Even a millimeter of extrusion of a reimplanted tooth may cause occlusal disharmony in some patients, and final occlusal adjustment should not be performed by the emergency physician.

Any tooth stabilized by the emergency physician should be evaluated by a general dentist or an oral or maxillofacial surgeon within 24 to 48 hours. Several techniques are available to the emergency physician for the stabilization of a single avulsed tooth. The application of Erich arch bars is perhaps the oldest technique used for stabilization of avulsed teeth. These arch bars are also used for stabilization of mandibular and maxillofacial fractures. Figure 69-9 (Figure Not Available) shows the application of the Erich arch bar. Application of the arch bar is not a simple technique and should not be performed by the nondentist. [8]

A temporary and readily available measure to stabilize avulsed or subluxated teeth in the emergency unit by nondentists has been described. [9] A temporary splint that has been accepted by the Council on Dental Therapeutics of the American Dental Association [10] is made using the commercially available Coe-pak (Coe Laboratories,

Inc., Chicago). Coe-pak, a zinc oxide-eugenol preparation that sets to semihardness, is prepared from tubes that contain a base and a catalyst (Fig. 69-10) (Figure Not Available) . Following reimplantation of the tooth, the splint material is applied in a soft, clay-like consistency and is molded over the gingival line, in the front and rear of the tooth, and into the spaces between the teeth. A liquid diet is possible with the splint in place, and the patient should be directed to see a dentist within 24 hours.

Avulsed teeth are stabilized for approximately 10 days to 2 weeks and are then brought back into function. However, if teeth are reimplanted by an emergency department physician, the patient should be seen by the dentist in 24 to 48 hours. As in any type of dentoalveolar trauma, general precautions that should be taken to avoid long-term complications are explained to the patient. When there are concomitant alveolar fractures, stabilization of avulsed or subluxated teeth serves to stabilize not only the teeth, but also their alveolar bone. In this situation, therapy is directed toward the conservation and healing of bone, and therefore stabilization is left for a minimum of 6 weeks, recognizing the risk of possible ankylosis of the teeth. The indiscriminate loss of alveolar bone leads to much more difficult prosthetic restoration of this area than with the removal of an ankylosed tooth. Generally, prophylactic antibiotics (phenoxymethyl penicillin or erythromycin, 250 to 500 mg orally 4 times a day) are used when avulsed teeth are reimplanted in the oral cavity, but their benefit is unproven. Tetanus prophylaxis should also be instituted when needed.

**Lacerations Associated with Dentoalveolar Trauma**

Subluxated or avulsed teeth are frequently accompanied by associated lacerations of the gingiva, the mucosa, the lips, should preferably precede definitive closure of any soft tissue laceration. The lips and the oral mucosa undergo constant manipulation during the stabilization procedures for teeth; therefore, carefully placed sutures may be torn, increasing the existing soft tissue injury and making aesthetic closure more difficult. Plastic closure, once dental stabilization has been first performed, may then be left undisturbed.

Gingival avulsions should be repaired in approximately their original position with fine silk sutures (Fig. 69-11) . Small lacerations may be left open to heal. Gingiva is very friable, and suturing may be difficult. The suture may be passed between teeth using the suture needle and then passed back between the teeth again like dental floss and tied in the front anchoring the buccal and lingual flaps; or the suture may be anchored to the mucosa on the other side of the teeth. When appropriate, patients with large gingival avulsions should be referred to an oral surgeon.

A frequently encountered problem is that of the through-and-through laceration resulting in an open communication between the skin and the oral cavity. This may result from a tooth being forced through the upper or lower lip. Occasionally, pieces of a fractured tooth are found imbedded in the soft tissue. Their presence should be suspected any time a tooth is chipped or avulsed. Devitalized tissue should be removed by sharp debridement, and the area should be irrigated under pressure. After appropriate debridement and irrigation, it is best to close mucosal lacerations larger than 0.5 cm with sutures. Smaller lacerations may be left alone. Mucosal lacerations can be closed

with 4-0 Dexon or Vicryl or 3-0 to 4-0 black silk sutures; gingival lacerations are closed with 4-0 black silk. The cut ends of nylon sutures are very irritating, and therefore nylon should not be used. Large, open intraoral mucosal lacerations result in much discomfort for the patient and frequently become infected from the accumulation of debris.

Patients with sutured intraoral lacerations are advised to keep the area clean by rinsing with warm saline and swabbing locally with hydrogen peroxide. Lip sutures may be covered with a thick coating of petrolatum jelly or antibiotic ointment. Patients are given the usual wound precautions and are checked within 24 to 48 hours. A certain amount of edema may be found when the wound is checked; in addition, a whitish granulation tissue, resembling pus, may have developed. The emergency physician should not immediately assume that this means the area is infected. Significant pain usually signifies infection, however. If there is any suspicion of infection, the patient should be rechecked at 24-hour intervals until the edema has resolved. Frank infection should be treated with penicillin and hot compresses. Occasionally, sutures may need to be removed to promote drainage. Like patients with reimplanted avulsed teeth, individuals with through-and-through lacerations should be given tetanus prophylaxis; antibiotic coverage is often prescribed. Penicillin or erythromycin is advocated by many. [11] Skin sutures are frequently removed in 3 to 5 days to minimize scar formation. Intraoral sutures are removed in 5 to 7 days.

## HEMORRHAGE

Oral hemorrhage may occur spontaneously from the gingiva or, more commonly, may be the result of dental treatment, especially surgical extraction of teeth. Patients with this complaint frequently present at night, when their dentist or oral surgeon is not immediately available. A patient presenting with a bleeding gingiva should be questioned about any recent dental scaling, curettage, or other procedures. Such bleeding usually responds to peroxide mouth rinses and local pressure with gauze. In patients with advanced periodontal disease, small remnants of missed granulation tissue may ooze continuously for hours. Spontaneous gingival hemorrhage without a history of recent dental therapy may be the initial presentation of a systemic process (e.g., leukemia or coagulopathy). A decision to investigate such hemorrhage with laboratory testing is based on the extent of the hemorrhage, the age of the patient, and other information obtained from the history and physical examination.

Most commonly, patients present with bleeding following dental extraction. Postextraction bleeding most often responds to sustained pressure, which can be produced by having the patient bite on gauze. Patients frequently report that they have already been doing this for hours. One should then ask whether the patient has been spitting excessively, smoking cigarettes, or using straws, each of which may create negative intraoral pressure and promote bleeding. Negative intraoral pressure removes blood clots from sockets and aggravates postextraction bleeding.

The procedure for the management of postextraction bleeding is a very systematic one. If clots are present, they should be removed with suction or wiped with gauze. Patients are then instructed to bite on gauze for 15 to 20 minutes. If bleeding continues after 15 to 20 minutes, local anesthesia consisting of 2% lidocaine (Xylocaine) with 1:100,000 or

1:50,000 epinephrine is infiltrated in the area of the socket until the tissue blanches (turns white). Gauze pressure is then reapplied. The epinephrine reduces bleeding, and the lidocaine allows the patient to bite without pain. Continued bleeding after 20 minutes may respond to the placement of a small piece of surgical foam or Gelfoam in the socket. This is then secured with a 3-0 black silk suture.

Sustained vigorous oozing after all of the aforementioned procedures have been performed warrants a screening coagulation profile, consisting of a complete blood cell count with differential, prothrombin time and partial thromboplastin time, and a platelet count. Postextraction bleeding is often caused by aspirin use. The patient must be questioned about all medications that may contain aspirin. Postextraction bleeding may also be the initial manifestation of a coagulopathy, such as hemophilia, especially in younger individuals. In some instances, postextraction bleeding results from improper surgical technique or flap design or lack of a sufficient number of sutures. Such flaps may need to be revised and resutured.

Following treatment, the emergency physician should warn the patient to avoid all liquids and solid foods for 2 hours and to avoid extremes in temperature, excessive spitting, smoking, or the use of straws or aspirin. If bleeding occurs at home, gauze pressure should be used.

The patient should be advised of the possibility that a *dry socket* will develop in 2 to 3 days. The patient becomes aware of a dry socket when excruciating pain develops in the area of the socket and a foul odor or taste occurs in the mouth after a day or two of no pain. Patients should know that the pain of a dry socket is easily managed by their dentist or an emergency physician. Application of local anesthesia to the area of the involved tooth, gentle irrigation of the socket, and application of an iodoform pack dipped in eugenol or Campho-Phenique or a commercial sedative dressing relieves the pain. The packing is placed gently in the socket, avoiding manipulation of the walls of the socket. A dry socket is a localized alveolar osteitis. Because of the high incidence of osteomyelitis secondary to scraping of a dry socket to initiate bleeding, aggressive debridement of the dry socket is contraindicated.

Patients with dry sockets are frequently given oral antibiotics; penicillin or erythromycin is preferred. The use of topically applied antibiotics is not indicated because of the increased incidence of sensitivity reactions. The dressing is replaced at 24-hour intervals until the patient is pain free.

In addition to dental extractions, bleeding is frequently seen after periodontal (gingival) surgery. The tissue involved in the surgical site is usually covered with a surgical dressing, which may be dislodged by bleeding. Bleeding following periodontal surgery will generally respond to gauze pressure. It should be noted, however, that periodontal surgical dressings are extremely important for wound healing, and incorrect placement of the pack can result in treatment failure. Therefore, the periodontist should be informed immediately that bleeding has occurred and should see the patient as soon as possible.

# DRAINAGE OF AN INFECTION OF DENTAL ORIGIN

Acute infection of the oral cavity and the jaws can be minor or life threatening. The most common dental infection is the periapical abscess, or the acute alveolar abscess, which usually begins in the periapical region of the tooth as a result of nonvitality or degeneration of the pulp. One can usually easily manage these infections by treating the tooth with endodontics or extraction. [12]

Periodontal and pericoronal infections are generally more difficult to manage. Most intraoral infections form an abscess and drain intraorally. When the infection is well contained, drainage of these areas is generally easy. Extension of this same infection to the fascial planes of the head and neck, however, may result in much more serious infection in the parapharyngeal space or may track exteriorly to drain at the surface of the skin (Fig. 69-12) (Figure Not Available) . Drainage of an infection of dental origin by the emergency physician should be limited to well-confined intraoral abscesses requiring intraoral drainage and fluctuant extraoral swellings that require drainage externally by an incision on the face.

## Pulpal Infection (Abscessed Tooth)

Although physical and chemical injuries to teeth result in pulpal necrosis and infection, dental infection most frequently is the result of carious (decay) exposure of the pulp. Destruction of the enamel and the dentin by caries opens a portal to the pulp for oral microorganisms. Before invasion of the pulp by microorganisms, the affected tooth usually becomes sensitive. Invasion of the pulp produces either a localized or a generalized pulpal infection. If the portal of entry into the pulp is adequate to allow spontaneous drainage, the patient may be asymptomatic. If drainage is obstructed, rapid involvement of the entire pulp results in pulpal necrosis. The patient can experience moderate to severe pain that has been ranked second only to that of renal colic. Untreated inflammation progresses through the teeth and extends into the periapical region; irritation in this area produced by inflammatory products in the necrotic pulp may result in the formation of periapical granuloma. This process may be asymptomatic and persists as long as drainage through the tooth continues.

Treatment consists of removing the infected pulp and obliterating the pulp chamber, allowing the tooth to remain (root canal therapy), or extracting the infected tooth and draining the periapical region. Antibiotic therapy is indicated if drainage cannot be established when the infection has perforated the cortex and spread into the surrounding soft tissue. Frequently, physical examination reveals a grossly decayed tooth. Alternatively, no apparent pathologic condition may be seen, or many teeth may appear decayed. One may localize the offending tooth by percussing individual teeth with a tongue blade; in most cases, tapping the involved tooth elicits a sharp pain.

In most minor dental infections, oral phenoxymethyl penicillin in doses of 250 to 500 mg 4 times a day is the drug of choice. Erythromycin 250 to 500 mg 4 times a day, clindamycin (Cleocin) 150 to 300 mg 4 times a day, and cephalexin (Keflex) or cephradine (Velosef) 250 to 500 mg 4 times a day are alternatives. Analgesics can be

prescribed. If the infection has broken through the cortex and there is subperiosteal extension of the infection with swelling, incision and drainage is the treatment of choice, as in any abscess.

## Periodontal Infection

Periodontal disease is a progressively destructive bacterial process involving the supporting structures of the teeth, which include the gingiva, the periodontal ligament, and the alveolar bone. Unlike the relatively confined pulp chamber, inflammatory exudate produced in periodontal infection usually drains freely, and the patient experiences little, if any, discomfort. Significant symptoms may not be apparent for many years. If for any reason drainage from the infected area is interrupted, the inflammation becomes acute and is similar to an acute periapical infection that spreads to the soft tissue. The lack of a carious lesion in the involved tooth and a dental radiograph help in differentiating these 2 processes. In most cases, acute periodontal infections tend to remain in the intraoral soft tissues rather than spread to the face and the neck.

Immediate treatment of periodontal infections includes drainage of the infected tissue (see later discussion), which may require removal of the involved tooth. Antibiotic therapy is usually instituted only if drainage cannot be achieved or if spread to tissues of the face and the neck has occurred. Follow-up periodontal therapy is needed to prevent recurrence. Warm saline rinses frequently promote drainage in periodontal areas, and antibiotics are prescribed if there are systemic manifestations. The condition usually does not require urgent referral. Frequently, scaling and curettage of the area relieve the abscess. In the emergency unit, however, incision and drainage of the abscess are generally preferred.

## Pericoronal Infection

Pericoronitis occurs when debris and microorganisms become trapped under soft tissues that partially overlie the crown of a tooth, usually in an erupting or partially impacted third molar (wisdom tooth). A localized infection that drains from under the tissue becomes established. If drainage is interrupted by sudden swelling of the overlying tissue, caused either by trauma from an impacted third molar or by the inflammatory process itself, the entrapped exudate will spread through other pathways, usually into the pterygomandibular or submasseteric spaces (see Fig. 69-12) (Figure Not Available) . The underlying bone is generally not involved. Clinically, marked trismus secondary to irritation of the masseter or the medial pterygoid muscles predominates. The pericoronal tissues are erythematous and swollen. Digital pressure in the area often elicits pain and produces a small amount of exudate under the infected flap.

Treatment includes antibiotics in virtually all cases. Removal of the impacted tooth or the infected tissue ensures good drainage. Frequent irrigations with warm saline during this period are beneficial and should be instituted immediately. An oral or maxillofacial surgeon should be consulted within 24 to 48 hours. Fluctuant swelling is amenable to incision and drainage. Soft tissue dissection should be performed with caution, because the space extends posteriorly in the retropharyngeal area. Incisions must be made with

great care in this region because of the proximity of the internal carotid artery, which may have been moved anteriorly with tissue swelling.

In many instances, progression of oral infection may cause acute facial cellulitis. The extent of the cellulitis, of course, is dependent on the virulence of the organism and the resistance of the host. In general, cellulitis from an odontogenic infection arising from the maxillary teeth involves the lower half of the face and the neck. This pertains only to the earlier stages of infection, because in many instances the entire side of the face may be involved, regardless of the origin of infection. In the nondebilitated host, most untreated odontogenic infections tend to localize and drain spontaneously (usually extraorally).

## Technique of Incision and Drainage

### Intraoral Technique

Before attempting incision and drainage [13] of an intraoral abscess, the emergency physician should first determine whether the infection or abscess is a simple one. The patient should have minimal trismus and should be able to open the mouth widely to allow inspection of the pharynx. The area of maximal fluctuance is anesthetized superficially with either a topical anesthetic spray, such as benzocaine-tetracaine-butamben-benzalkonium (Cetacaine), or (better) an injection of 2% lidocaine (Xylocaine) with 1:50,000 epinephrine until tissue blanching occurs. One should always remember that it is difficult to obtain profound anesthesia in an infected site with topical or local anesthetics. Superficial anesthetic techniques are used so as not to track infection more distally with a long needle. When regional nerve blocks distant to the area of infection are possible, they should be considered (see Chapter 33) .

The required equipment for incision and drainage consists of the instruments found in a standard incision-and-drainage tray (Fig. 69-13) . A No. 11 scalpel blade is recommended, as are a mosquito hemostat and drain material (such as ¼-inch iodoform gauze or a small 3-cm fenestrated Penrose drain). Antiseptic cleansing of the area to be drained is generally not necessary. A small 1-cm incision is made superficially over the area of fluctuance, with the point of the scalpel blade always facing toward the alveolar bone (Fig. 69-14) . Blunt dissection is then carried out with a mosquito hemostat to avoid any vascular structures. Cultures of draining pus may be obtained at the discretion of the clinician but are usually not required. However, cultures are recommended for complicated abscesses and when the patient is immunocompromised. The drained area is copiously irrigated.  If possible, one places the iodoform gauze or the Penrose drain by packing the site and securing 1 end of the gauze or the drain with a black silk suture (4-0) to prevent aspiration of the drain.

The patient is instructed to continue warm salt-water rinses hourly for the next 24 to 48 hours and to rinse several times during the night. The drain is removed in 24 to 48 hours, and intraoral rinsing is continued every 4 hours for another day. Patients generally begin taking oral antibiotics during the course of this therapy. Although the emergency physician may manage the initial phase of the dental infection, referral to a

dentist or an oral or maxillofacial surgeon is required for definitive therapy of the infection.

**Extraoral Technique**

If the dental infections described in the preceding section do not drain intraorally, they may spread to the face. The approach to drainage of these abscesses is similar to that of other soft tissue abscesses (see Chapter 40) . However, the emergency physician must determine that the infection, although it has an extraoral spread, is a simple one. The patient should have no trismus, and the retropharynx should be adequately visualized. Drainage of an infection of dental origin on the face requires more attention and care because of the cosmetic consequences of the procedure (Fig. 69-15) .

The patient is placed in a reclining position and is externally draped well so as to prevent drainage of purulence on the patient's clothing, the stretcher, and other materials in the treatment area. The skin is cleansed with a scrub, prepared with povidone-iodine solution, and draped in an appropriate manner. The skin is anesthetized superficially with 2% lidocaine (Xylocaine) and 1:100,000 epinephrine.

It is important to note that the incision in this case will not be made over the area of maximal fluctuance but inferiorly to the area of infection in a zone of *healthy* skin. A 1.0- to 1.5-cm incision that follows the natural tissue line as closely as possible is made through skin and subcutaneous tissue; blunt dissection with a mosquito hemostat is then carried out toward the area of infection to establish drainage. In compromised patients, a culture should be taken, and the area should be gently irrigated with saline. A Penrose or gauze drain should be placed. The area is covered with a gauze dressing, and the patient is instructed to remove the dressing 4 times a day and apply heat to promote drainage in this zone.

It is important to note that this is one of the few indications for the application of warm compresses to the face in the presence of dental infection. In general, all attempts should be made to establish drainage intraorally, but this procedure is used when extraoral drainage is inevitable. Any facial infection of dental origin that requires 24 hours of heat to obtain maximal fluctuance for drainage extraorally can probably be managed after that time with intraoral drainage. Obviously, scarring results from any extraoral drainage. Therefore, the procedure should be avoided if possible, but if it is necessary, the incision is made over healthy tissue.

## COMPLICATED HEAD AND NECK INFECTIONS

The emergency physician is often responsible for the actual incision and drainage of simple dental infections. When the infections described previously extend to the fascial compartments of the head and the neck, they immediately fall into the serious and complicated category (Fig. 69-16) . Toxic febrile patients with marked trismus require immediate attention to airway and early institution of IV antibiotics. The patient should immediately be referred to the oral and maxillofacial service for admission.

An uncommon complicated infection of dental origin seen by the emergency physician is Ludwig's angina. This infection is a bilateral, board-like swelling involving the submandibular, submental, and sublingual spaces, with elevation of the tongue. Emergency physicians frequently see affected patients before elevation of the tongue, during the initial manifestations of the infection. In the early stages, Ludwig's angina may appear deceptively benign (Fig. 69-17 A). Brawny induration is characteristic, and there is no fluctuance present for incision and drainage. Infection is commonly caused by hemolytic *Streptococcus* organisms, although it may be a mixed *Staphylococcus-Streptococcus* infection or a combination of aerobic or anaerobic organisms. The presence of anaerobes commonly accounts for the presence of gas in the tissues. Chills, fever, difficulty in swallowing, stiffness of tongue movements, and trismus are common presenting signs. Respiration becomes increasingly difficult as the tongue is elevated, and the oral pharynx becomes edematous (Fig. 69-17 B). Progression to airway obstruction may be rapid, occurring within only a few hours.

Treatment consists of high-dose IV antibiotic therapy. Intubation or tracheostomy should be considered in the acute stage to maintain the airway if respiration becomes embarrassed. Constant observation is important, since the airway may become obstructed without much warning. Although hospital admission (preferably in a surgical intensive care setting) is mandatory, surgical intervention may be necessary only if antibiotic therapy is not successful. The most common locations of dental infection in this condition are the lower second and third molar teeth; pus formation usually occurs medially or on the lingual aspect of the mandible. In Ludwig angina, as in any parapharyngeal space infection, mediastinal descent can occur because of the communication of the parapharyngeal space with the visceral space.

**Therapeutic Considerations**

The most important therapeutic modality for pyogenic orofacial infections of odontogenic origin is surgical drainage and removal of necrotic tissue. The need for definitive restoration or extraction of the infected teeth, the primary source of infection, is readily apparent. Antibiotic therapy, although important in halting local spread of infection and preventing hematogenous dissemination, cannot substitute for evacuation of pus.

Penicillin remains the antibiotic of choice for treating orofacial infections of odontogenic origin. *Bacteroides fragilis*, which is highly resistant to penicillin, is not normally a resident in the oral cavity; however, this organism has been recovered in 15% to 20% of anaerobic pleuropulmonary infections and is presumably related to aspiration of oropharyngeal flora. It is interesting to note that penicillin has remained effective in such cases, despite recovery of this organism as part of the mixed flora.

Cephalosporins, particularly cefoxitin (Mefoxin), may be excellent alternatives for penicillin-allergic patients, because the action of these agents against oral-obligate anaerobes is comparable to that of penicillin, and they also are effective against certain aerobic bacteria. The usual cephalosporin dosage is 1 to 2 g IM or IV every 6 hours. Clindamycin (600 mg IM or IV every 8 hours) is also highly effective, especially against anaerobes, when used in a controlled fashion and when possible side effects are

monitored. Erythromycin is generally active against most indigenous oral bacteria but is comparatively less active against anaerobic and microaerophilic streptococci, *Fusobacterium*, and anaerobic gram-negative cocci. Chloramphenicol, although highly active against obligate anaerobes, is potentially toxic and should be reserved for situations in which the pathogenic role of *B. fragilis* is of prime importance or for use as an alternative agent in patients allergic to penicillin, cephalosporins, or clindamycin.

# DISORDERS OF THE TMJ AND RELATED STRUCTURES

## Mandibular Dislocation

In acute dislocation of the mandible, the condyle moves too far anteriorly in relation to the eminence and becomes locked (Fig. 69-18) (Figure Not Available) . Subsequent muscular trismus prevents the condyle from moving back into the temporal fossa. Spasm of the external pterygoid, masseter, and internal pterygoid muscles, as well as the associated edema, results in extreme discomfort and anxiety for the patient. It is difficult for the patient to verbalize a complaint because he or she cannot close the mouth. Predisposing factors include anatomic disharmonies between the fossa and the interior articular eminence, weakness of the capsule forming the temporomandibular ligaments, and torn ligaments. Dislocation is likely to occur during maximum opening, such as during yawning, laughing, or "popping" of the mandible in an open position. Although the TMJ is a double joint, dislocation may occur bilaterally or unilaterally. The jaw may be locked open symmetrically or may deviate to the side opposite the side of dislocation. Palpation of the TMJs may reveal them to be anterior to the articular eminence. In the face of trauma, radiographs should be taken to rule out fracture, since the clinical picture of both of these conditions is similar and similar occlusal disturbances are produced. Radiographs may not be necessary on an emergency basis if there is no history of trauma or if the condition is recurrent. [14]

### Technique for Reduction

If one appreciates the anatomy of the TMJ, the proper sequence for manual manipulation to reduce the dislocation is clear (see Fig. 69-18 (Figure Not Available) *D*). Following dislocation, the powerful masseter muscles may be in tremendous spasm. For a smooth reduction, it is mandatory to relieve the patient's pain and muscle spasm with slow IV injection of a benzodiazepine, with or without an opioid (see Chapter 35) . Some clinicians advocate direct injection of the condylar area with a local anesthetic (Fig. 69-19 (Figure Not Available) *A*). When the patient is sufficiently relaxed and analgesia is obtained, the physician faces the patient and grasps the mandible with both hands--one on each side--with the thumbs (which have been wrapped with gauze alone or tongue blades wrapped in gauze) facing the occlusal surfaces of the posterior teeth. The fingertips are placed around the inferior border of the mandible in the region of the angles. Some prefer to have the patient seated in a chair or on the floor with the patient's back against the wall. Alternatively, the patient may be in a recumbent position (Fig. 69-19 (Figure Not Available) *B* and *C*). Downward pressure is slowly and steadily applied to free the condyles from their anterior position to the eminence. The chin is then pressed backward after the jaw has been forced downward, and the mouth is closed while the condyle returns to its position in the fossa. In cases of severe muscle

spasm, the jaw may snap back quickly. Therefore, protection of the thumbs is essential (see Fig. 69-19) (Figure Not Available) .

Following the procedure, the patient should be instructed to stay on a soft diet for 1 week, to avoid wide opening of the mandible, and to take analgesics and muscle relaxants. Local heat may also provide relief. Chronic dislocators or patients who suffer acute recurrences may be helped by a bandage applied around the head holding the mandible to the maxilla for 2 weeks to prevent maximum opening, but this is rarely used. Very severe cases may require intermaxillary wiring and fixation for complete control with the use of Erich arch bars (described in Dental Alveolar Trauma). Patients who have suffered dislocation of the TMJ should be referred to an oral or maxillofacial surgeon for follow-up, since chronic dislocation may require a surgical alteration of the eminence for relief.

## Temporomandibular Myofascial Pain Dysfunction Syndrome

The temporomandibular myofascial pain dysfunction syndrome, or simply the TMJ syndrome, is a complex neuromuscular disturbance possibly resulting from and definitely aggravated by occlusal disturbances and disharmonies between occlusal relations and the anatomy of the TMJ. Other entities, such as trauma, psychologic tension, and neuromuscular habits (e.g., bruxism and clenching), contribute to the problem. Patients who present to the emergency unit frequently complain of unilateral facial pain. The pain is fairly nonspecific and is generalized to the region of the TMJ. The pain is of a dull nature in most patients and increases throughout the day with continued jaw motion. Clinical examination frequently reveals spasm of the masseter muscle externally and the internal pterygoid muscle intraorally. There is usually limitation of opening. Radiographs of the TMJ are usually normal unless there is an associated TMJ degenerative disease. When the diagnosis of TMJ syndrome is suspected, it is important for the emergency physician to rule out acute otalgia or pain of odontogenic origin. [15]

### Treatment

Severe trismus associated with TMJ syndrome may be treated by the emergency physician locally with the application of a refrigerant anesthetic spray, such as ethyl chloride, to the face. The skin of the patient's face around the masseter muscle at the angle of the mandible is covered with a light coating of petrolatum jelly. A towel is placed over the midline of the face to protect the region of the eye, and ethyl chloride is gently sprayed at the angle of the mandible in a rotary fashion from a distance of approximately 10 inches. The physician should take care not to produce frostbite of the tissues. The patient is instructed to open and close the mouth gently as the refrigerant spray is being applied. Frequently, this breaks the muscle spasm. Diazepam may also be of use. Physiotherapy is continued at home and consists of application of moist heat for 15 minutes 4 times a day. The patient should stay on a soft diet for approximately 2 weeks. Analgesics such as nonsteroidal anti-inflammatories, with or without muscle relaxants, are also effective.

Patients should be referred to a general dentist, a periodontist, or a periodontal

prosthodontist for follow-up and a course of therapy consisting of occlusal adjustment, if indicated. It is possible that some patients may have to be referred to an oral or maxillofacial surgeon for an intra-articular injection of cortisone or, rarely (in the most intractable cases), surgery (high intracapsular condylectomy). The various methods described here should always be attempted by a dentist before intra-articular injection of steroids is considered. Although useful, the intra-articular injection of steroids in the TMJ may be associated with a high incidence of subsequent fibrosis.

## CONCLUSION

Although patients having dental complaints do not represent a high percentage of emergency visits, individuals with dental emergencies frequently do present to the emergency unit. The most common procedures available to the emergency physician in the management of general dental emergencies have been described in detail in this chapter. Although many procedures, such as the stabilization of a reimplanted avulsed tooth, are more easily and perhaps better performed by a dentist or an oral or maxillofacial surgeon, in some cases this type of consultation may not be readily available for several days. Emergency physicians can render a great service to the patient in these cases by performing whatever stabilization is possible for the reimplanted tooth. The majority of procedures described in this chapter are useful for the emergency physician and should be performed whenever indicated. The emergency physician should feel free to call on dental colleagues for further demonstration and explanation when necessary.

# Chapter 70 - Vital Sign Measurement

**Jody Riva Lewinter, Thomas E. Terndrup, Terry M. Williams, Robert K. Knopp**

Documentation of temperature, pulse, and respiration is recommended for all emergency department (ED) patients, except for those with the most minimal of complaints. In patients with a pulse, blood pressure should generally be obtained. These measurements provide a unique, objective, capsule assessment of the patient's clinical condition. Vital signs are indicators of the severity of illness and may dictate the urgency of required intervention. Deteriorating vital signs are an important indicator of a deteriorating physiologic condition, whereas improving values provide reassurance that an unstable patient is responding to therapy. Hence, when a patient undergoes treatment over an extended time, selected vital signs, particularly previously abnormal ones, should be repeated. In some circumstances, the monitoring of selected vital signs should be continuous.

Vital signs should be measured and recorded at intervals dictated by the patient's clinical state (e.g., before and after fluid resuscitation, invasive procedures, or administration of medications with cardiopulmonary effects) or with any sudden change in the patient's clinical status. In addition, an abnormal vital sign can direct the physician toward a group of diagnoses or a particular organ system for further evaluation. An abnormal vital sign may constitute the patient's entire complaint, as in the febrile infant, or be the only indication of the potential for serious illness, as in the patient with resting tachycardia. For all of these reasons, accurate determination and interpretation of vital signs are mandatory.

In the ED, the accurate assessment and management of abnormal vital signs must reflect the priorities of resuscitation. Determination of airway patency with respiratory rate and pattern assumes first importance. Establishing the presence and quality of an arterial pulse is the second vital sign to be assessed, followed by blood pressure. Blood pressure and pulse are often evaluated in conjunction, as a measure of blood volume. Although body temperature is the last vital sign to be monitored during resuscitation, it has especial importance for patients suffering from thermal regulation failure (see Chapters 71 and 72) . This chapter is organized according to the priorities of patient resuscitation and evaluation.

Pulse oximetry and capillary refill are considered by some to represent additional "vital signs" in ED patients. The use of pulse oximetry is discussed in Chapter 6 . The assessment of capillary refill may be beneficial in children for determination of the adequacy of perfusion.

## BACKGROUND

Early pulmonary medicine was dominated by the concepts of Herophilus (4th century B.C.) and Galen (131 to 200 A.D.), whose belief in the humoral theory of medicine dictated that the lungs functioned as a cooling device and site for generation of body humors. The pulmonary circulation was correctly described in the 13th century by Ibnan-Nafis;

however, his observations passed unnoticed.

Respiratory physiology did not progress until the significance of the pulmonary circulation was recognized by Harvey in 1628. It was not until the 1700s that advances in physics and chemistry allowed the identification of the gases involved in respiration. [1]

Sphygmology, or palpation of the pulse, was first appreciated by Herophilus. He believed that interpreting the pulse required a knowledge of both music and geometry and defined the characteristics of the pulse as size, frequency, force, and rhythm. Chinese physicians (2nd century B.C.) timed the pulse by the respiratory rate of the examiner, believing that 4 pulsations per respiration was a normal rate for adults. The study of pulses was greatly influenced by Galen, who expanded the subject into a rather complex and obscure art form, writing no fewer than 18 books on the subject. [2]

Blood pressure was first measured directly in 1733 by Hales, who recorded the arterial pressures in a mare by cannulation with a brass pipe and a blood-filled glass column. [3] Frank used large-bore catheters connected to a rubber membrane in a 1903 manometer. [4] The invention of inflatable cuff manometers (Riva-Rocci, 1896) and the discovery of the arterial phase sounds (Korotkoff, 1905) allowed for the development of indirect blood pressure measurement. [3] [4]

The earliest recorded references to fever are from 6th century B.C. Akkadian cuneiform inscriptions, which appear to have adapted an ancient Sumerian icon of a flaming brazier to denote both fever and the local warmth of inflammation in a single ideogram. Clinical thermometry was introduced by Sanctorius in 1625. Mercury column thermometers were introduced by Fahrenheit in 1714. Although their routine use was supported by Boerhaave, thermometry

| TABLE 70-1 -- Normal Values for Vital Signs of Infants and Children (Mean ± SD) | | | | | |
|---|---|---|---|---|---|
| | Age | | | | |
| Parameter | 0-2 mo | 3-12 mo | 1-6 yr | 7-12 yr | 13-18 yr |
| Breaths/min | -- * | -- * | 24 ± 3 | 19 ± 2 | 17 ± 3 |

| | | | | | |
|---|---|---|---|---|---|
| Pulse/min | 126 ± 20 | 131 ± 20 | 88 ± 9 | 70 ± 8 | 64 ± 7 |
| Systolic BP | 72 ± 10 | 95 ± 15 | 93 ± 13 | 100 ± 10 | 112 ± 12 |
| Diastolic BP | 51 ± 9 | 53 ± 10 | 55 ± 10 | 63 ± 10 | 67 ± 10 |

* For data on children 0 to 35 months, see Table 70-2 (Table Not Available) .
As an estimate, for children 1 to 10 years: 2 × age (in years) + 90 mm Hg = 50th percentile for systolic BP.

was not established as routine clinical practice until the 1870s.

## NORMAL VALUES

The range of normal, resting vital signs for specific age groups must be recognized by the clinician to enable identification of abnormal values and their clinical significance. Normal ranges for vital signs also may be influenced by sex, race, pregnancy, and residence in an industrialized nation. These ranges have not been validated in ED patients, who may have many reasons for vital sign abnormalities that include anxiety, pain, and other forms of distress, in addition to altered physiology from disease states.

Published vital sign norms for children are not as well accepted as for adult patients. In Tables 70-1 and 70-2 (Table Not Available) , we report normal vital signs for children by age group as means and standard deviations. In Table 70-1 , the values for pulse and blood pressure for 0- to 2-month-olds are adapted from studies of newborn populations (i.e., <7 days). [6] [7] [8] During the newborn period, normal arterial blood pressure rises rapidly. Values for pulse and respiration in children older than 3 years reflect an average of male and female values for 0- to 1-, 3-, 9-, and 16-year-old populations. [9] The values for blood pressure reflect an average of male and female values for the 1- to 6-month and 3-, 9-, and 16-year-old populations. [8] Recent studies have reassessed reference values for respiratory rates in children. Table 70-2 (Table Not Available) reflects the age-related changes and the effect of state of wakefulness in the respiratory rates of children ages 0-3 years. [11] Hooker and colleagues measured resting respiratory rates in pediatric ED patients up to age 18 years. [10] They noted considerable patient variability and somewhat higher respiratory rates than are shown in Table 70-2 (Table Not Available) .

For the adult population, normal values for blood pressure are well established. Although there is an increase in systolic blood pressure with age, normotensive or normal systolic blood pressure is defined as 90 to 140 mm Hg, and normotensive or normal diastolic blood pressure is defined as 60 to 90 mm Hg.

The normal limits for resting heart rate of 60 beats/min and 100 beats/min were established by consensus in 1928 by the New York Heart Association. [13] Recent data indicate that 45 beats/min and 95 beats/min better define the heart rate limits of normal sinus rhythm in adults of all ages. Spodick recommends that the operational definition for the limits of resting heart rate in adults should be 50 beats/min and 90 beats/min. This view is widely supported among cardiologists. [15]

*There is currently no consensus on what constitutes a normal adult respiratory rate*. The majority of studies on respiratory rate support 16 to 24 breaths/min as the norm for adults.

Pregnancy results in alteration of the normal adult values for pulse and blood pressure. Respiratory rate is unchanged, although the physiologic hyperventilation of pregnancy is well recognized. This is a result of increased tidal volume and decreased residual and expiratory reserve volumes. [16] Resting pulse rate increases through pregnancy to 10 to 15% over baseline values. Norms for systolic and diastolic blood pressure are dependent on patient positioning. When the pregnant patient is sitting or standing, systolic pressures are essentially unchanged. Diastolic pressures decline until approximately 28 weeks' gestation, when they begin to rise to nonpregnant levels. When the pregnant patient is in the lateral decubitus position, both systolic and diastolic pressures decline until the 28th week and then begin to rise to nonpregnant levels (Table 70-3) (Table Not Available) .

## RESPIRATION

Respiratory frequency reveals only part of the clinical picture. The pattern, effort, and volume of respiration may be more indicative of altered respiratory physiology. An abnormality in respiration may be a primary complaint or a manifestation of other systemic disease.

### Physiology

Breathing is initiated and primarily controlled in the medullary respiratory center in the brainstem. The respiratory center is modulated by the pneumotaxic and apneustic centers in the pons. The pneumotaxic center serves as the limiter of length of the inspiratory signal and therefore can greatly increase or decrease respiratory rate.

In addition to being modified by other areas of the brainstem, the medullary respiratory center is modified by voluntary centers in the cerebral cortex; pulmonary stretch receptors of the airways; type J or juxtapulmonary capillary receptors of the pulmonary capillaries; arterial baroreceptors of the carotid sinus; and receptors found in skeletal muscle, tendons, and joints. Central and peripheral chemoreceptors also influence respiratory rate. [18]

### Indications and Contraindications

Generally, all ED patients should have respiratory rate documented during their evaluation. Repeated assessment and documentation of the patient's respiratory status is indicated in patients who present with an abnormal respiratory rate or a complaint referable to the airway or breathing.

The only contraindication to a careful measurement of respiratory rate is in the scenarios of respiratory distress, apnea, or upper airway obstruction that require immediate therapeutic intervention. A measurement of respiratory rate and effort should be performed as soon as patient care demands allow it in these circumstances.

Observation and palpation of chest movement are the techniques most frequently used to monitor respiratory frequency and amplitude noninvasively. Discussion of inductive plethysmography and other noninvasive monitors are beyond the scope of this chapter. Periodic manual measurements as described below generally suffice for ED patients.

## Procedure

Respiratory rate is the number of inspirations per minute. Generally, respiratory rate is best measured with the patient unaware that breathing is being observed, because awareness makes the patient conscious of the breathing pattern, which may alter the rate. Commonly, examiners count respirations while appearing to count the pulse. The respiratory rate is most accurately determined by counting for a full minute. Because the frequency is much less than the pulse, and breathing is less regular, an inaccurate measurement is more likely to occur if a 15-second interval is used.

Infants, in addition to being principally nasal breathers, are predominantly diaphragmatic breathers, and an infant's respiratory rate is easily determined by observing or palpating excursion of the chest or the abdominal wall. [19]

## Complications

There are no inherent complications from measuring respiration by observation. Problems related to the measurement of respiratory rate are generally due to failure to recognize a patient in obvious respiratory distress or failure to monitor respiratory rate in a patient who may be at risk for respiratory depression (e.g., in the case of sedative-hypnotic or narcotic overdose).

## Interpretation

### Respiratory Rate

A limited number of studies have examined respiratory rates. Hutchinson evaluated respiratory rates in 1897 healthy males at rest and found that 91% had respiratory rates between 16 and 24 breaths/min. He also noted that 30% had exactly 20 breaths/min. [20] Hooker and colleagues note that current texts vary considerably in their definitions of a

normal respiratory rate and cite published values that range from 8 to 20 breaths/min.

Hooker and associates, in the only study that specifically investigated normal respiratory rates in an ED, measured respiratory rates in 110 afebrile ambulatory patients without respiratory complaints (53 females and 57 males). [21] They report a mean rate of 20.1 breaths/min. For patients whose respiratory rate was measured again before release from the department, no significant difference was noted between the initial and subsequent respiratory rates. When analyzed by sex, females had a mean respiratory rate of 20.9 breaths/min and males had a mean respiratory rate of 19.4 breaths/min, a statistically significant difference. The researchers concluded that a normal respiratory rate in the adult patient population was 16 to 24 breaths/min. [21]

Recent studies, performed in young children, have provided additional information on normal resting and sleep state respiratory rates in children younger than 7 years. Respiratory rates obtained with a stethoscope were higher than those obtained by observation (mean difference, 2.6 breaths/min in awake and 1.8 breaths/min in asleep children). Smoothed percentile curves demonstrated a larger dispersion at birth (5th percentile, 34 breaths/min; 95th percentile, 68 breaths/min), while at 36 months of age (5th percentile, 18 breaths/min; 95th percentile, 30 breaths/min) dispersion was less.

Gravelyn and Weg compared changes in respiratory rates with respiratory dysfunction in 46 postoperative patients and found that 9 patients had respiratory rates >24 breaths/min. Of these, 5 patients had developed infection or congestive heart failure and 1 patient demonstrated an elevated respiratory rate in response to pain. In the remaining 35 patients, only 1 had respiratory dysfunction and a normal respiratory rate. [22] Bell and colleagues, using data from the Urokinase-Streptokinase Pulmonary Embolism Trials, found that 92% of patients with pulmonary emboli have a respiratory rate of >16 breaths/min. [23] Unfortunately, many normal patients have a resting respiratory rate of >16 breaths/min.

**Respiratory Pattern and Amplitude**

Abnormal respiratory patterns may be characteristic of metabolic or central nervous system pathologic conditions. Hyperventilation and hypoventilation may result from an extensive differential diagnosis including primary pulmonary disorders, such as pneumonia or chest wall pain. Respiratory disturbances also occur secondary to other disease processes. For example, *Kussmaul respiration* describes the hyperventilation pattern seen in diabetics with ketoacidosis. Abnormal respiratory patterns in adults can be used in differential diagnosis or in determining the location of central nervous system lesions.

Respiratory patterns in children must be observed carefully. In infants, periodic breathing, which may be normal, must be distinguished from apnea. By definition, periodic breathing consists of three or more respiratory pauses >3 seconds in duration, with <20 seconds between pauses. There is no associated bradycardia or cyanosis. This contrasts with apnea, which is a particular problem in preterm infants. Apnea is defined as a respiratory pause of >20 seconds, and it may be associated with bradycardia and hypoxia. [19] Periodic breathing and apnea are believed to be disorders

on a continuum, both stemming from abnormal physiologic control of respiration. However, periodic breathing is felt to be a benign disorder, whereas infants with symptomatic apneic episodes resulting in an apparent life-threatening event (ALTE) are thought to be at increased risk for sudden infant death syndrome.

# PULSE

The pulse is examined primarily to establish cardiac rate and rhythm. However, palpation of peripheral pulses yields clues to cardiac disease, such as aortic insufficiency, and information about the integrity of the peripheral vascular supply. Doppler ultrasound provides a noninvasive method of assessing blood flow in the ED. It has utility in the location of a pulse, in the assessment of fetal heart tones beyond the first trimester, for evaluation of peripheral lower extremity vascular insufficiency, and for the evaluation of blood pressure in infants or in patients with low-flow states.

### Physiology

Blood flowing into the aorta with each cardiac cycle initiates a pressure wave. Blood flows through the vasculature at approximately 0.5 m/sec; however, pressure waves in the aorta move at 3 to 5 m/sec. Therefore, palpated peripheral pulses represent pressure waves, not blood flow.

### Indications and Contraindications

The evaluation of pulse presence and rate is indicated in every patient who presents to the ED. The necessity of repeated evaluations is dictated by the clinical complaint and status of the patient. Pulse assessment is essential in all patients with potential peripheral vascular insufficiency.

No contraindications exist to assessment of pulse, but a few cautionary notes about the examination of the carotid pulse should be kept in mind: Concurrent bilateral carotid artery palpation should be avoided, as this maneuver could endanger cerebral blood flow. In addition, massage of the carotid sinus, found at the bifurcation of the external and internal carotid arteries at the level of the angle of the mandible, may result in reflex slowing of the heart rate (see Chapter 10) . To avoid inadvertent carotid sinus massage, the pulse should be palpated at or below the level of the thyroid cartilage. A risk of precipitating a cerebrovascular event by vigorous palpation of the carotid artery is present in adults with atherosclerotic disease. This risk may be minimized by prior auscultation of the carotid artery. If a bruit is present, avoid palpation of the carotid pulse in that patient.

### Equipment

Assessment of the pulse may be performed by the clinician at the bedside with any timepiece that has a second-hand display. This allows simultaneous assessment of all the characteristics of the pulse. If continuous monitoring is deemed necessary, bedside cardiac monitors can constantly monitor heart rate and rhythm. Pulse oximetry (see

, although primarily intended to measure oxygen saturation, also may be used to monitor the pulse rate. In a critical care situation, more sophisticated invasive monitoring techniques are available (see for arterial pressure measurement and rate assessment.

## Procedure

Pulses are palpable at numerous sites, although for convenience, the radial pulse at the wrist is routinely used. The examiner should use the tips of the first and second fingers to palpate the pulse. The two advantages to this technique are (1) that the fingertips are quite sensitive, enabling the pulse to be easily located and counted, and (2) the examiner's own pulse may be erroneously counted, if the thumb is used instead of the first and second fingers. Pulses are also easily palpated at the carotid, brachial, femoral, posterior tibial, and dorsalis pedis arteries. Palpation of the pulse at the brachial artery may facilitate the appreciation of pulse contour and amplitude. It is located at the medial aspect of the elbow and is more easily palpated when the elbow is held slightly flexed. [25] Pulse rate is ideally determined by counting the pulse for 1 minute, particularly if any abnormality is present.

In newborns, direct heart auscultation and umbilical palpation are the methods of choice to determine heart rate. Instantaneous changes in newborn heart rates are best indicated for the resuscitation team by the examiner tapping out each heartbeat. [26] In unstable children, palpation of central arteries, particularly femoral and brachial pulses, is recommended over palpation of more peripheral arteries.

## Interpretation

### Pulse Rate

Individual physiology must be considered in pulse interpretation. In infants and children, pulse rate must be interpreted with reference to age. Pulse varies with respiration, increasing with inspiration and slowing with expiration. This is known as a *sinus dysrhythmia* and is physiologic.

Although bradycardia is defined as a heart rate of <60 beats/min in adults, a well-conditioned athlete may have a normal resting heart rate of 30 to 40 beats/min. [27] As discussed earlier, a redefinition of bradycardia to <50 beats/min and tachycardia to >90 beats/min has been proposed.

The clinician must consider whether an abnormal pulse rate is a primary or secondary condition. The examination of the entire set of vital signs is instrumental in discerning the cause for the abnormal rate. For example, hyperthermia causes a sinus tachycardia. An increase in heart rate of 18 beats/min can be expected for each degree centigrade and 10 beats/min for each degree Fahrenheit of elevated temperature. [17] Drug fever, typhoid fever, and central neurogenic fever are suggested when no corresponding tachycardia is found in a patient with elevated body temperature. Hypothermia, with its

reduced metabolic demands, may be associated with bradycardia.

Clinical evaluation of the patient with an abnormal pulse rate dictates a consideration of medications the patient may be taking or the presence of a mechanical pacemaker. Digitalis compounds, beta-blockers, and antidysrhythmics may alter the normal heart rate and the ability of this vital sign to respond to a new physiologic stress. These cardioactive medications may be the cause of the patient's heart rate abnormality.

**Heart Rhythm**

In addition to determining the pulse rate, information about the regularity of the pulse is obtained during palpation. An irregularly irregular pulse suggests atrial fibrillation or flutter with variable block, and accurate assessment of the pulse should be obtained by auscultation of apical cardiac sounds. The apical pulse is frequently greater than the peripheral pulse, reflecting inadequate filling time and stroke volume, with resultant nontransmitted beats. A greater pulse deficit generally reflects more severe disease.

**Pulse Amplitude and Contour**

Amplitude and contour of the pulse are generally assessed simultaneously. Figure 70-1 (Figure Not Available) compares normal and abnormal pulse amplitudes and contours. Accurate examination and description provides additional clinical information. Superimposition of one pathophysiologic state on another may modify the pulse. For example, sepsis may manifest with variable pulse amplitude, depending on the stage in the development of the disease at which the patient presents. Early in sepsis, cardiac output increases and vascular resistance decreases, causing bounding pulses. In advanced sepsis or septic shock, falling cardiac output and increased vascular resistance are seen, and pulses are diminished. [28]

**Pulses During Cardiopulmonary Resuscitation**

Palpated "femoral pulses" during chest compression may represent either forward arterial blood flow or "to-and-fro" movement of blood from the right heart to the venous system. A carotid pulse is preferred when assessing the adequacy of chest compressions during cardiopulmonary resuscitation (see Chapter 16) .

# ARTERIAL BLOOD PRESSURE

Changes in arterial blood pressure over time may be an indication of the success of treatment or the worsening of the patient's overall condition. An abrupt reduction in the patient's arterial blood pressure is usually an indication of the need for immediate intervention or reconsideration of therapy. The technique of arterial cannulation and direct intra-arterial blood pressure monitoring is discussed in Chapter 19 . This section discusses indirect blood pressure monitoring. Discussions of the specific use of the Doppler device for pulse and blood pressure measurement and the measurement of orthostatic blood pressure and pulse changes follow this section.

## Physiology

The arterial blood pressure indicates the overall state of hemodynamic interaction between cardiac output and peripheral vascular resistance. The arterial blood pressure is the lateral pressure or force exerted by the blood on the vessel wall. The arterial blood pressure indirectly measures perfusion, where blood flow equals the change in pressure divided by resistance. [4] However, because peripheral vascular resistance varies, a normal blood pressure does not confirm adequate perfusion. [29] Mean arterial blood pressure can be estimated by adding one third of the pulse pressure (i.e., the difference between the systolic and diastolic blood pressure) to the diastolic pressure.

## Indications and Contraindications

Blood pressure measurement and documentation are essential for most ED patients seen for the first time. Patients with minor ambulatory complaints not related to the cardiovascular system may not receive blood pressure measurements in the ED. Frequent monitoring of the blood pressure is necessary for patients with hemodynamic instability.

In low-flow states, Doppler measurement of blood pressure may be obtained rapidly. Repeated measurements will provide an evaluation of the adequacy of resuscitation in patients whose blood pressure cannot be auscultated by standard techniques. Placement of a catheter for direct intra-arterial measurement of blood pressure has a higher risk of complications, but it may be performed safely in the ED (see Chapter 19). In particular, direct measurement of arterial pressure during pulseless electrical rhythms may help to discriminate between severe shock and an otherwise nonresuscitatable status. [31]

Relative contraindications to specific extremity blood pressure measurement include an arteriovenous fistula, ipsilateral mastectomy, axillary lymphadenopathy, lymphedema, and circumferential burns over the intended site of cuff application.

## Equipment

The equipment required for indirect blood pressure measurement includes a sphygmomanometer (cuff with inflatable bladder, inflating bulb, controlled exhaust for deflation, and manometer) and a stethoscope or Doppler device (for auscultation) or an oscillometric device. To ensure an accurate reading, the sphygmomanometer cuff should be of an appropriate size for the patient. The width of the bladder should be 40% of the distance of the midpoint of the limb (i.e., from the acromion process to the lateral epicondyle). The length of the bladder should be 80% of the midarm circumference or twice the recommended width. [30]

Manometers in common use are either an aneroid or a mercury gravity column. Both types of manometers are convenient for bedside use, although the mercury gravity column must be placed vertically to ensure accurate measurements. An aneroid

manometer uses a metal bellows that elongates with the application of pressure. This elongation is mechanically amplified, transmitting the motion to the indicator needle.

Manometers require annual servicing. Mercury columns may require the addition of mercury to bring the edge of the meniscus to the zero mark. The air vent or filter at the top of the mercury column also should be checked for clogging. The aneroid manometer should be calibrated against a mercury column at least yearly. If the aneroid indicator is not at zero at rest, the device should not be used. [35]

Automatic sphygmomanometers may improve physiologic monitoring with alarm and self-cycling capabilities. They offer indirect arterial blood pressure measurement with little pain and lack the risks associated with invasive arterial lines. [36] Various manufacturers use oscillometric (Dinamap 845, Applied Medical Research), Korotkoff sound (Pressurometer [Avionics]; Diasyst-T [Siemens Co]), and ultrasonic (Arteriosonde, Hoffmann-LaRoche Co) techniques. Oscillometric blood pressure monitors detect motion of the blood pressure cuff transmitted from the underlying artery. A sudden increase in the amplitude of arterial oscillations occurs at systolic pressure and mean arterial pressure, and an abrupt decrease occurs at diastolic pressure.

Dinamap blood pressures obtained in 29 hemodynamically stable children (mean age, 18 months) demonstrated a smaller mean error (systolic, -0.24 vs 3.26 mm Hg) than auscultatory measurements in 20 children (systolic, -1.65 vs 6.68 mm Hg) using direct radial arterial pressures for comparison. [37] Park and Menard also found less variability with the Dinamap vs the auscultatory method in children. [38] Similarly, in adult patients, good correlation was found with mean diastolic and systolic arterial pressures using Dinamap. [39] Automatic devices do not provide a continual monitoring of blood pressure, and the static digital readouts may fail to pick up sudden hypotension.

**Procedure**

Indirect blood pressure measurements may be obtained at the patient's bedside by palpation, auscultation, Doppler, or oscillometric methods. The technique is straightforward and accurate when well-maintained, calibrated equipment is used by practitioners who follow accepted standards. The patient may be lying or sitting, as long as the site of measurement is at the level of the right atrium and the arm is supported.

Palpation of arterial blood pressure requires cuff inflation to 30 mm Hg above the level at which a palpable pulse disappears. Once properly inflated, the examiner should palpate directly over the artery and deflate the cuff at 2 to 3 mm Hg per second. The initial appearance of arterial pulsations is reported as the palpable blood pressure. The same technique may be used with the Doppler device with the Doppler auditory signal replacing the palpated pulse. Arterial pressure measurement by palpation and Doppler yields only systolic blood pressure estimates.

When auscultating the blood pressure at the brachial artery, the blood pressure cuff is applied about 2.5 cm above the antecubital fossa with the center of the bladder over the artery. [32] The bell of the stethoscope is applied directly over the brachial artery but with as little pressure as possible. [40] The systolic arterial blood pressure is defined as the

first appearance of faint, clear, tapping sounds that gradually increase in intensity (Korotkoff phase I), whereas the diastolic blood pressure is defined as the point at which sounds disappear (Korotkoff phase V). In children, phase IV defines the diastolic blood pressure (Fig. 70-2) (Figure Not Available) . [8] Phase IV is marked by a distinct, abrupt muffling of sound when a soft, blowing quality is heard. Measurement by auscultation over the brachial artery is preferred because of accepted standardization of measured values. Alternate sites include the radial, popliteal, posterior tibial, or dorsalis pedis arteries, although any fully compressible extremity artery may be used. Studies correlating direct and indirect blood pressure measurements have demonstrated a good correlation between these methods.

The accuracy of palpatory, Doppler, and oscillometric methods has also been demonstrated. However, when phase I and V Korotkoff sounds are used, indirect methods typically underestimate systolic and diastolic pressure by several millimeters of mercury. [41] [48] In addition, during shock, palpatory and auscultatory methods underestimate simultaneous direct arterial pressure measurements. [49] The flush method, in which return of color after deflation of the cuff is used for estimating blood pressure in infants, may underestimate systolic blood pressure by up to 40 mm Hg. [48] This method is unreliable and is not recommended.

**Complications**

Complications of indirect blood pressure measurement are minimal when the proper procedure is followed. Inadvertent prolonged application of an inflated blood pressure cuff may result in false elevation of diastolic pressure and in ischemia distal to the site of application, with attendant complications. Invasive blood pressure monitoring is associated with a number of potential problems (see Chapter 19) .

**Interpretation**

Normal blood pressure increases with decreasing distance from the aorta. The blood pressure tends to increase with age and is generally higher in males. Individual factors that influence blood pressure include body posture, emotional or painful stimuli, environmental influences, vasoactive foods or medications, and the state of muscular and cerebral activity. Exercise and sustained isometric muscular contraction increase blood pressure in proportion to the strength of the contraction. [4] A normal diurnal pattern of blood pressure consists of an increase throughout the day, with a significant, rapid decline during early, deep sleep. [50]

Normal lower limits for systolic blood pressure for infants and children can be estimated by adding 2 times the age (in years) to 70, with the result expressed in millimeters of mercury. The 50th percentile for a child's systolic arterial blood pressure from 1 to 10 years of age can be estimated by adding 2 times the age (in years) to 90 mm Hg. Children older than 2 years are considered hypotensive when systolic blood pressure is <80 mm Hg. [51] Children, in particular, are able to maintain mean arterial blood pressure until very late during shock. [52] Thus the finding of a normal blood pressure in a child with signs of poor perfusion should not dissuade the emergency physician from appropriate treatment. Adults are considered hypotensive if the systolic blood pressure

is <90 mm Hg. When accompanied by signs of shock, immediate treatment is indicated. In patients with shock, blood flow cannot be reliably inferred from heart rate and blood pressure values.

**Hypertension**

Adults are hypertensive if either the systolic or the diastolic pressure consistently exceeds 140 or 90 mm Hg, respectively. [55] [56] The applicability of population norms for hypertension in a stressful emergency situation is controversial. One should not make diagnostic or therapeutic decisions based solely on an abnormal initial measurement. Patients with hypertension require repeat measurement to assess whether ED therapy is required. Because sustained hypertension may be seen in more than a third of initially hypertensive ED patients, careful evaluation and follow-up are required. [57]

One study of 48 hospitalized patients found that during a physician visit, 94% experienced a mean peak increase in systolic pressure (27 ± 2 mm Hg), diastolic pressure (15 ± 2 mm Hg), and heart rate (16 ± 2 beats/min). The response peaked at approximately 4 minutes and abated at about 10 minutes after initiation. [58] By using noninvasive, ambulatory blood pressure recordings, investigators at New York Hospital found that 21% of 292 patients had "white coat" hypertension. These patients were (1) more likely to be recently diagnosed as hypertensive, (2) female, (3) younger, and (4) to have had their office blood pressure measured by a physician. [59]

Recent ED data indicate that patients with stroke have spontaneous reductions in blood pressure during the first 90 minutes after onset of symptoms (mean change in systolic and diastolic readings, -29 mm Hg and -10 mm Hg, respectively). [60]

**Measurement Errors**

Erroneous blood pressure measurements may result from several factors.  Falsely low blood pressure may be caused by using an overly wide cuff or by rapid cuff deflation. [62]  Falsely high blood pressure may be caused by the use of an overly narrow cuff, anxiety, pain, tobacco use, exertion, an unsupported arm, or slow inflation of the cuff.

In 470 unselected adults, investigators at Duke University found no spurious effect of cuff size in 350 patients weighing <95 kg and with an arm circumference <35 cm. However, in 120 patients weighing >95 kg and with an arm circumference >35 cm, the use of a large cuff reclassified 33% of those with systolic hypertension to borderline, 62% of those with borderline systolic hypertension to normal, and 79% of patients with borderline diastolic hypertension to normal. [63] Of note, 41% of adults observed at the University of Pittsburgh required nonstandard size cuffs, and use of small cuffs was associated with a mean error of 8.5 and 4.6 mm Hg in the systolic and diastolic pressures, respectively.  Hypotensive patients have unreliable Korotkoff sounds. However, Doppler measurements are well correlated with direct arterial systolic pressure in hypotensive patients. [65]

An auscultatory gap can be appreciated in hypertensive patients and may mislead the

examiner. It is heard during the latter part of phase I and should not be confused with diastolic readings. Auscultation until the manometer reading approaches zero should prevent misinterpretation. In patients with aortic insufficiency or hyperthyroidism, in persons who have just finished exercising, and in children younger than 5 years of age, the measurement of diastolic blood pressure should occur at Korotkoff phase IV.

Irregular heart rates also may interfere with accurate blood pressure determination. A second or third reading, with 2 minutes of deflation between recordings, should be used to obtain an average when premature contractions or atrial fibrillation is present. Ultrasonic methods may be more accurate during shock states and in infants for systolic blood pressure measurement. [65]

Dewar demonstrated that hemiplegic patients may exhibit different blood pressures in the affected and unaffected arm. [66] A flaccid extremity tended to yield lower systolic and diastolic pressures, whereas a spastic extremity tended to yield higher values than the extremity with normal motor tone. Although these differences are generally small, it is preferable to monitor blood pressure in the unaffected limb.

**Pulse Pressure**

Increased pulse pressure (i.e., 60 mm Hg) is commonly observed in anemia, exercise, hyperthyroidism, arteriovenous fistula, aortic regurgitation, and patent ductus arteriosus. A narrowed pulse pressure (20 mm Hg) may be a manifestation of hypovolemia, increased peripheral vascular resistance, or decreased stroke volume.

**Differential Brachial Artery Pressures**

A difference in bilateral brachial artery pressures up to 10 mm Hg may be normal, but it is uncommon. Hashimoto and colleagues found that only 1.4% of elderly patients had a systolic brachial blood pressure difference of >10 mm Hg, although 6.5% had a difference exceeding 7.5 mm Hg. [67] Most patients with subclavian steal syndrome, supravalvular aortic stenosis, and aortic dissection have a 15 mm Hg difference in contralateral brachial artery systolic pressures.

**Pulsus Paradoxus**

Normal respiration decreases the systolic blood pressure by approximately 10 mm Hg during inspiration. Pulsus paradoxus occurs when there is a >12 mm Hg decrease in the systolic blood pressure during inspiration. Pulsus paradoxus may occur in patients with chronic obstructive pulmonary disease, pneumothorax, severe asthma, and pericardial tamponade. [68]

To measure a paradoxical pulse, the patient should be lying comfortably, at a 30° to 45° angle, and breathing normally, in an unlabored fashion (unusual conditions in a patient suspected of cardiac tamponade). [69] The blood pressure cuff is inflated well above systolic pressure and is slowly deflated until one first hears the systolic sounds that are synchronous with expiration (Fig. 70-3) . Initially, one will hear the arterial pulse only

during expiration, and it will disappear during inspiration. The cuff is then further deflated until arterial sounds are heard throughout the respiratory cycle. A paradoxical pulse can be palpated if it is very large. During palpation the pulse may completely disappear during inspiration. When present, this technique is a quick bedside confirmation of the possibility of severe tamponade. Palpation for this purpose is best done at peripheral arteries, such as the radial or femoral.

An alternative approach to measurement of pulsus paradoxus is to use a finger arterial pressure monitor (Finapres; Ohmeda, Englewood, Colo) and to subtract the peak systolic blood pressure during expiration from the lowest systolic blood pressure during inspiration. [70] The pulsus paradoxus obtained with this technique was found to have less variability (when compared to intra-arterial measurements) than with manual measurements. Furthermore, pulsus paradoxus obtained using the finger pressure monitor correlates well with the pulmonary index score in asthmatic children. [71] Changes in the pulsus paradoxus were found to correlate with other markers of clinical status and admission decisions.

If the difference between these inspiratory and expiratory pressures is >12 mm Hg, the paradoxical pulse is high. [72] Most patients with proven tamponade have a difference of 20 to 30 mm Hg during the respiratory cycle. This may not be true of patients with very narrow pulse pressures (typical of advanced tamponade), who have a "deceptively small" paradoxical pulse of 5 to 15 mm Hg. The relative decrease in pulsus paradoxus occurs because the paradoxical pulse is a function of actual pulse pressure, and the inspiratory systolic pressure may be below the level at which diastolic sounds disappear. [69] For this reason, the ratio of paradoxical pulse to the pulse pressure is a more reliable measure. A paradoxical pulse >50% of the pulse pressure is abnormal. [69]

Pulsus paradoxus has been correlated with the amount of impairment of cardiac output by tamponade. In atraumatic patients, a 15% pulsus paradoxus in the presence of relative hypotension was found in 97% of patients with moderate or severe tamponade and in only 6% of patients with absent or mild tamponade. A similar study of right ventricular diastolic collapse by echocardiography found that an abnormal pulsus paradoxus had a sensitivity of 79%, specificity of 40%, positive predictive value of 81%, and negative predictive value of 40%. [75] The absence of a paradoxical pulse does not rule out tamponade (see Chapter 15) .

**Shock Index**

The ratio of the pulse rate over the systolic blood pressure has been suggested as a measure of clinical shock. The shock index (SI) has a normal range of 0.5 to 0.7. Rady and colleagues demonstrated that an SI of >0.9 was more commonly associated with acute illness requiring immediate treatment (56% vs 45%) or hospital admission (85% vs 45%) than was an SI of <0.9. [76] However, the presenting pulse rate alone had nearly the same predictive power. Further, Rady and coworkers demonstrated that although the SI appeared to correlate with left ventricular stroke work index, it had little correlation with systemic oxygen transport in hemorrhagic and septic shock.

# DOPPLER ULTRASOUND FOR EVALUATION OF PULSE AND BLOOD

# PRESSURE

## Principles of Doppler Ultrasound

Doppler ultrasound is based on the Doppler phenomenon: the frequency of sound waves varies depending on the speed of the sound transmitter in relation to the sound receiver. Doppler devices transmit a sound wave that is reflected by flowing erythrocytes, and the shift in frequency is detected. Frequency shift can only be detected for blood flow >6 cm/sec.

### Indications and Contraindications

Doppler ultrasound is commonly used in the ED for the measurement of blood pressure in low-flow states, evaluation of lower extremity peripheral perfusion, and assessment of fetal heart sounds after the first trimester of pregnancy. Doppler sensitivity allows the detection of systolic blood pressure down to 30 mm Hg in the evaluation of a patient in shock. In the patient with peripheral vascular disease in whom there is concern about the adequacy of peripheral perfusion, the ankle/brachial index provides a rapidly obtainable, reproducible, and standardized assessment. [78] Fetal heart sounds provide a baseline assessment of any patient with 12 weeks' gestation in whom there is possible abdominal trauma or fetal distress due to a complication of pregnancy.

The use of Doppler ultrasound in the evaluation of deep venous thrombosis is a valuable tool; however, it requires specific training and experience to attain proficiency. Discussion of this topic is beyond the scope of this chapter.

### Equipment

A nondirectional Doppler device has a probe that houses 2 piezoelectric crystals. One crystal transmits the signal and the other receives it. Reflected signals are converted to an electrical signal and fed to an output that transforms them to an audible sound. Two commonly used Doppler units are the pocket Doppler stethoscope (model BF4A, Medsonics, Inc, Los Altos, Calif) and the ultrasonic Doppler flow detector (model 811, Parks Medical Electronics, Aloha, Ore) (Figs. 70-4 (Figure Not Available) and 70-5) (Figure Not Available) .

Probes with a frequency of 2 to 5 MHz are best for detecting fetal heart sounds. Frequencies of 5 to 10 MHz are appropriate for limb arteries and veins. The probes should be monitored periodically for electrical damage and integrity of the crystal. Sphygmomanometers used in conjunction with the Doppler device should be calibrated periodically, as described in the section on blood pressure evaluation.

### Procedure

The Doppler probe is placed against the skin using an acoustic gel as an interface. The gel ensures optimal ultrasound signal transmission and reception and protects the crystals. In an emergency, water-soluble lubricant (e.g., Surgilube or K-Y jelly) may be

substituted for commercial acoustic gel. The probe is angled at 45° along the length of the vessel to optimize frequency shifts and signal amplitude.

In the evaluation of peripheral perfusion, a sphygmomanometer cuff is placed proximal to the arterial pulse and inflated. The probe is placed over the arterial pulse and the cuff is slowly deflated. The pressure at which flow is first heard is the systolic pressure.

In the evaluation of peripheral vascular disease, the ankle/brachial index is determined. Both brachial arteries are examined at the medial aspect of the antecubital fossa. The probe is angled until the most satisfactory signal is obtained. The cuff is inflated and slowly deflated until the systolic pulse is heard. The procedure is repeated for the posterior tibial and dorsalis arteries of both lower extremities.

In the evaluation of fetal heart tones, variable positioning of the fetus may require examination at several locations over the uterus and angling of the probe to search for the optimal signal. It is best to begin in the mid-suprapubic area and to explore the uterus via angulation of the probe. Once tones are located, the probe can be moved along the abdomen to reach a position closer to the origin of the sound. Fetal heart tones are distinguished from placental flow by the discrete quality of the fetal heart tones and the rate of placental flow, which matches the maternal pulse.

**Interpretation**

As noted earlier, in low-flow states, Doppler ultrasound can detect a blood pressure as low as 30 mm Hg. The ankle/brachial index of each limb is calculated by dividing the higher systolic pressure of the posterior tibial or the dorsalis pedis artery of the limb by the higher of the systolic pressures in the brachial arteries. In normal individuals, the index should be >1.0. Patients with claudication have values between 0.6 and 0.8. Values <0.5 imply severe impairment and are consistent with rest pain or gangrene. [78] Patients with ankle/brachial index values of 0.9 have been found to have increased cardiovascular morbidity and mortality. [79] Segmental lower extremity pressure measurements may help to identify the level of obstruction (Figs. 70-6 (Figure Not Available) through 70-8). [80] Obese patients, diabetic patients, or patients with calcified vessels that are not compressible may have abnormally high systolic pressures (e.g., 250 to 300 mm Hg) and indices that do not accurately reflect flow.

Normal fetal heart tones should be between 120 and 140 beats/min. Fetal tones may be heard as early as the 12th week of gestation.

# ORTHOSTATIC VITAL SIGNS MEASUREMENT

Orthostatic vital signs are used to evaluate patients with fluid loss, hemorrhage, syncope, or autonomic dysfunction. They are also used to assess the patient's response to therapy. The emergency physician is often concerned with the accurate detection of acute blood loss or volume depletion. When the clinical syndrome of shock exists, assessment of a blood volume deficit poses little difficulty. It is preferable, however, that volume loss be detected before loss of physiologic compensation and clinical shock

occurs. This section addresses the utility of orthostatic vital signs in the detection and monitoring of acute volume depletion.

Many techniques have been advocated to assess volume status. Unfortunately, most of these procedures lack a database against which to judge their reliability. Methods that have been recommended include evaluation of the following parameters: skin color; skin turgor; skin temperature; supine, serial, and orthostatic vital signs; neck vein status; transcutaneous oximetry; and hemodynamic monitoring (e.g., monitoring of central venous pressure) (see Chapter 24) . Serial vital sign measurements have been used for assessing blood loss, but they do not reliably detect small degrees of blood loss. *Up to 15% of the total blood volume can be lost with minimal hemodynamic changes or any alteration of the supine vital signs.* [81] A decrease in the pulse pressure occurs with acute blood loss, [83] but the patient's baseline blood pressure values are often unknown. Clinical examination of neck veins adds useful information but is less precise than measurement of central venous pressure. Most clinicians use skin color, temperature, and moisture as a reflection of skin perfusion and sympathetic tone but not as an accurate guide to circulatory volume, because the vasomotor tone of the skin is affected by numerous diseases as well as by emotional and environmental factors. Capillary refill has been advocated as an ideal noninvasive test for hypovolemia, but it has not been found to be accurate in adults [84] (see the following discussion regarding its use for children).

The ideal test for determining volume status would rapidly and accurately detect volume depletion of 5% or more using a noninvasive technique. At present, no such test exists. Orthostatic vital signs meet the criteria of being noninvasive and easily used at the bedside. However, *in patients with acute blood loss of <20% of total blood volume, orthostatic vital signs lack both sensitivity and specificity.* [85] Further, ethanol ingestion exaggerates postural pulse changes, thus mimicking the hemodynamic changes seen with acute blood loss. [86]

The medical literature is replete with unsubstantiated claims regarding what constitutes a positive or negative orthostatic test, and its value for estimating volume status may be overstated. Some authors [87] [88] have stated that postural hypotension or postural tachycardia occurs with varying degrees of hypovolemia, but they do not define specific criteria for a positive test. Other sources (without documentation) have perpetuated the notion that relatively small changes in the orthostatic blood pressure or the pulse are reliable in detecting hypovolemia. For example, Hayes and Briggs state erroneously that "a decrease of 10 mm Hg or more on assuming the sitting position indicates significant hypovolemia." [89]

In this section, the physiologic compensatory mechanisms that are activated by hypovolemia and postural tilting and the clinical use of orthostatic vital signs to detect acute volume loss are discussed.

**Physiologic Response to Hypovolemia**

Acute blood loss decreases the pressure gradient between the venules and the right atrium. A fall in this pressure gradient decreases venous return. [90] As a result, cardiac

output falls, and clinical manifestations of shock ensue. Several homeostatic mechanisms are initiated by acute blood loss (Table 70-4) . The dominant compensatory mechanism in shock is a reduction in the carotid sinus baroreceptor inhibition of sympathetic outflow to the cardiovascular system. This increased sympathetic outflow results in several effects: (1) an arteriolar vasoconstriction, which greatly increases total peripheral vascular resistance; (2) a constriction of venous capacitance vessels, thereby increasing venous return to the heart; and (3) an increase of heart rate and force of contraction, which helps to maintain cardiac output despite significant volume loss. [91] These sympathetic reflexes are geared more for the maintenance of arterial pressure than for the maintenance of cardiac output (Fig. 70-9) (Figure Not Available) . The value of

| **TABLE 70-4** -- Homeostatic Mechanisms in Hemorrhagic Shock |
|---|
| Sympathetic reflex compensation |
|   Arteriolar vasoconstriction |
|   Venous capacitance vasoconstriction |
|   Increased inotropic and chronotropic cardiac activity |
|   Central nervous system ischemic response |
| Selective increase in cerebral and coronary perfusion by means of local autoregulation |
| Increased oxygen unloading in tissues |
| Restoration of blood volume |

| Renin-angiotensin-aldosterone axis activation |
| --- |
| Antidiuretic hormone secretion |
| Transcapillary refill |
| Increased thirst resulting in increased fluid intake |
| Increased erythropoiesis |

sympathetic reflex compensation is illustrated by the fact that 30 to 40% of the blood volume can be lost before death occurs while these reflexes are intact. When the sympathetic reflexes are absent, loss of only 15 to 20% of the blood volume may cause death. [91]

Several other reflexes maintain cardiac output in the presence of volume loss. The central nervous system ischemic response stimulates the sympathetic nervous system after the arterial pressure falls below 50 mm Hg and is responsible for the second plateau on the arterial pressure curve (see Fig. 70-9) (Figure Not Available) . [91] Other compensatory mechanisms that tend to restore the blood volume to a normal level include the release of angiotensin and antidiuretic hormone, which cause arteriolar vasoconstriction and conservation of salt and water by the kidneys. [92] A fluid shift from the interstitium to the intravascular space occurs that helps to restore blood volume over a longer period (1 to 40 hours).

When blood loss results in anemia, part of the loss in oxygen-carrying capacity is countered by an increase in tissue oxygen extraction. [95] Finally, the lost red blood cell mass is slowly replaced by erythropoiesis.

Several investigators have examined the changes in blood pressure and pulse that occur in the supine patient with blood loss. Collectively these studies have shown variable individual hemodynamic responses to acute blood loss of up to 1 L. The frequent inability to detect significant volume loss with supine vital signs and the observation that patients with acute volume loss frequently develop syncope on arising led to the investigation of the use of orthostatic vital signs to detect occult hypovolemia.

**Physiologic Response to Postural Changes**

When an individual assumes the upright posture, complex homeostatic mechanisms compensate for the effects of gravity on the circulation to maintain cerebral perfusion. These responses include (1) baroreceptor-mediated arteriolar vasoconstriction, (2) venous constriction and increased muscle tone in the legs and the abdomen to augment venous return, (3) sympathetic-mediated inotropic and chronotropic effects on the heart, and (4) activation of the renin-angiotensin-aldosterone system. [96]

These compensatory mechanisms preserve cerebral perfusion in the upright position with minimal changes in vital signs. Currens found that when normal subjects stand, the pulse increases by an average of 13 beats/min, no change or a small drop is seen in systolic blood pressure, and either no change or a small rise occurs in diastolic blood pressure. [97] These changes have been confirmed by others.

In patients with *vasodepressor syncope*, the normal compensatory reflexes that preserve cerebral perfusion with postural changes are altered. The normal increased sympathetic tone on standing is paradoxically inhibited, and an exaggerated enhancement of parasympathetic activity (bradycardia) occurs, which can lead to syncope. [100]

Few data exist on the effect of acute blood loss on postural vital signs. One early study [83] looked at 23 young adult volunteers from whom 500 to 1200 mL of blood was withdrawn. They found no reliable change in the postural blood pressure, but a consistent postural increase in the pulse of 35 to 40% was noted after a 500-mL blood loss. In the 6 subjects who were bled approximately 1 L, only 2 were able to tolerate standing; each of them had a postural increase in pulse of >30 beats/min. The other four subjects experienced severe symptoms on standing, followed by a marked bradycardia and syncope if they were not allowed to lie down.

Knopp and colleagues [85] phlebotomized 450 to 1000 mL of blood from healthy volunteers. By using the criterion of a pulse increase of 30 beats/min or the presence of severe symptoms (syncope or near syncope) during a supine-to-standing test, they were able to distinguish accurately between a 1000-mL blood loss and no blood loss. Changes in blood pressure and pulse were not evaluated in the symptomatic subjects. In this study population of 100 normal healthy volunteers with acute blood loss, the sensitivity and specificity using the aforementioned criteria for detecting a 1000-mL blood loss (Table 70-5) were both 98%, giving an accuracy of 96% (2% false-negative results and 2% false-positive results). The investigators were unable to consistently detect a blood loss of 500 mL by using these criteria.

**Variables Affecting Orthostatic Vital Signs**

Many conditions affect the compensatory mechanisms that allow us to assume the upright posture (Table 70-6) (Table Not Available) . [96] Because of decreased vasomotor tone, limited chronotropic response, and other factors, the elderly have a

higher incidence of orthostatic hypotension, which can lead to syncope

| |
|---|
| **TABLE 70-5** -- Summary of Orthostatic Tilt Testing [*] |
| **Test Procedure** |
| 1. Blood pressure and pulse are recorded after patient has been supine for 2-3 min |
| 2. Blood pressure, pulse, and symptoms are recorded after patient has been standing for 1 min; the patient should be permitted to resume a supine position immediately should syncope or near-syncope develop |
| **Positive Test** |
| 1. Increase in pulse of 30 beats/min or more in adults, *or* |
| 2. Presence of symptoms of cerebral hypoperfusion (e.g., dizziness, syncope) |

* The predictive ability of orthostatic vital signs to assess volume status is often overestimated in clinical practice. This suggested guide is based on the ability of the pulse change and patient symptoms to distinguish between no acute blood loss and a 1000 mL acute blood loss in healthy, previously normovolemic volunteers (sensitivity of 98% for detecting 1000 mL acute blood loss). [85]This guide may not be applicable to elderly patients, sick children, medicated patients, and those with autonomic dysfunction.

and fall-related injury. [101] One should note that drugs that antagonize the normal autonomic compensatory mechanisms can also produce orthostatic changes. These changes can be severe enough to produce frank syncope, especially in the elderly. However, in one study of *euvolemic* adult volunteers, orthostatic changes in patients with diabetes or in those using various antihypertensive agents were similar to changes in normal adults. [102]

Even in normal subjects, passive tilting generates a high incidence of orthostatic syncope. [103] Patients with chronic anemia (and a compensated blood volume) seem to have the same postural response as normal subjects. [104] Most of the conditions that

affect postural blood pressure regulation involve a pathologic condition that affects the sympathetic nervous system. Orthostatic hypotension caused by autonomic insufficiency is usually not accompanied by tachycardia, whereas the orthostatic hypotension produced by acute volume depletion is commonly accompanied by a pronounced reflex tachycardia.

As noted in Table 70-6 (Table Not Available) , many conditions, diseases, and medications have been implicated as a cause of abnormal orthostatic vital sign changes. Most variables have been poorly studied. In the elderly, orthostatic vital sign changes in *volume-depleted* patients have not been studied. In one study of *normovolemic* nursing home patients 62 years or older, 39 of 476 (8%) had a drop in systolic blood pressure of >20 mm Hg along with a drop in diastolic blood pressure of >10 mm Hg. [105] As expected, this postural blood pressure change was more common in patients taking cardiovascular or psychotropic drugs.

Ethanol ingestion exaggerates postural pulse changes up to 8 hours following ingestion, [86] thus mimicking the hemodynamic changes seen with acute blood loss. However, in the setting of ethanol intoxication and trauma, one must be vigilant for associated occult hemorrhage and not reflexively assign tachycardia to being a result of intoxication alone.

The utility of orthostatic vital signs in children has been questioned. Horam and Roscelli found that healthy adolescents had heart rate changes of 21.5 ± 21.2 beats/min with orthostatic measurements made after 2 minutes of standing. [106] They found similar variation in the systolic blood pressure change (+19 to -17 mm Hg). Bergman and colleagues found that 25% of clinically normovolemic children had a postural increase in pulse of >20 beats/min and 11% had a postural fall in systolic blood pressure of >20 mm Hg. [107] However, children with fever and diarrhea were included in this "normal" study group. Another study comparing mildly dehydrated children with normal children found a significant difference in the orthostatic rise in pulse between the two groups. [108] Using near-syncope or a change in heart rate of >25 beats/min, orthostatic vital signs have a specificity of 95%, a sensitivity of 75%, and a predictive value of 92% in detecting mild clinical dehydration in children. [108] No difference in orthostatic blood pressure was found between normal and dehydrated children. Considering resting tachycardia as a positive sign of dehydration increased the predictive value of the test. The investigators concluded that in the appropriate clinical setting, an orthostatic increase in pulse >25 beats/min constitutes a positive tilt test, and an orthostatic pulse increase of <20 beats/min constitutes a negative test for hypovolemia. [108]

Another complicating factor in interpreting orthostatic vital signs is the development of *paradoxical bradycardia* in the presence of blood loss. Bradycardia in the face of hemorrhage has generally been considered a preterminal finding of irreversible shock, but bradycardia has been documented in hypovolemic, yet conscious, trauma patients. Several of the early studies [82] [83] noted that when orthostatic syncope occurred, it was accompanied by hypotension and often bradycardia. Many central nervous system factors can contribute to *vagal-mediated* syncope in ED patients with acute traumatic blood loss. These factors include pain, the sight of blood, stress, and nausea. Several investigators  have described women with hemoperitoneum secondary to ruptured

ectopic pregnancy who were hypotensive but did not have tachycardia. Jansen reviewed other cases of this relative bradycardia that occurred in hypotensive patients with acute intraperitoneal bleeding and postulated a parasympathetic mechanism triggered by the presence of free blood in the peritoneal cavity. [111] This bradycardia may be reversed with atropine, but *aggressive fluid replacement is the treatment of choice,* because anecdotal reports mention serious ventricular arrhythmias when atropine was used in this setting. [112] Paradoxical bradycardia has also been described in patients with abdominal or thoracic trauma or arterial bleeding from extremity wounds. [112] This paradoxical bradycardia may be more frequently associated with rapid and massive bleeding, whereas patients with a more gradual blood loss tend to have a more typical tachycardiac response. When the patient's clinical presentation is consistent with volume loss or shock, the clinician should not allow the absence of tachycardia to change the assessment.

## Indications and Contraindications

When the volume status of a patient is assessed by use of orthostatic vital signs, several points should be remembered. Many factors influence orthostatic blood pressure including age, preexisting medical conditions, the use of medication, and autonomic dysfunction (see Table 70-6) (Table Not Available) . Data relating the effect of *blood loss* to orthostatic vital signs are limited to phlebotomized healthy volunteers. Great care must be used when extrapolating these data to patients with anemia, dehydration, or painful trauma. The clinician must consider the clinical condition of the patient as well as the orthostatic vital signs in evaluating a patient for volume depletion.

Orthostatic vital signs are indicated as part of the evaluation of any patient with known or suspected volume loss or a history of syncope, except in the presence of the following contraindications: The use of orthostatic vital signs is unnecessary and dangerous in a patient with supine hypotension or the clinical syndrome of shock. Orthostatic vital sign evaluation also is contraindicated in patients with a severely altered mental status, in the setting of possible spinal injuries, and in patients with lower extremity or pelvic fractures.

The use of medications that block the normal vasomotor and chronotropic response to orthostatic tests also represents a contraindication to use of this test for assessment of volume status. However, when the patient's volume status is believed to be adequate and the clinician seeks to determine if specific medications may have affected the patient's ability to respond to postural changes, the test may be useful. In the latter situation, the primary finding may be the feeling of near-syncope with little or no change in vital signs.

Orthostatic vital signs are often used to assess a patient's response to therapy. In patients receiving intravenous rehydration therapy, serial orthostatic vital signs are widely used to judge the end point to therapy prior to release. Johnson and colleagues used this technique to demonstrate that the individual orthostatic vital signs response to saline infusion in women with hyperemesis gravidarum was associated with other measures of rehydration, including weight gain and decreased urine specific gravity. [113] Although the individual improvement in orthostatic vital signs in response to rehydration

was of clinical value, the presenting orthostatic vital signs were considered insufficient for use as the sole indicator of clinical dehydration in this population.

## Technique

Once the decision to obtain orthostatic vital signs has been made, the blood pressure and pulse are recorded after the patient has been in the supine position for 2 to 3 minutes (see Table 70-5) . The patient should rest quietly. No painful or invasive procedures should be performed during the test. Anxiety, fever, and other causes of resting tachycardia may make the test uninterpretable. [107]

The patient is then asked to stand, and the examiner should be prepared to assist the patient if severe symptoms or syncope develops. A supine-to-standing test is more accurate than a supine-to-sitting evaluation. Knopp and coworkers found that the supine-to-sitting test was not reliable for detecting 1000 mL of blood loss (55% false-negative results). [85] If severe symptoms develop (defined as syncope or extreme dizziness requiring the patient to lie down) on standing, the test is considered positive and should be terminated. If the patient is not symptomatic, the blood pressure and pulse should be recorded after the patient has been standing for 1 minute. This interval resulted in the greatest difference between the control and 1000-mL phlebotomy groups in the study by Knopp and colleagues. [85]

## Complications

The possible complications of orthostatic vital sign assessment can be avoided if the aforementioned contraindications and precautions are remembered. Complications include syncope with a resulting fall and injury and the possibility of exacerbating an existing fracture or spinal cord injury.

## Interpretation

Criteria for positive orthostatic vital sign changes are either tachycardia greater than a specific threshold (see below) or symptoms of cerebral hypoperfusion (e.g., near-syncope). Although blood pressure changes may be seen, they are too variable to be used as an indicator of blood volume loss. Although specific population-based thresholds for pulse rate and blood pressure changes have some value for identifying patients at high risk for significant volume loss, great individual variability limits the use of this technique as a screening test. That is, a volume loss of 500 mL (and occasionally more) may be associated with a negative orthostatic vital sign assessment (see below). [114] However, the use of serial measurements to ascertain the response to therapy of patients considered at risk for volume loss appears to have clinical utility. [113]

In the setting of possible blood loss, if the patient has a pulse rise of 30 beats/min or severe symptoms and if other complicating factors have been excluded, then blood loss is highly likely (2% false-positive rate). [85] The presence of a negative test indicates only that an acute blood loss of 1000 mL is unlikely (2% false-negative rate); a blood loss of

500 mL cannot be excluded (43% false-negative rate).

In children, postural near-syncope or an orthostatic pulse increase of 25 beats/min may be a predictor of mild dehydration. The accuracy of these criteria is increased by the addition of resting tachycardia. [108] However, one cannot quantify the amount of volume depletion with this test in children.

Criteria for significant orthostatic blood pressure changes cannot be definitively set for the following reasons: (1) in the study by Knopp and coworkers, a lack of correlation between blood pressure in the phlebotomy and control groups was seen [85] ; (2) a large variability in postural blood pressures has been found in the adult ED population ; (3) results of studies using passive tilt tables cannot be extrapolated to the bedside use of orthostatic vital signs; (4) studies using healthy patients with acute blood loss may not reflect the orthostatic changes that are seen in the elderly or those with chronic bleeding, dehydration, and various other medical problems; and (5) many of the studies of orthostatic changes never used a gold standard (measurement of actual volume loss) in their determinations. Because of the lack of agreement about the degree of postural blood pressure change that constitutes a positive test result, the most reasonable definition may be any postural fall in blood pressure that results in symptoms of cerebral hypoperfusion.

# CAPILLARY REFILL

The capillary refill test is a measurement of the time interval from the release of nailbed or soft-tissue pressure (sufficient to blanch the nailbed or superficial soft tissue) until the return to normal coloration. Delayed capillary refill is an indication of reduced skin turgor, often as a result of volume depletion. Measurement of the capillary refill time interval appears to be somewhat accurate in children, but its accuracy in assessing dehydration and reduced perfusion in adults is highly suspect. [84] [119]

**Physiology**

Skin elasticity is the characteristic of skin that allows it to spring back into its original shape after it has been deformed. One sign of decreased elasticity is skin tenting. The normal fullness of blood vessels or capillaries is referred to as *turgor vitalis*. The presence of normal skin turgor is a sign of adequate circulatory perfusion, because the speed of refilling of the capillary bed after compression is responsible for the return of color to the skin. [26]

**Indications and Contraindications**

There are no specific contraindications to performing capillary refill measurements in ED patients. However, the capillary refill time interval should not be obtained in a dependent extremity, a recently burned or injured extremity, or at the site of an infection or acute injury. Frequent monitoring of capillary refill is useful in accessing responses to rapid fluid resuscitation in children. Because capillary refill is available without additional equipment and takes only a few seconds to perform, it is a useful bedside assessment

of perfusion and dehydration.

**Procedure**

The preferred sites for performing capillary refill are the nailbed, the thenar surface of the palm, and the heel. The current standards are best developed for capillary refill obtained at the nailbed. [119] Regardless of the site chosen, the extremities should be positioned at about the level of the right atrium. The minimum pressure necessary to produce blanching yields the most reproducible values. Timing can be performed with a stop watch or simply by counting out "one-thousand-one, one-thousand-two" for approximation of the interval. A repeated measurement should be obtained at the same location as the initial test, because alternate sites may have different capillary refill times.

**Interpretation**

The normal capillary refill interval increases with age, degree of dehydration, and degree of hypoperfusion. Hypothermia, hyponatremia, congestive heart failure, malnutrition, and edema all increase the capillary refill interval.

The main difficulty in interpreting the capillary refill interval is that normal values in healthy patients fall into a wide range. In 30 normal infants from 2 to 24 months of age, the mean capillary refill interval was 0.8 ± 0.3 seconds. Measurements obtained from the nailbed were more reproducible than those from the heel. Dehydration was strongly correlated with a capillary refill time of >3 seconds, and suggested a fluid deficit of >100 mL/kg. [119]

The role of serial capillary refill interval measurements for assessing the response to rehydration in adults is unknown.

However, the test does not appear to be useful for assessing acute blood volume loss. In adults, the capillary refill interval was found to be less sensitive and less specific than orthostatic vital signs for detecting a 450 mL blood loss during blood donation.

# TEMPERATURE

Accurate measurement of body temperature is an essential part of clinical medicine. When taken in the context of other vital signs, abnormalities of core body temperature are excellent guides to the severity of illness.

Detection of abnormal body temperature facilitates proper diagnosis and evaluation of presenting complaints. The inability of any patient to maintain normal body temperature is indicative of a vast number of potentially serious disorders, including infections, neoplasms, shock, toxic reactions, and environmental exposures. [120] [123] Fever in neutropenic, immunocompromised, and intravenous drug-abusing patients may be more reliable than laboratory tests or physician assessment in diagnosing serious illness. [123] Infants are particularly sensitive to thermal stress and may demonstrate lower body

temperatures during asphyxia or necrotizing enterocolitis.  Normalization of body temperature following intervention may have important prognostic and therapeutic implications. [123]

## Physiology

Under normal conditions, the temperature of deep central body tissues (i.e., core temperature) remains within ±0.6 °C or ±1.08 °F. [127] [128] Core body temperature can be maintained within a narrow range while environmental temperature varies from as much as 13 to 60 °C (55 to 140 °F), [129] whereas surface temperature rises and falls with environmental and other influences. Maintenance of normal body temperature requires a balance of heat production and heat loss. Heat loss occurs by radiation, conduction, and evaporation. Approximately 60%, 18%, and 22% of heat loss, respectively, occurs by these methods. Heat loss is increased by wind, water, and lack of insulation (e.g., clothing). Sweating, vasodilation, and decreased heat production serve to decrease temperature, whereas piloerection, vasoconstriction, and increased heat production serve to increase body temperature. Heat production is increased by shivering, fat catabolism, and increased thyroid hormone production.

Temperature control occurs by feedback mechanisms operating through the preoptic area of the hypothalamus. Heat-sensitive neurons in this area increase their rate of firing during experimental heating. Receptors in the skin, spinal cord, abdominal viscera, and central veins primarily detect cold and provide feedback to the hypothalamus, which signals an increase in heat production. Stimuli that change the core body temperature result in reflex changes in mechanisms that increase either heat loss or production. [130]

## Indications and Contraindications

Clinicians generally measure body temperature to know if the patient's body temperature is outside the normal range and as an indication of pathologic conditions that can affect core body temperature. Because actual core body temperature measurement requires the placement of invasive monitors, such as an esophageal or a pulmonary artery probe, clinicians commonly use estimates of core body temperature, which conveniently and safely assess abnormalities of core temperature. Unfortunately, all noncore body sites and methods have inherent accuracy limitations, which physicians have come to accept in assessing most patients.

Oral temperature measurement requires a cooperative adult or child, generally older than 5 years. Patients who are grossly uncooperative, hemodynamically unstable, septic, or in respiratory distress require another method of temperature measurement. This group includes children <5 years and patients who are intubated.

Special techniques of measuring actual core body temperature may be indicated in certain patients in the ED, such as those with profound hypothermia, frostbite, or hyperthermia. Measurement of core body temperature is indicated in these individuals because it serves as an accurate measure of treatment effects.

## Measurement Sites

### Core Body Temperature

It has been demonstrated that the following sites accurately reflect core body temperature and its changes: esophageal (in the distal third of the esophagus), the tympanic membrane (using *direct thermistor contact* at the anterior inferior quadrant of the tympanic membrane), [131] [132] and the pulmonary artery temperature. [133]

Other sites may represent core body temperature under certain conditions. For example (1) the rectum, when the temperature is obtained at least 8 cm from the anus using an indwelling thermistor and the body temperature is relatively constant, and (2) the bladder when measured with an indwelling thermistor. [89] Data on core temperature in pediatric patients is limited and it is unclear if bladder, rectal, or oral temperature is a good measurement of core body temperature in children. [134]

### Peripheral Body Sites Approximating Core Body Temperature

Oral temperature measurement with a digital electronic probe is commonly used for ambulatory patients. [135] Advantages include convenience, timing, safety, and availability. Disadvantages include a variety of factors that have an impact on clinical accuracy and sensitivity. Electronic oral temperature probes must be covered with disposable covers, although these have been shown not to be completely effective in preventing probe contamination with microorganisms. [136] Although there are no absolute contraindications for oral temperature assessment, patients with factors shown to produce unreliable results (see below) require temperature measurement at other sites.

Rectal temperature is often considered the gold standard of body temperature for ambulatory patients and is often routine for children younger than 3 years. [140] Advantages include accuracy, sensitivity, and availability. One intensive care unit study found rectal probe temperatures to demonstrate limited variability or bias when compared with pulmonary artery temperatures. [141] Disadvantages include longer intervals for measurement, safety concerns, and inconvenience. Neutropenia and recent rectal surgery represent relative contraindications to rectal temperature measurement. [142] It may produce autonomic changes in patients with acute myocardial infarction.

Body temperature measured as a function of infrared radiation (IR) detected from the ear, including the auditory canal and tympanic membrane, is easy to use, hygienic, convenient, and quick. [144] The noncontact IR ear thermometer readings correlate well with other methods of body temperature measurement under a variety of clinical conditions. [145] Concerns have been raised over the accuracy of these thermometers in screening for fever in children <3 years of age.  Romano and colleagues found the Thermoscan Pro-1 IR thermometer to perform similar to rectal probe temperature, but the FirstTemp IR thermometer displayed considerably more variability. [141]

Axillary and tactile temperature assessment have been demonstrated to be unreliable and insensitive and should not be used as screening methods for core temperature abnormalities in the ED. Similarly, the use of liquid crystal chemophototropic strips applied to the skin of the forehead are not accurate for single measurement or fever screening. Single-use Tempa-DOT thermometers, which show increasing temperature dot darkening with increasing temperature, have been adopted by many EDs. The sensitivity of these thermometers for fever identification remains to be determined.

## Equipment

Mercury-in-glass thermometers remain popular despite requiring longer equilibration times and having cumbersome cleansing requirements. Electronic methods of temperature measurement are based on the thermocouple principle. Modern electronic thermometers signal once extrapolation of the temperature-time curve occurs. [153] Current in vitro standards call for an accuracy of ± 0.1 °C (± 0.18 °F) over the range of 37 to 39 °C (98.6 to 102.2 °F). [154] Thermistor probes (i.e., small thermocouples with instantaneous readouts) for esophageal and vascular temperature measurement provide continuous temperature readouts when attached to a potentiometer. [5] Thermistor products are available for esophageal, bladder, and rectal probes (Mallinckrodt Medical, Inc, St. Louis; or Yellow Springs Instruments, Yellow Springs, Ohio) with appropriate readout monitors.

Noncontact IR ear thermometers were introduced in 1985. These thermometers were initially used only in hospitals, but they are now sold over the counter for home use. The IR ear thermometers work by incorporating an IR sensor in the field of view of the IR emissions from the ear. Ear IR thermometers generally detect naturally occurring IR emissions over a brief time period, generally <1 sec. The emissions are converted to an electrical analog signal, which is digitized and analyzed by a microprocessor with resultant digital display. [155] The primary determinant of radiation emission is the temperature of blood perfusing this area, and the warmest spot in the auditory canal is the tympanic membrane. Thus, current IR ear thermometers are operator-dependent in the same fashion that otoscopy requires proper positioning to enable the examiner to "see" the tympanic membrane. [156]

A variety of IR ear thermometers are available commercially (Table 70-7) with varying operating temperature ranges, features, and reported accuracy. Clinical data assessing relative performance of the many commercial devices are limited. In one study comparing many of these IR thermometers, the Pro-1 and FirstTemp gave significantly higher readings. [156]

## Procedure

Temperature measurement begins with body site selection. Consideration should be given to the accuracy of using a site to reflect core temperature, the site sensitivity to temperature

| | **Brand Name** | | | | | |
|---|---|---|---|---|---|---|
| | **Genius** | **Diatek-9000** | **LighTouch** | **Pro-1** | **HM-1** | **Pro-LT** |
| Manufacturer § | IMS | Diatek | Exergen | Thermoscan | Thermoscan | Thermoscan |
| Temperature range (°C) ¶ | 16-34 | 24-42 | 16-54 | 26-42 | 34-42 | 34-42 |
| Read modes | O.R.C.Cal.S | O.R.T.S | T | O.R.C.T | O.R | O.R.T |
| Response time (sec) | 2 | 3 | 1 | 1 | 1 | 1 |
| Continuous mode | Yes | No | Yes | No | No | No |
| Charge use time | 10,000 uses | 20,000 uses | 4000 uses | 1500 uses | 1500 uses | 10,000 uses |
| Warranty | 3 yr | 1 yr | Lifetime | 1 yr | 1 yr | 1 yr |
| Anti-theft | No | Yes | No | Yes | No | No |

**TABLE 70-7** -- Commercial Infrared Ear Thermometers *

| Offsets (°C) [*] | O=0.80 | O=3.30 | None | O=0.30 | O=0.30 | O=0.30 | |
|---|---|---|---|---|---|---|---|
| | R=1.10 | R=1.20 | -- | R=1.15 | R=1.15 | R=1.15 | |
| | C=1.20 | -- | -- | C=0.74 | -- | -- | |

O , Oral; R, rectal; C, core: Cal, calibration; T, tympanic; S, surface.

* Available in the United States as of May 1995.
A "pediatric" infrared ear thermometer (Pedi-Q) also is available from this manufacturer.
Not intended for professional use.
§ IMS, Intelligent Medical Systems, San Diego; Diateck, Diateck Corp, San Diego; Exergen, Exergen Corp, Newton, Mass; Thermoscan, Thermoscan Inc, San Diego; IVAC, IVAC Corp, San Diego.
¶ Thermometers listed meet laboratory standards of the American Society for Testing and Materials (±0.1°C).
** Temperature value, which is added to unadjusted infrared ear thermometer reading to estimate body temperature at specific sites.

changes, convenience, time required, safety, and site availability.

When reusable probes are used, the thermometer end should be covered with a probe cover. Temperature measurement continues with insertion of the temperature probe and probe equilibration with the temperature of local body tissues. Proper placement of temperature probes significantly influences results for oral, rectal, esophageal, and vascular temperatures.

Sublingual oral temperatures should be obtained in either posterior sublingual pocket, with the mouth closed. [158] The patient should be sitting upright or lying, holding the base of the probe with one hand. [137] Rectal temperatures require removal of clothing, lubrication of the probe, and gloving of personnel, and should be obtained with the patient relaxed in the left or right lateral decubitus position. Gentle insertion to 3 to 5 cm should ensure accurate, atraumatic results. [127]

Axillary temperatures are frequently used for neonatal patients in incubators because of convenience. The technique is not indicated unless other sites are unavailable.

Although not commonly used, temperature measurement in a freshly voided urine specimen can validate temperature measurement at other body sites. A nomogram of expected urinary temperatures has been derived from measurements of oral and urinary temperatures in 55 subjects (Fig. 70-10) (Figure Not Available) . [161]

Placement of an esophageal catheter for measurement of core body temperature is similar to placement of a nasogastric or orogastric tube (see Chapter 43) . In this case, the distal tip of the esophageal catheter contains a thermistor to measure body temperature. In normal adults, the catheter is inserted approximately 34 cm deep into the esophagus, with the objective of locating the probe tip at the level at which the esophagus is between the aorta and the left atrium. The catheter is connected to a potentiometer and allowed to equilibrate briefly before a body temperature reading is taken.

For pulmonary artery (PA) catheter assessment of core body temperature, a PA or thermistor-tipped catheter is placed into the PA. A detailed discussion of the placement of PA catheters is beyond the scope of this chapter. Once a PA catheter is in the correct position, the temperature value may be obtained by using the potentiometer attached to the distal thermistor. Because of the risks associated with catheter placement, PA temperatures are generally reserved for patients with another clinical indication for PA pressure monitoring.

**Complications**

Complications associated with axillary, oral, ear IR, and liquid crystal thermometers are rare or unreported. Tympanic membrane perforation and pain have been reported as complications of thermistor probe placement in the auditory canal. Complications associated with rectal temperature measurement are extremely rare, but include rectal perforation, pneumoperitoneum, bacteremia, dysrhythmias, and syncope. Falsely low, but supranormal, rectal temperature measurements may be seen during shock. [162] Rectal temperatures also may lag behind core temperature changes.

Complications associated with esophageal temperature measurement include those associated with placement of an orogastric or nasogastric tube (see Chapter 43) . Confirmation of accurate placement can be verified with a chest radiogram. In addition, spurious temperatures may arise from an improperly calibrated potentiometer, a damaged thermistor, a proximal esophageal location, and during mechanical ventilation or thoracotomy.

**Interpretation**

Normal values for body temperature are affected by the following variables: (1) site and methods used for measurement, (2) perfusion, (3) environmental exposure, (4) pregnancy, (4) activity level, and (5) time of day. Clinicians must interpret body temperature with knowledge of the range of normal values at the intended site of measurement. Although the core body temperature remains nearly constant (37.0 ± 0.6 °C or 98.6 ± 0.18 °F), the surface temperature rises and falls with changes in ambient temperature, exercise, and time of day. The definition of fever varies by the site of

measurement and is defined by a temperature greater than 2 SD above the mean. Fever has been defined as an oral temperature 37.8 °C (100.0 °F), [163] a rectal

temperature 38.0 °C (100.4 °F), [164] or an IR ear temperature 37.6 °C (99.6 °F). [165] Hypothermia has been defined as a core body temperature <35 °C (<95 °F), whereas hyperthermia has been defined as a core body temperature >41 °C (>105.8 °F), with accompanying symptoms and signs. [166] A useful nomogram for conversion of degrees centigrade to degrees Fahrenheit is provided in Figure 70-11 .

Temperature probes that require the transfer of heat energy from local tissues to the temperature probe require a period of equilibration and reliable tissue contact at the intended body site. Acceptable equilibration times for mercury-in-glass thermometers for oral, rectal, and axillary sites are 7, 3, and 10 minutes, respectively. Used in a predictive mode, electronic digital thermometers generally require 30 seconds for oral or rectal temperature equilibration. The predictive mode uses temperature changes vs time to predict an equilibration temperature.

Normal ranges and suggested febrile thresholds for common body sites and methods should be considered in the interpretation of temperature values (Table 70-8) . The interpretation of temperature measurements during clinical assessment must consider the use of antipyretics, level of activity, pregnancy, environmental exposure, and patient age. The duration of antipyresis with acetaminophen or aspirin is 3.5 to 4 hours. When both drugs are given together, the duration of action may be extended up to 6 hours. [167] Body temperature is increased during sustained exercise, during pregnancy and the luteal phase of the menstrual cycle, and in the later afternoon by diurnal variation. Body temperature is generally reduced by advanced age.

The interpretation of temperatures obtained with pulmonary artery or esophageal thermistors is generally straightforward.

| TABLE 70-8 -- Normal Ranges and Suggested Febrile Thresholds for Human Body Temperature (in Healthy Resting Patient) | | | |
| --- | --- | --- | --- |
| Body Site | Type of Thermometer | Normal Range (°C) | Fever (°C) |
| Core * | Electronic | 36.4-37.9 | 38.0 |
| Oral | Mercury-glass, electronic | 35.5-37.7 | 37.8 |
| Rectal | Mercury-glass, electronic | 36.6-37.9 | 38.0 |

| Ear | Infrared emission | 35.7-37.5 | 37.6 |

* Temperature obtained with a properly positioned pulmonary artery, esophageal, or tympanic membrane thermistor.
For unadjusted ear temperature using Thermoscan Pro-1 (Thermoscan, Inc, San Diego)

Comparison of measured values to the expected normal range should be performed to determine if the patient has an abnormal core body temperature. In addition to improper placement of the thermistor, sources of temperature error include an improperly calibrated potentiometer or thermistor, damaged thermistor, or improper placement. The biomedical staff at the institution should periodically verify readout and calibration of these instruments.

Oral temperature measurements are affected by ingestion of hot or cold liquids, [142] tachypnea, [168] and cold ambient air. [169] Smoking appears to result in little change in oral temperatures. [142] [170] Therefore, before taking an oral temperature, the examiner should inquire about these features and possibly delay taking the temperature. Also, Erickson found a 2.7 °C (4.9 °F) reduction of oral temperature measurement when the probe was placed under the tip of the tongue instead of under the posterior sublingual pocket. [137] When using a mercury-glass thermometer, optimum placement time was found to be 7 minutes for oral temperatures in children. [171]

Axillary temperatures obtained in 108 children by Kresch had a sensitivity of only 33% and a specificity of 98% for fever. [149] Ogren obtained similar results using an electronic thermometer in the axilla. [172] Hence, axillary temperatures should not be used to screen for fever.

When rapid changes in body temperature are occurring, oral and tympanic temperature measurements appear to be more reliable than rectal temperature. In 20 adults examined during open-heart surgery, oral temperatures showed a better correlation with blood temperature during rapid cooling and rewarming. [159] In 12 adults, the average rectal temperature lag during warming was 5.3 minutes during water immersion and 3.8 minutes during exercise. [173] Sublingual (oral) temperature delay (1.3 and 0.9 minutes for the two experiments, respectively) was less than auditory canal delay (1.1 and 4.1 minutes, respectively) in temperature response.

Infrequently, ED patients require constant monitoring of temperature (e.g., in cases of hypothermia or hyperthermia). This can usually be performed using a bladder or esophageal probe attached to a potentiometer. Patients with indwelling central venous or pulmonary arterial catheters may have electronic thermistors inserted into the central circulation to measure core body temperature. As noted above, rectal temperature measurements are less desirable for monitoring patients undergoing rapid core temperature changes. Periodic IR tympanic temperature monitoring may represent one useful option in the hypothermic patient.

The interpretation of ear IR temperatures requires a knowledge of the mode of

thermometer operation and ambient temperature. Cerumen occlusion of the ear canal may produce a falsely low reading. [175] Most IR ear thermometers allow the user to select a mode that allows prediction of the equivalent temperature at other body sites. IR ear thermometers appear moderately sensitive for fever. [146] [147] If these devices are used, the clinician must be aware of the potential for a falsely low temperature and, when in doubt, the measurement should be repeated with a more standard method.

## CONCLUSION

Vital signs must always be interpreted in relationship to each other to obtain a more complete clinical picture. All vital signs are subject to errors in measurement and therefore must be verified when the initial result does not match the clinical presentation. Abnormal vital signs may lead the clinician to a diagnosis, and abnormalities should be explained within the context of the patient's illness.

# Chapter 71 - Procedures Pertaining to Hypothermia

**David Doezema, David P. Sklar**

The spectacular reports of survival after up to 66 minutes of cold water immersion fuel our fascination with hypothermia. [1] [2] The severely hypothermic patient appears at times to lie suspended somewhere between life and death. Generally, physicians continue cardiopulmonary resuscitation (CPR) long past the time other patients would be declared dead, occasionally succeeding in resuscitating a hypothermic patient, who later leaves the hospital neurologically intact.  Unfortunately, the medical literature on treatment of hypothermia is generally characterized by anecdotal reports, patient selection bias, and uncontrolled case series. However, because the condition is relatively rare, this information must be used to augment the individual physician's limited clinical experience.

This chapter critically reviews approaches and procedures appropriate to the management of several categories of hypothermic patients. The recommendations combine treatment efficacy with safety. Before describing procedures and making recommendations, essential terms are defined and the pathophysiology of hypothermia is briefly reviewed.

## DEFINITIONS

*Accidental hypothermia* (AH) has been defined as an unintentionally induced decrease in the core (vital organ) temperature below 35 °C (95 °F). [5] Victims of hypothermia can be separated into the following categories: *mild hypothermia*, 35 to 33 °C (95.0 to 91.4 °F); *moderate hypothermia*, <33 to 28 °C (<91.4 to 82.4 °F); and *severe hypothermia*, <28 to 9 °C (<82.4 to 48.2 °F). Other factors that may be useful in separating groups of patients with AH include the presence of underlying illness,  altered neurologic state on arrival, hypotension, and the need for out-of-hospital CPR. [11] A hypothermia outcome score has been developed that incorporates some of these factors and may permit comparison of outcomes for patient groups treated with different modalities. [11]

Because signs and symptoms may be misleading, mild to moderate hypothermia may easily be overlooked in the emergency department (ED). A common error is failure to routinely obtain a core temperature on all patients at risk. Presenting symptoms such as confusion in the elderly and combativeness in the intoxicated patient may not initially be recognized as symptoms of hypothermia. *Hypothermic patients frequently will not feel cold or shiver*, and a "paradoxical undressing" has been described in confused patients who apparently have the sensation of heat at lowered body temperatures.

## CORE TEMPERATURE MEASUREMENT

Accurate temperature measurement is important to guide diagnostic and therapeutic decisions. Any thermometer that does not record temperatures in the hypothermic range is *inappropriate* for evaluating significant hypothermia. Standard glass/mercury

thermometers generally cannot record temperatures of <34 °C (93.2 °F), although some models are available to record temperatures as low as 24 °C (75.2 °F) (Dynamed, Inc, Carlsbad, Calif). An electronic probe and accompanying calibrated thermometer (Fig. 71-1) (Figure Not Available) is recommended when monitoring this vital sign. A detailed description of core temperature measurement is provided in Chapter 70 .

Core temperature can be estimated with a rectal probe, although rectal temperature often lags behind core temperature. Esophageal probes may be used, although they may be affected by warm humidified air therapy, which is commonly used in severe hypothermia. Other possible sites for temperature measurement include the tympanic membrane and the urinary bladder. [12] Fresh urine temperature can closely approximate core temperature. [13] For monitoring purposes, rectal or bladder probe use is preferred. Although there are excellent correlations between tympanic probe and core temperatures, the role of infrared tympanic temperatures for monitoring the temperature of hypothermic patients is unknown (see Chapter 70) . When a rectal probe is used, it should be inserted at least 10 cm beyond the anal sphincter and its position frequently verified. One should remember that temperature gradients exist in the human body and consistency of monitoring at one or more sites is mandatory. A chart and formula that convert centigrade to Fahrenheit temperatures will assist the clinician in assessing the severity of hypothermia (Fig. 71-2) .

## PATHOPHYSIOLOGY

*Accidental hypothermia* occurs when the body's heat-producing mechanisms cannot keep up with heat loss. Heat production occurs through basal metabolic functions and muscular activity. Heat production can be increased from the basal rate of 100 to 500 kcal/hour by shivering.  Heat loss occurs through radiation, conduction, evaporation, and convection. Vasoconstriction and shunting of blood can preserve core temperature at the expense of a further temperature decrease of the skin. As the core body temperature drops

below 33 °C (91.4 °F), the patient becomes confused and ataxic. [15] Shivering stops at about 32 °C (89.6 °F). Atrial fibrillation occurs frequently as the temperature continues to drop, [16] and the patient loses consciousness. [15] A J-wave in the electrocardiogram often appears before ventricular fibrillation (Fig. 71-3) (Figure Not Available) . Ventricular fibrillation may occur below 29 °C (84.2 °F) and becomes common as the core drops to 25 °C (77 °F). [15] The electroencephalogram flattens at 19 to 20 °C (66.2 to 68 °F), [17] and asystole commonly occurs at 18 °C (64.4 °F) but has been seen at higher temperatures. The lowest recorded temperature for a survivor of AH is 16 °C (60.8 °F).

## INITIAL EVALUATION AND STABILIZATION OF THE HYPOTHERMIC PATIENT

Overall, one must realize that with the reduction of core and cellular temperatures, it is not surprising to find a parallel reduction in all parameters of vital activity, because the enzymatic rate of metabolism itself decreases two to three times with each 10 °C (18

°F) drop and cerebral blood flow decreases 6 to 7% per 1 °C (1.8 °F) drop. Profound hypothermia results in coma, hyporeflexia, fixed and dilated pupils, severe bradycardia, and often unobtainable blood pressure. In severe hypothermia, a pulse may not be palpable and blood pressure measurement may require the use of a Doppler device (see Chapter 70) . Electrocardiographic monitoring provides rate and rhythm status. All patients who have more than minimal impairment require frequent determination of their oxygenation, ventilation, and acid-base status by means of arterial blood analysis.

All patients should have an adequate-bore IV line established for fluid, glucose, and possible drug administration. Most hypothermic patients are *dehydrated*, because fluid intake is reduced and cold causes a diuresis; maintenance IV fluids should be given routinely. Warming of all IV fluids to 40 to 42 °C (104 to 107.6 °F) is reasonable, but the usual volumes administered will not contribute significant calories of heat.

With a mild to moderate reduction in core temperature, the level of mentation correlates with the severity of the AH or associated illness or both. In alcoholics or diabetics, coma at higher core temperatures may be due to unsuspected hypoglycemia, and a trial of glucose by bolus infusion is justified. In the 22 cases of AH reviewed by Fitzgerald, all except 2 patients were alcoholics. [19] The serum glucose level was <50 mg/dL in 41% (9 patients). This study noted glycosuria in 2 patients, even when low serum glucose values were evident, and described a renal tubular glycosuria in AH. Such glycosuria may worsen or cause hypoglycemia; *hence, glycosuria in AH is no guarantee of an adequate serum glucose concentration*. This supports the routine use of supplemental IV glucose unless a normal serum glucose value can be quickly ensured. Also, IV thiamine (100 mg) and a trial dose of 2 mg IV naloxone (Narcan) should be given to *all obtunded victims* to treat potential thiamine deficiency and narcotic overdose, respectively. Although failure to rewarm spontaneously has been noted in victims with hypothyroidism and other endocrine deficiencies, the use of thyroid hormones and corticosteroids is reserved for those patients with suspected thyroid and adrenal insufficiency, respectively.

Antibiotics are not routinely indicated in uncomplicated mild hypothermia. Although there is no consensus or clinical proof on the value of routine antibiotic therapy in severe AH, the inaccuracy of historical, laboratory, and clinical data in distinguishing the infected from the noninfected hypothermic patient has prompted some authors to advocate the routine empirical initiation of broad-spectrum antibiotic therapy on admission of severely hypothermic patients.

## TREATMENT OVERVIEW

The treatment of AH involves support of basic bodily functions (i.e., airway, breathing, and circulation) while preventing further heat loss and augmenting heat production. Although AH may be lethal, the speed and manner of warming the patient may also be harmful by causing or worsening hypotension, a paradoxical decrease in core temperature, [20] [21] and cardiac dysrhythmias. [22] Other complications may include bleeding [23] and infection of surgical incisions. Invasive therapy should be carefully considered and individualized to the severity of the hypothermia and the condition of the patient, and one should avoid the temptation to *overtreat and overmonitor with invasive*

*techniques the otherwise stable hypothermic patient.*

As opposed to the need for rapid temperature correction with heat stroke, a condition in which it is essential to lower the core temperature as rapidly as possible, a more gradual and conservative approach is generally advocated for rewarming the mildly to moderately *hypothermic* patient. Although the ideal rewarming rate is unknown and clearly varies with each case, it is logical to be content with a 0.5 to 2.0 °C (0.9 to 3.6 °F)/hour rise in temperature *in the otherwise stable patient* (Table 71-1) (Table Not Available) . Patients who slowly become hypothermic over hours or days may not tolerate nor require invasive procedures or aggressive therapy.

The patient with severe underlying problems such as hypoglycemia, hyperglycemia, sepsis, adrenal crisis, drug overdose, or hypothyroidism should be treated appropriately for those conditions as well as aggressively for hypothermia, because long-term outcome may depend more on treatment of the underlying illness than the hypothermia. [24] [25]

For patients with AH that is mild, removal of wet clothing followed by *passive external rewarming* with blankets generally suffices. Passive external warming results after the addition of increased insulation, preventing further heat loss so that the body's own mechanisms for heat production can restore normal temperature. The technique is simple; however, the patient must be capable of generating enough body heat for this method to be successful. Patients who cannot shiver, patients who are hypotensive, or patients who are intoxicated or malnourished may not have this capability; however, most patients with mild hypothermia can be warmed gradually with passive rewarming techniques (Fig. 71-4) . Survival rates using passive external rewarming have ranged from 55 to 100%.

For patients in the moderate or severe category of hypothermia, a more aggressive approach may be warranted. The options available are active external rewarming and active core rewarming. Active core rewarming techniques can be further divided into less invasive and more invasive techniques. Generally, the aggressiveness of therapy depends more on the patient's underlying health, hemodynamic status, and response to initial therapy than the initial temperature.

## Active External Rewarming

The application of heat to the skin of the hypothermic patient has been termed *active external rewarming.*

### Indications

Although there is some suggestion that active external rewarming of profoundly hypothermic patients by immersion may be associated with an increase in mortality over other treatments, [11] [20] more recent studies have suggested that this technique is highly effective for mild hypothermia. [28] Other forms of active external rewarming are increasingly used in the ED as adjunctive care of moderately hypothermic, otherwise

healthy individuals. Vasoconstriction limits the ability to increase core temperature using techniques that primarily warm the skin. [29]

Finally, in settings where more aggressive warming techniques are precluded owing to lack of equipment or personnel, active external rewarming (e.g., with body-to-body contact) may be the only option available to the rescuer. However, the rewarming contribution of body-to-body contact appears limited. [30]

**Equipment**

Traditionally, immersion therapy has used a heated (40 to 42 °C) water tank of the type present in most burn units. Generally, all of the hypothermic patient except the extremities and head are immersed (Fig. 71-5) , although immersion of the extremities may hasten rewarming. [28] Alternatively, conduction warming is performed using warm water-filled heat exchange blankets (e.g., Blanketrol; Cincinnati Sub-Zero Products, Cincinnati). A forced warm air convection system (Bair Hugger; Augustine Medical, Eden Prairie, Minn) has been used for postsurgical rewarming [31] and holds promise for AH treatment.

**Technique**

Because profound fluid shifts can occur with conduction warming, the patient should receive supplemental IV fluid [32] that has been warmed to 40 °C, given at a rate sufficient to generate a urinary output of 0.5 to 1.0 mL/kg/hour. An initial fluid bolus of 500 mL of 5% dextrose and normal saline may be beneficial. (Note that blood pressure is not an accurate way to gauge fluid resuscitation, because serious hypothermia is always accompanied by "physiologic" hypotension.) Because patients requiring mechanical ventilation have rarely been subjected to tank immersion, it cannot be recommended for hypothermic patients who require intubation. Rewarming rates ranging from 0.9 to 8.8 °C (1.6 to 15.8 °F)/hour have been reported with immersion therapy. [20] [33]

A heat exchange blanket allows the patient to receive other treatments that may be difficult or impossible to carry out in a tub, such as defibrillation, CPR, or more invasive warming techniques. With this latter form of conduction heating, the heating blanket and overlying cloth sheet is placed under the patient. The blanket temperature is set at 40 to 42 °C, and the measures described under passive rewarming techniques (see Fig. 71-4) are initiated.

Forced-air rewarming (convection) uses a blanket cradle to create an environment through which heated air is blown. Access to the patient is quite good with this system in that the overlying blankets can be raised temporarily to evaluate the patient or perform procedures. One study found this approach superior to a heating blanket. [31] Experience with mild immersion-induced hypothermia in volunteers suggests that the forced-air technique warms at a rate comparable to vigorous shivering, but with less metabolic stress and less afterdrop. [34]

**Complications**

There is concern that surface warming with accompanying vasodilation may produce a relative hypovolemia in the hypothermic patient. A further decrease in the core temperature may occur as the blood courses through still-cold extremities and returns to the heart. In the study by Miller and coworkers, survival with this method was only 36%. [20] However, others have described a 95% survival rate. [33] One study of human volunteers with mild to moderate hypothermia comparing external rewarming versus inhalation with warm air demonstrated superior warming rates using external rewarming without adverse effect. [35]

Importantly, CPR and other advanced cardiac therapy and monitoring are impossible with immersion rewarming. Until studied further, active external rewarming should only be considered in a clinically monitored setting for mildly hypothermic patients who can protect their airway. When using a heating device, the potential for burns to the areas in greatest contact with the heating source should also be monitored.

## Active Core Rewarming

There is evidence that active core rewarming may decrease mortality from *severe* hypothermia exposure compared with other techniques. [11] Several methods have been described, including the use of warm humidified air through an endotracheal tube or mask (Fig. 71-6) , peritoneal lavage, gastric or bladder lavage with warm fluid, thoracic tube lavage, cardiopulmonary bypass (Fig. 71-7) , hemodialysis, and thoracotomy with mediastinal lavage (Fig. 71-8) . These techniques transfer heat actively to the body core, achieving varying rewarming rates. The specific techniques as well as some of the advantages and disadvantages for each procedure are described below.

**Less Invasive Techniques**

**Inhalation of heated humidified $O_2$ or air**

The use of warm humidified oxygen to treat hypothermia was described by Lloyd in 1973. [36] Miller and colleagues reported average rates of rewarming of 0.74 °C (1.3 °F)/hour via mask and 1.22 °C (2.2 °F)/hour via endotracheal tube with heated aerosol at 40 °C. [20] Faster rewarming rates may be accomplished using a maximum safe aerosol temperature of 45 °C (113 °F). The Multicenter Hypothermia Study showed an average first- and second-hour rewarming rate of 1.5 to 2.0 °C (2.7 to 3.6 °F) for mask and endotracheal tube inhalation in severe hypothermia. [37] This rate was somewhat less in moderate hypothermia.  Core rewarming with the technique occurs through the following mechanisms. The warmed alveolar blood returns to the heart, thereby warming the myocardium. The warmed humidified air delivered to the alveoli also warms contiguous structures in the mediastinum by conduction. Finally, warming the inhaled air or oxygen eliminates a major source of heat loss.

**Indications and contraindications**

The use of heated humidified air or oxygen is a simple technique that *should be used routinely*, either by itself or in combination with other methods, in all patients with hypothermia, regardless of severity. If the correct equipment is available, it can be used in the field as well as the hospital. [36] [38] However, one must address the risk of burns during inhalation of warm air in the field environment. [39] Mouth-to-tube ventilation in the intubated hypothermic out-of-hospital patient has the theoretical advantage of providing warm humidified air without special equipment. The ventilating rescuer can inhale oxygen before expiring into the patient's endotracheal tube to provide air with increased oxygen content. There are no contraindications for or reported complications from the use of warm humidified air for hypothermia, and there is no afterdrop. [21]

**Technique**

A heated cascade nebulizer can be used with a mask for patients with spontaneous respirations or with a volume ventilator for intubated patients. It is important to monitor the inspired air to maintain a temperature of approximately 45 °C. [40] Temperatures >50 °C (122 °F) may burn the mucosa, and temperatures <45 °C (<113 °F) do not deliver maximum heat. The air or oxygen must be humidified, and the heater module may need modification, because many units have feedback mechanisms that shut off at a given temperature. As a practical issue, it may be difficult to deliver oxygen at the recommended temperature because of equipment limitations. In our institution, we found that without modification, we could not deliver humidified air at >40 °C. In many cases the air temperature was only 30 °C (86 °F).

**Conclusions**

Studies have suggested that the rewarming rate of inhalation therapy is inferior to that of peritoneal lavage, thoracic lavage, and bath rewarming. [41] However, because inhalation therapy can be combined with any and all other methods of rewarming and because it is relatively noninvasive and inexpensive, it should be considered as the initial treatment of choice for hypothermic patients.

**Peritoneal dialysis**

Peritoneal dialysis (lavage) is an ideal treatment for severe hypothermia because it is available in most hospitals and does not require any unusual equipment or training. Rewarming rates of 1 to 3 °C (1.8 to 5.4 °F)/hour, depending on the dialysis rate, can be achieved without sophisticated equipment that may delay therapy or require transfer of the patient to a tertiary care facility. [37] [42]

Peritoneal dialysis rewarming was first used successfully in a patient in ventricular fibrillation with a temperature of 21 °C (69.8 °F). [43] Since that time, there have been reports of successful rewarming with peritoneal lavage in stable, severely hypothermic patients and unstable hypothermic patients in cardiac arrest. [42] [44] Peritoneal lavage

works through heat transfer from lavage fluid to the peritoneal cavity. The peritoneal great vessels and abdominal organs provide a large surface area for heat exchange. There are few controlled comparisons with other rewarming methods, [41] and failures are probably not reported.

**Indications and contraindications**

Peritoneal dialysis is indicated in any severely hypothermic patient (temperature <28 °C [82.4 °F]). Although theoretically less effective than other techniques that directly warm the thorax in the setting of cardiac arrest, it has been used successfully in that situation. It is particularly useful in hypothermic patients who have overdosed with a dialyzable toxin. Other less invasive methods, such as gastric or bladder lavage or warm nebulized air or oxygen inhalation, may be preferred in stable patients with temperatures >26 to 28 °C (78.7 to 82.4 °F). Peritoneal dialysis should not be performed on patients with previous abdominal surgery and should be used with extreme caution in patients with a coagulopathy after the risks and benefits have been considered.

**Equipment**

The equipment described in Chapter 46 is needed. We recommend using the Seldinger technique with a commercially available disposable kit (e.g., Arrow peritoneal lavage kit, product No. AK-09000, Arrow International, Inc, Reading, Pa) because of the ease of performance and minimal morbidity associated with this procedure.

**Technique**

In the noncritical patient, a coagulation profile should be obtained before the procedure, but immediate initiation is warranted in life-threatening situations. The patient should be supine with a Foley catheter and nasogastric tube in place. After infiltration with lidocaine, an infraumbilical stab incision is made with a No. 11 blade, and an 18-ga needle is placed into the peritoneal cavity directed toward the pelvis at a 45° angle. A standard flexible J-wire is inserted through the needle, and the needle is removed. The 8-Fr dialysis catheter is passed over the wire with a twisting motion, and the wire is removed.

Lavage rates of 4 to 12 L/hour can be achieved with 2 catheters. Fluid is warmed with a standard blood warmer to 40 to 45 °C. We recommend using standard 1.5% dextrose dialysate solution, adding potassium (4 mmol/L) if the patient becomes hypokalemic. Ringer's solution and normal saline have also been used successfully. [45] The rate should be at least 6 L/hour and preferably 10 L/hour. [45]

**Complications**

The Seldinger method has a complication rate of <1%. [46] [47] A "mini-lap" using direct dissection may also be used but may have a higher complication rate. [47] Further

discussion of potential complications is provided in Chapter 46 .

### Conclusions

Peritoneal dialysis is a useful method because it uses readily available fluid and can be done with a self-contained disposable kit. If a hospital also treats trauma victims, the same lavage kit can be used for evaluation of abdominal trauma. If this technique is combined with warm nebulized inhalation, warming rates of 4 °C (7.2 °F)/hour can be achieved. [37]

### Gastrointestinal and bladder rewarming

Gastric or bladder irrigation offers some of the same advantages as peritoneal dialysis without invading the peritoneal cavity. Heat is delivered to structures in close proximity to the core. In the Multicenter Hypothermia Study, gastric/bladder/colon lavage had a first hour rewarming rate of 1.0 to 1.5 °C and a second hour rewarming rate of 1.5 to 2.0 °C for severe hypothermia. [37] In a multifactorial analysis of the Multicenter Hypothermia Study there was a trend toward improved survival in patients on whom this method was used. [11]

Although the amount of heat delivered with gastric lavage appears less than that delivered with peritoneal dialysis, it is somewhat easier to use and less invasive. When combined with other methods, gastric or bladder lavage provides significant warming. [37] [48] Serum electrolyte levels should be monitored if large volumes of tap water are used, because delusional electrolyte disturbances may occur. Children may be more susceptible to electrolyte changes with tap water irrigation. [49]

### Indications and contraindications

Warmed gastric or bladder lavage is indicated in moderate or severe hypothermia. It can be combined with other warming techniques when rapid rewarming is needed. Patients who are obtunded and lack protective airway reflexes should have endotracheal intubation before gastric lavage to prevent aspiration of gastric contents. The reader is referred to the appropriate chapters concerning nasogastric tube placement (see Chapter 43) , gastric lavage (see Chapter 45) , and urethral catheterization (see Chapter 59) for specific contraindications to these procedures.

### Equipment

We recommend using large-diameter 32- to 40-Fr lavage tubes with normal saline or lactated Ringer's solution warmed to 40 to 45 °C (in a microwave or blood warmer with verification of temperature before use), and a Y connector and clamp as described in Chapter 45 . Although smaller tubes are easily passed nasally, we recommend oral placement of the large lavage tubes. A modified Sengstaken tube with gastric and esophageal balloons may also be used. [32]

### Technique

From 200- to 300-mL aliquots of fluid may be instilled into the stomach before removal by gravity drainage. For bladder irrigation, the optimal volume is not known but bladder distension should be avoided (100-200 mL aliquots should be sufficient). The amount of time that the irrigant should be left before removal is not known, but we recommend rapid exchanges with a dwell time of 1 to 2 minutes.

### Complications

The lavage complications include trauma to the nasal turbinates (especially if a large tube is passed nasally), gastric and esophageal perforation, dilutional hyponatremia, inadvertent placement of the tube in the lungs, and pulmonary aspiration. All of these can be minimized by careful, proper technique. Fluid overload or electrolyte disturbances when using tap water are potential complications.

### Conclusions

Gastrointestinal and bladder lavage with heated fluids is easily performed using equipment and solutions available in any hospital. Because of its ease and availability, it can be started early in the resuscitation and combined with any other rewarming method to give a significant added increment of heat.

## More Invasive Techniques

### Thoracic cavity lavage

Thoracic cavity lavage can be performed either *closed*, through chest tubes placed in the one hemithorax, or *open*, after resuscitative thoracotomy. The former approach offers the advantages of being less invasive and being based on procedures more commonly used by the emergency physician. Furthermore, closed-chest CPR can be continued while this technique is used. The open thorax approach offers the theoretical advantage of direct heart warming and the option of open-chest cardiac massage. Rapid warming rates of 6 to 7 °C in 20 minutes have been described. [56]

### Indications and contraindications

Thoracic cavity lavage should be considered for patients requiring rapid core rewarming in the setting of inadequate perfusion (e.g., shock or during CPR) when cardiac bypass is not available. Open thoracic lavage should be considered in those patients who will receive open chest massage or thoracotomy for other reasons (e.g., hypothermic arrest with penetrating trauma). Generally, thoracic lavage is not necessary for patients with mild or moderate hypothermia who can be rewarmed by other less invasive methods. The technique should be avoided in the patient with a coagulopathy unless needed as a

life-saving measure.

**Technique**

**Closed thoracic lavage**

Two large-bore thoracostomy tubes (e.g., 36 to 38 Fr in 70-kg adults) are placed in one hemithorax (see Chapter 9) . Iversen and colleagues [51] recommend one placed in the fourth intercostal space in the posterior axillary line and the other in the third intercostal space in the midclavicular line. A nonrecycled system has been described for thoracic lavage. [51] One chest tube is infused with 3-L bags of heated normal saline (40 to 41 °C [104 to 105.8 °F]) using a high-flow fluid infuser (e.g., Level-1 fluid warmer, Technologies, Inc, Marshfield, Mass). The effluent is collected with an autotransfusion thoracostomy drainage set (e.g., Pleur-evac, Deknatel A-5000-ATS, Fall River, Mass) and the removable reservoir repeatedly emptied as needed. Iversen and associates [51] were able to deliver fluid at an average rate of 180 mL/min for 2.5 hours using this system. Hall and Syverud described a simple and effective method of using large volumes of tap water and two chest tubes with gravity drainage. [56] The use of a single chest tube system using a Y-connector arrangement similar to gastric lavage is also effective. Aliquots of 200 to 300 mL with a 2-minute dwell time followed by suction drainage (at 20 cm $H_2O$) are recommended.

Closed-chest massage is used until adequate spontaneous perfusion occurs. Closed-chest defibrillation may be required in the patient warmed to 30 °C (86 °F) with persistent ventricular fibrillation. Thoracic lavage is generally continued until the patient's temperature approaches 35 °C (95 °F).

**Open thoracic lavage.**

A left thoracotomy is performed as described in Chapter 17 . Saline warmed to 40 to 41 °C can then be continuously poured into the thoracic cavity, bathing the heart while an assistant suctions the excess fluid from the lateral edge of the thoracotomy. Alternatively, fluid may be added to the thorax and mediastinum intermittently and suctioned after several minutes, and more warm saline may be added (see Fig. 71-8) . This technique also allows for direct myocardial temperature monitoring. Direct cardiac massage is used until adequate spontaneous perfusion occurs. Direct cardiac defibrillation may be required in the patient warmed to 30 °C with persistent ventricular fibrillation. When defibrillation is successful, direct myocardial warming should continue until the patient's temperature approaches 35 °C. If defibrillation is unsuccessful at a core temperature of 30 °C, further warming is warranted while oxygenation, perfusion, and other physiologic parameters are optimized before further defibrillation attempts.

**Conclusions**

Although thoracic lavage clinically has not been investigated in a controlled fashion, it has been anecdotally successful in hypothermic arrest patients.  Animal studies of closed thoracic lavage suggest that it is an effective rewarming technique. [41] [50]

Nonetheless, thoracic lavage should be restricted to those patients with inadequate spontaneous perfusion to permit less aggressive techniques and in the setting where more definitive therapy (e.g., cardiac bypass) is not rapidly available.

**Cardiac bypass**

The use of cardiac bypass or an extracorporeal shunt through either the femoral artery-femoral vein or aorta-caval procedure can result in rapid rewarming but requires surgical expertise, availability of appropriate equipment, and technical support. [2] [3] [22] This procedure has not been compared with other rewarming methods in a controlled fashion. Its main advantages would appear to be the rapid rate of warming it produces and optimal patient oxygenation and perfusion. Femoral flow rates of 2 to 3 L/min with the warmer set at 38 to 40 °C (100.4 to 104 °F) will raise the core temperature 1 to 2 °C every 3 to 5 minutes. [57] Drawbacks include potential delays in assembling the appropriate team and equipment, delays due to the time necessary to complete the operation, complications from the operation, the expense of the procedure and bypass equipment, and the potential for infection. Its use in extreme situations that may include cardiac arrest should be based on individual characteristics of the patient, physician team, and hospital resources, with the full knowledge that no consensus concerning its use presently exists. If rapidly available, it should be strongly considered in hypothermic patients with asystole or ventricular fibrillation. [58] If oxygenation is not a consideration, venovenous rewarming with an extracorporeal venovenous rewarmer can achieve rapid rewarming rates (2 to 3 °C [3.6 to 5.4 °F]/hour) but slower than cardiopulmonary bypass. [59] Such a device is relatively easy to use, uses readily available technology, and probably does not require heparin. However, it needs to be assembled prior to patients presenting with hypothermia.

**Hemodialysis**

Core rewarming by hemodialysis has been achieved after placement of a dialysis catheter or with use of an existing shunt. Some of the potential advantages and drawbacks of cardiac bypass also apply to this procedure, although slower warming rates have been reported. For patients who have ingested a dialyzable toxin, hemodialysis can be used to both remove the toxin and rewarm the blood. [60] In such cases its use may be appropriate.

**Experimental Techniques**

Radiowave rewarming appears to be a rapid, safe, noninvasive technique with promise in animal studies. [55] However, the technique seems less effective than immersion therapy and equivalent to passive rewarming techniques in a volunteer study. [61]

Very hot intravenous fluids (65 °C [149 °F]) have been used in animals with little vascular damage or hemolysis and may have promise for future human trials.

# SPECIAL SITUATIONS

## Cardiac Arrest

Rapid rewarming and restoration of cardiac rhythm is essential for patients in cardiopulmonary arrest and can best be achieved by a combination of passive and multiple active core rewarming techniques. Because numerous cases of survival from hypothermic cardiac arrest with prolonged external cardiac compression exist, [37] [48] thoracotomy is not mandatory but does offer some theoretical advantages, such as increased cardiac output with open-chest massage, [63] direct observation of cardiac activity, and direct warming of cardiac tissue with thoracic cavity lavage of warm fluid. The optimal rate of cardiac compression in hypothermia is not known, but because of decreased oxygen consumption of vital organs, the rate required in hypothermic cardiac arrest may be less than that recommended in normothermic cardiac arrest. However, there is no evidence that standard CPR is harmful in the hypothermic patient; therefore, we recommend standard CPR for nonperfusing hypothermic patients.

The duration of CPR depends on the time required to raise the core temperature to a level at which defibrillation should be successful (i.e., >30 °C [86 °F]), but other factors that may occur in individual cases make any specific temperature recommendation for cessation of CPR efforts impossible. Certainly survival is highly unlikely in patients with a core temperature <10 °C (50 °F), and patients who persist in asystole or go from ventricular fibrillation to asystole as they are warmed past 32 °C (89.6 °F) are unlikely to respond. Elevated potassium levels >6.8 mmol/L [64] and blood urea nitrogen levels may portend an irreversible process [11] [22] and should be measured in the early stages of resuscitation. However, whereas high plasma potassium levels have considerable prognostic value in crushed, hypothermic avalanche victims, they are less valuable in patients with hypothermia from cold water submersion. [65]

Isolated reports of survival with prolonged CPR in hypothermic patients make extended efforts to resuscitate such patients reasonable. Under ideal conditions, hypothermic cardiac arrest patients may reasonably be admitted to an intensive care unit for a 4- to 5-hour trial of rewarming with CPR in progress. Manual CPR should be replaced by mechanical methods if equipment is available (see Chapter 16) . Absence of responsiveness to treatment in conjunction with a highly elevated potassium level is an indication for termination of resuscitative efforts.

## Airway Management

A secure functioning airway must be maintained for the hypothermic patient, just as in any critically ill patient. In mild hypothermia, heated humidified oxygen can be delivered effectively by a face mask. The hypothermic patient can be combative and uncooperative and may require arm restraints if a mask is used. For the patient with decreased sensorium who cannot reliably maintain his or her airway or the hypothermic patient who may be hypoxic, endotracheal intubation may be performed safely without the added risk of ventricular dysrhythmias. [20] [37] The technique for endotracheal intubation depends on the specific presenting circumstances and the expertise of the operator. Once an endotracheal tube has been placed and secured, it may be used for

treatment of the patient with warm humidified oxygen.

## Acid-Base Disturbances

The interpretation of arterial blood gases in the hypothermic patient has been the cause of some confusion. Because of dehydration, tissue hypoxia, increased lactate production, and impairments in acid excretion, metabolic acidosis can occur in hypothermic patients. Concurrent ventilatory decreases may increase the systemic acidotic state. Although temperature correction of reported arterial blood gas results to reflect the hypothermic state has been described, the use of *uncorrected* pH to guide therapy with bicarbonate or hyperventilation has more recently been advocated.  This approach appears appropriate to support optimal enzymatic function. A gradual correction of acid-base imbalance will allow for the increased efficiency of the bicarbonate buffering system as the body warms. Arterial pH did not correlate with patient death in the Multicenter Hypothermia Study [37] and should not be used as a prognostic guide to resuscitation.

## Coagulopathies

Abnormal clotting frequently occurs in hypothermia probably because cold inhibits the enzymatic coagulation cascade. [57] Platelet function is also impaired during hypothermia because the production of thromboxane B2 is inhibited. Hypercoagulability with risks of thromboembolism may also occur, but the main importance of cold induced coagulopathy is in patients with coincidental trauma. Such patients are often noted to have bleeding from traumatic injuries that is difficult to control. Replacement of appropriate clotting factors and use of warm blood may limit further blood loss and worsening of hypothermia.

## Trauma and Hypothermia

It is clear that there is an increased mortality in trauma patients with temperatures <32 °C (89.6 °F). It is not clear that this increased mortality is actually a result of hypothermia or that hypothermia is merely an indicator of severe injury and response to a massive transfusion of cold fluid. [68] Patients with severe trauma are prone to hypothermia because their injury often may expose them to environmental heat loss. Concurrent alcohol intoxication may add to the heat loss. Severe injury victims also lose heat because of exposure during resuscitation and rapid administration of cold fluids.

The degree to which correcting the hypothermia improves outcome is unknown. Nevertheless, devices to rapidly infuse warm fluids such as the Level 1 fluid warmer (Level 1 Technologies, Rockland, Mass) and the Thermostat 900 (Arrow International, Reading, Pa) are frequently used to warm large volume fluid transfusions. Although not subjected to controlled trials, these devices seem reasonable to prevent the hypothermia associated with massive transfusions (see Chapter 23) . Their use in hypothermia not associated with severe trauma is limited by the relatively low fluid requirements of environmental exposure hypothermia.

## PHARMACOTHERAPY AND MONITORING

Cardiac irritability, particularly refractory ventricular fibrillation, may be present on the patient's initial arrival or may occur during rewarming. Bretylium tosylate is an antidysrhythmic agent that has been used successfully to treat two hypothermic patients in ventricular fibrillation [69] [70] and may prevent ventricular fibrillation in patients at risk for this dysrhythmia. [71] However, routine administration of this medication to hypothermic patients has not been studied on humans in a controlled fashion. Magnesium also has been used successfully to defibrillate hypothermic ventricular fibrillation. [72] Other pharmacologic agents, including lidocaine, have limited value for ventricular dysrhythmias in hypothermia. [73]

Even though digitalis may be less dysrhythmogenic in hypothermia for a given blood level, there is no clear indication for its use. [74] Most supraventricular dysrhythmias, including atrial fibrillation, convert spontaneously as the patient is warmed and therefore do not necessarily require treatment in the hypothermic state. Dopamine has been used in reports of successful resuscitation of hypotensive hypothermic patients, [73] and there is some experimental support for its use in hypothermia patients needing inotropic support. [75]

Intravenous fluid should be given early because most hypothermic patients have intravascular volume depletion. Dextrose 5% with normal saline has been advocated as the ideal initial resuscitation fluid. [5] Potassium should be avoided until electrolytes are measured and normal renal function is confirmed.

Placement of a Swan-Ganz catheter and close monitoring of urinary output may assist in the fluid management of severely hypothermic patients. The risks of precipitating ventricular fibrillation should be weighed against the potential benefits of the Swan-Ganz catheter.

Elevation of creatine phosphokinase in hypothermic patients may indicate rhabdomyolysis, and careful monitoring of renal function with aggressive fluid replacement may prevent the development of renal failure.

Finally it should be noted that hypothermic patients exhibit a "physiologic" (and probably somewhat protective) hypotension, hypoventilation, and bradycardia, the extent of which depends on the core temperature. The clinician should avoid aggressive therapies or medications that are aimed at providing the hypothermic patient with vital signs that would be desirable in the normothermic patient but which may be supraphysiologic in the hypothermic patient.

## FROSTBITE

Hypothermic patients frequently suffer from frostbite in addition to their systemic hypothermia. Frostbite usually involves the hands, feet, ears, and nose, and the exposed skin goes through a number of progressively deleterious physiologic changes

on exposure to the cold. Mild cold injury is termed *frostnip*, a condition that involves only the skin, sparing the subcutaneous tissues. The skin is blanched and numb, but the injury is immediately reversible with no permanent sequelae if the area is quickly rewarmed. Nonfreezing temperatures also produce *trenchfoot*, an intermediate in the progression to true frostbite.

In frostbite the skin and subcutaneous tissue are actually frozen. The affected extremity is hard, solid, and blanched. Clear or hemorrhagic bullae may be present. Initially there is no pain or feeling in the frostbitten extremity. After rewarming, the affected area develops severe edema and blistering, eventually exhibiting dry gangrene and mummification, leading to tissue sloughing.

Physiologically, frostbite involves the initial extracellular formation of ice crystals, followed by delayed but progressive ischemia in the affected area. Interstitial ice crystal formation is a hypertonic state that leads to cellular dehydration and cell death. Cellular water loss enhances extracellular ice formation, leading to further damage from increased pressure on blood vessels and cells. Cold also increases blood viscosity, promotes vasospasm, and precipitates microthrombus formation. The cascade of injurious events in frostbite may be mediated by prostaglandins.

Rapid rewarming is the treatment of choice for frostbite. [76] The aim is to limit the length of time the tissue remains in the frozen state. The most practical way to rewarm an extremity is to totally immerse the area in warm water at 40 to 42 °C for 15 to 30 minutes. The affected area should be carefully protected to ensure that the tissue is not additionally injured through contact with the sides or rim of the container. After thawing, the area should be meticulously protected from injury. An extremity should be elevated and cotton or gauze placed between the toes/fingers to limit maceration.

The use of topical aloe vera (a thromboxane inhibitor) and systemic antiprostaglandins (such as ibuprofen) may be helpful. There is no proven benefit to the use of heparin or low-molecular-weight dextran, vasodilators, corticosteroids, or immediate surgical sympathectomy. The value of thrombolytic agents is unknown. The use of antibiotics is controversial, although some authors advocate penicillin prophylactically. Debridement of tissue should be avoided in the ED.

## CONCLUSION

As a general guideline, one should take a conservative approach to rewarming the stable hypothermic patient, with avoidance of overtreatment and the selective and careful use of invasive monitoring. The patient's "physiologic" hypotension, hypoventilation, and bradycardia should be evaluated with regard to that which is expected for the core temperature.

In moderate hypothermia, underlying problems should be sought, passive rewarming and basic support started, and less invasive core rewarming begun. This approach should include mask ventilation with warm humidified air or oxygen in the conscious patient and intubation and ventilation in the unconscious patient. In selected patients,

gastric or peritoneal lavage with warm fluid may be considered.

For severely hypothermic, *unstable* patients, cardiac bypass and thoracic lavage may offer additional benefits, including rapid warming rates and direct heart warming. The benefits should be weighed against the time, expense, and danger of complications that these procedures entail.

It is ironic that as we consider costly procedures for resuscitating hypothermic patients, many of whom are elderly and alcoholic, we have not considered preventive measures. In fact, many of the same patients who become interesting hypothermic cases visit EDs as part of a daily quest for shelter. Some physicians may take pride in sending these ED "abusers," without obvious acute medical problems, back out into the streets to make room for "real" sick patients, whom we can help. However, it may well be that the most effective ED procedure to combat hypothermia is to provide a chair in the waiting room on a cold winter night.

# Chapter 72 - Hyperthermia Procedures

**Timothy B. Erickson, Dwight E. Helmrich, Scott A. Syverud**

Human epidemics of heat-related illness have been well documented historically. [1] A Roman army was decimated by heat in 24 B.C., and King Edward's heavily armored crusaders were defeated by "heat and fever" during the final battle of the Holy Land. In Peking, 11,000 residents died during a heat wave in 1743. More than 1000 heat-related deaths occurred during a pilgrimage to Mecca in the early 1960s. In the United States in 1980, 1983, and 1988 (years with prolonged heat waves), 1700, 556, and 454 deaths, respectively, were attributed to heat exposure. [2] More recently, a large number of hyperthermia-related fatalities (465) was reported in Chicago during a severe heat wave in July 1995. [3]

In contrast, malignant hyperthermia and neuroleptic malignant syndrome (NMS) have been recognized and described only since the 1960s. [4] These conditions are largely iatrogenic and are most commonly triggered by modern pharmacologic therapy. In addition, the incidence of hyperthermic conditions induced by psychostimulant drugs of abuse, such as cocaine and amphetamine derivatives, is on the rise. [5]

Heatstroke remains a common clinical problem with significant morbidity and mortality. As is the case with accidental hypothermia, published clinical studies of heatstroke therapeutic techniques have largely been limited to retrospective case series without concurrent control or comparison groups. A variety of cooling techniques have been advocated since World War II. Although some cooling techniques have been compared in controlled human and animal models of heatstroke, practice decisions are not solely based on the theoretical rate of cooling. Other important factors include the ease of use, rapidity of initiation, and safety.

Before considering the various cooling techniques, it is essential that the underlying disorder of hyperthermia be clearly understood. Heat illness presents a spectrum of disease ranging from mild heat exhaustion (primarily a volume loss disorder) to severe heatstroke (with thermal-related end-organ injury). The latter includes disorders such as malignant hyperthermia and NMS. Treatment of this spectrum of disease requires a discriminating approach, including supportive care only for heat exhaustion and rapid cooling for heatstroke. Malignant hyperthermia requires specific pharmacologic therapy (e.g., dantrolene) in addition to cooling measures. A brief discussion of hyperthermic disorders is therefore necessary before describing cooling techniques.

## NORMAL THERMOREGULATION

Body temperature is a balance between heat production and heat dissipation. Heat is produced as a by-product of all metabolic processes and when ambient temperatures exceed the body temperature. The body temperature increases when the rate of heat production exceeds the rate of heat dissipation. In response to rising core temperature, the thermal center (located in the preoptic nucleus of the anterior hypothalamus) activates efferent fibers of the autonomic nervous system to produce vasodilation and

increase the rate of sweating. Vasodilation dissipates heat by convection, and sweat dissipates heat by evaporation. Hyperthermia occurs when thermoregulatory mechanisms are overwhelmed by excessive metabolic production of heat, excessive environmental heat, or impaired heat dissipation. In contrast, fever occurs when the hypothalamic setpoint is increased by the action of circulating pyrogenic cytokines, causing peripheral mechanisms to conserve and generate heat until the body temperature rises to the elevated setpoint. Hyperthermia and fever cannot be differentiated clinically on the basis of magnitude of temperature or on the pattern of its changes.

In the ensuing discussion, temperatures will be given in degrees Celsius (centigrade). A temperature conversion scale is provided in the previous chapter (see Fig. 71-2) .

## TYPES OF HYPERTHERMIA

### Mild Heat Illness

Heat cramps and heat exhaustion are induced by a hot environment. [14] [15] In general, the body's heat dissipation mechanisms are able to keep up with heat production and absorption in these disorders. Symptoms are largely due to the mechanisms used by the body to dissipate heat, and body temperatures remain at or near normal. Rapid cooling techniques are *not* required, and supportive care and hydration in a cool environment are usually adequate therapy.

*Heat cramps* are intensely painful but generally benign, involuntary skeletal muscle spasms (most often in the calves). The pain may also be in the legs, arms, and back. The cramps may be severe and prolonged and rarely may lead to rhabdomyolysis. Heat cramps occur after strenuous exercise or heavy labor in a hot environment. Diaphoresis with inadequate hypotonic fluid replacement (water) is a common historical point. Rest in a cool environment and vigorous oral fluid replacement with isotonic solutions are usually adequate therapy, but in some cases IV saline is required. The pain may be surprisingly resistant to narcotics in the absence of adequate fluid replacement.

*Heat exhaustion* is a poorly defined syndrome with a spectrum of nonspecific symptoms that occur after heat exposure. Malaise, flu-like symptoms, orthostasis, dehydration, nausea, and headache may all occur. As distinct from the more severe heat disorders, mental status is normal and body temperature is normal or mildly elevated. Rehydration (usually IV), rest, and supportive care in a cool environment are adequate therapy for heat exhaustion. Recovery is usually evident within a few minutes to hours. Occasionally heat exhaustion is accompanied by heat cramps, presenting a confusing scenario if the diagnosis is not suspected. Rapid cooling techniques are not required, but patients should be observed for progression to heatstroke.

### Heatstroke

When the body's normal heat dissipation mechanisms are overwhelmed, core temperature elevation and heatstroke rapidly ensue. Two forms of heatstroke are described in the literature. *Classic (nonexertional) heatstroke* usually occurs during summer heat waves. Poor, urban elderly, infants, and persons with impaired mobility are at greatest risk. Dehydration, lack of air conditioning, marginal cardiovascular status, impaired mentation, and medications that interfere with heat dissipation (e.g., phenothiazines and anticholinergics) predispose this population to heatstroke. *Exertional heatstroke*, a consequence of strenuous physical activity, usually afflicts a younger segment of the population. Highly motivated, poorly acclimatized, or unconditioned athletes and military recruits are common victims, as are individuals who perform heavy physical labor in hot, humid conditions.

The degree of hyperthermia necessary to produce heatstroke in humans is unknown. In tissue culture cells, thermal injury is observed in the range of 40 to 45 °C. Studies of hyperthermia in cancer therapy reveal that tissue sensitivity to heat is increased by relative hypoxia, ischemia, and acidosis. [21] The duration of thermal insult, measured as degree-minutes, is also an important variable. [22]

The key clinical findings in the diagnosis of heatstroke are (1) a history of heat stress or exposure, (2) a rectal temperature greater than 40 °C, and (3) central nervous system (CNS) dysfunction (i.e., altered mental status, disorientation, stupor, seizures, or coma). Although anhydrosis is traditionally described as a classic sign of heatstroke, recent investigations have demonstrated that cessation of sweating is only a latent sign. Failure to consider the diagnosis of heatstroke in a diaphoretic patient with mental status changes could prove disastrous.

The sequelae of heatstroke are caused by thermal damage to multiple organ systems. [24] Delirium, seizures, and coma can result as direct effects of heat on the CNS. Cardiovascular collapse results from dehydration, maximal cutaneous vasodilation, and direct heat-induced myocardial depression. Coagulopathies and liver dysfunction (elevated levels of bilirubin and transaminases) occur as consequences of thermal breakdown and consumption of serum proteins, as well as direct heat damage to hepatic cells. Children often demonstrate diarrhea. Renal failure can result from myoglobinuria (related to rhabdomyolysis) and acute tubular necrosis. [24]

The treatment of these sequelae in acute heatstroke does not differ from that in other disorders, with the sole exception that rapid cooling is necessary to prevent further damage and reverse heat stress. Importantly, the human body tolerates hyperthermia poorly. Unlike patients with hypothermia, in whom slow, gentle rewarming and supportive care often result in a favorable outcome, victims of severe heatstroke must be aggressively treated with measures designed to rapidly lower the core temperature.

## Malignant Hyperthermia

Malignant hyperthermia results from a rare inherited autosomal dominant abnormality of the skeletal muscle membrane. [25] In response to certain stresses or drugs (Table 72-1) , patients with this disorder sustain a massive afflux of calcium from skeletal muscle

sarcoplasmic reticulum, resulting in contraction of the sarcomeres; skeletal muscle rigidity; increased skeletal muscle metabolism and heat production; and finally, systemic hyperthermia. Hyperthermia is a late development occurring after rigidity has been present for

| **TABLE 72-1** -- Triggers for Malignant Hyperthermia | |
|---|---|
| **Drugs** | **Conditions** |
| Halothane | Heat stress |
| Methoxyflurane | Vigorous exercise |
| Enflurane | (?)Emotional stress |
| Diethyl ether | |
| Cyclopropane | |
| Succinylcholine | |
| Tubocurarine | |
| Lidocaine | |
| Mepivacaine | |

| | |
|---|---|
| Isoflurane | |
| Ketamine | |
| Trichloroethylene | |
| Chloroform | |
| (?)Gallamine | |
| (?)Nitrous oxide | |

some time and the body's normal heat dissipation mechanisms have been overwhelmed. The earliest signs of malignant hyperthermia are increased $CO_2$ production, muscle rigidity, and tachycardia. Cardiac output and cutaneous blood flow also increase to maximize heat loss. Malignant hyperthermia is diagnosed based on the clinical triad of (1) exposure to an agent or stress known to trigger the condition, (2) skeletal muscle rigidity, and (3) hyperthermia.

Malignant hyperthermia is usually encountered in the operating room while patients are undergoing general anesthesia, particularly with halogenated inhalation agents and depolarizing muscle relaxants. However, cases of malignant hyperthermia may be encountered anywhere general anesthetics or neuromuscular blocking agents are used. [27]

As with heatstroke, treatment of malignant hyperthermia requires rapid cooling and supportive care for the sequelae described previously. Unlike heatstroke, malignant hyperthermia requires specific pharmacologic therapy to stop excess heat production by skeletal muscle. Dantrolene sodium induces muscle relaxation in malignant hyperthermia by blocking calcium release from muscle cell sarcoplasmic reticulum. [28] In all cases of malignant hyperthermia, the inciting stimulus (see Table 72-1) should be discontinued immediately and dantrolene therapy administered (2.5 mg/kg IV every 6 hours) until the episode resolves. [25]

It has been suggested that dantrolene administration might speed cooling of heatstroke victims by reducing skeletal muscle heat production. [29] However, in a canine study of externally induced hyperthermia, dantrolene administration did not result in faster

cooling rates than those seen with spontaneous room air cooling alone. [30] Although 1 small study of exercise-induced heatstroke in humans during a religious pilgrimage suggested that dantrolene may speed cooling rates in exertional heatstroke, [31] a larger controlled trial in a similar setting found no benefit. [32] Currently, dantrolene administration is best reserved for patients with clinical muscle rigidity or suspected malignant hyperthermia. Routine use of this drug in heatstroke patients is not recommended.

## Neuroleptic Malignant Syndrome

First described in the late 1960s, NMS is characterized by fever, muscle rigidity, altered level of consciousness, and autonomic instability. [4] This idiosyncratic disorder follows the therapeutic use of neuroleptic drugs, including phenothiazines, butyrophenones, thioxanthenes, lithium, and tricyclic antidepressants. The reaction is triggered by blockade of dopaminergic receptors, resulting in spasticity of skeletal muscle, which generates excessive heat and impairs hypothalamic thermoregulation and heat dissipation. [12] Muscle rigidity, described as "lead-pipe" rigidity in its most severe form, [4] can manifest as oculogyric crisis, dyskinesia, akinesia, dysphagia, dysarthria, or opisthotonos. Temperatures can exceed 42 °C. Initial agitation often progresses to stupor and coma. Catatonia and mutism may also be present. Autonomic instability is manifested as tachycardia, labile blood pressures, sweating, and incontinence. Ventilations may be impaired by chest wall rigidity.

This syndrome is more likely to occur at the initiation of or after an increase in neuroleptic dosage. It may also occur if antiparkinsonian drugs are suddenly discontinued. NMS resembles malignant hyperthermia but usually takes considerably longer to develop (2 to 3 days) and lasts longer (5 to 10 days) after the inciting drug is discontinued. The syndrome may be misinterpreted as a worsening of the underlying psychiatric disorder, drug intoxication (e.g., cocaine, amphetamines), a severe dystonic reaction, tetanus, or a variety of CNS infections. The mortality rate is high (20%), and death is usually caused by respiratory failure, renal failure, cardiovascular collapse, or thromboembolic disease. Unlike malignant hyperthermia, NMS is thought to be caused by a central disorder of thermoregulation.

Treatment of severe NMS (i.e., hypotension, hyperthermia, marked rigidity) closely follows that of malignant hyperthermia, except that therapy must be maintained for several days until symptoms resolve. Discontinuation of the triggering agent, rapid cooling, and supportive treatment for ensuing organ failure remain the cornerstone of therapy. Although the effects are not immediate, pharmaceutical therapy is directed at overriding the dopaminergic blockade caused by offending neuroleptic agents or dopamine depletion resulting from the cessation of antiparkinsonian medications. As with malignant hyperthermia, dantrolene (2 to 3 mg/kg) can be given to treat NMS-induced muscle rigidity. [25] The beneficial response stems not only from the effects at the sarcoplasmic reticulum of skeletal muscles, but also from central dopamine metabolism of calcium in the CNS. [33] In addition, bromocriptine, a central dopamine agonist, has been reported to be efficacious in NMS therapy at doses of 2.5 to 10 mg three times a day. Although both of the above agents have been noted to reduce the duration of hyperthermia, neither has been evaluated in well-controlled trials.

A more recently described disorder that is often confused with NMS is the *serotonin syndrome*. This syndrome involves the newer antidepressants (fluoxetine and sertraline), which are selective serotonin reuptake inhibitors. These drugs can adversely react with other stronger serotonin receptor agents such as monoamine oxidase inhibitors (MAOIs) to induce a clinical presentation similar to that of NMS, only milder. Treatment is primarily supportive and consists of prompt recognition and withdrawal of the offending agent.

### Hyperthermia and Psychostimulant Overdose

As mentioned above, the recognized incidence of hyperthermia induced by sympathomimetic psychostimulant drugs of abuse is on the rise. [5] The offending agents most commonly described are cocaine, phencyclidine, amphetamine, and the amphetamine derivatives such as 3,4-methylenedioxymethamphetamine (MDMA) ("ecstasy") and 3,4-methylenedioxyethamphetamine (MDEA) ("Eve"). [39] Hyperthermia is a common feature of these potentially severe to lethal poisonings and may be the primary cause of fatality in many cases. [5] Some have applied a pathophysiologic model of exertional heatstroke or NMS to profound cocaine intoxication. In addition to profound hyperthermia (>42 °C), acute rhabdomyolysis, renal failure, coma, seizures, and death have been described in these patients. As demonstrated by Roberts and colleagues, even a patient with a core temperature of 114 °F due to acute cocaine intoxication may survive with aggressive cooling methods. [45]

Treatment requires prompt recognition and general supportive care, coupled with rapid cooling, as outlined below, and the aggressive use of sedative and/or paralyzing agents to control agitation. Importantly, the longer that psychostimulant-overdosed patients remain hyperthermic, the higher their morbidity and mortality rates. Agitation and seizures must be chemically controlled, as they lead to continued generation of heat and muscle injury. Therefore, liberal doses of benzodiazepines are recommended. [35] Some have advocated the use of dantrolene [46] and bromocriptine [35] as for malignant hyperthermia and NMS, but their efficacy in the setting of drug-associated hyperthermia remains controversial.

### Hemorrhagic Shock and Encephalopathy Syndrome

The condition of hemorrhagic shock and encephalopathy (HSE) in children (mainly infants, but some older children) resembles heatstroke in adults. The full-blown syndrome includes hyperthermia, coagulopathy, encephalopathy, and renal and hepatic dysfunction. Although there may be an association with concurrent viral illness, the condition generally follows a temperature elevation, which may be triggered by the "bundling" of a child with a low-grade fever. Therapy is largely supportive and includes volume replacement and rapid cooling of the hyperthermic child while sources of bacterial infection are sought and treated.

## COOLING TECHNIQUES

**General Considerations**

Heatstroke mortality is proportional to the magnitude and duration of thermal stress measured in degree-minutes. [51] *Delay in cooling may represent the single most important factor leading to death or residual disability in those who survive.* [52] In addition, advanced age and underlying disease states are significant contributing factors. [53]

Many exertional heatstroke victims are volume depleted and may present with hypotension. As a result, initial stabilization with cooled (room temperature) IV fluids and correction of electrolyte abnormalities are valuable in the hypotensive patient. Traditional sources recommend a rate of 1200 mL over the first 4 hours, [54] whereas others advise a 2 L bolus over the first hour, with an additional 1 L/hour for the following 3 hours. [55] Seraj and colleagues have challenged this more aggressive recommendation. [56] In their study of pilgrims who suffered heatstroke, 65% had a normal or above normal central venous pressure (CVP) measurement on arrival. They found that an average of 1 L of saline was sufficient to normalize the CVP during the cooling period in their patients, who had a mean age of 55 years (range, 31 to 80 years). Hence, in older patients fluid resuscitation should be monitored carefully to avoid pulmonary edema.

Regarding antipyretics, *there is no indication for either salicylates or acetaminophen in the setting of heatstroke,* as their efficacy depends on a normally functioning hypothalamus. In addition, overzealous use of acetaminophen could potentiate hepatic damage, and salicylates may promote bleeding tendencies. [23]

Given that rapid cooling is accepted as the cornerstone of effective heatstroke therapy, the clinician must choose which cooling technique to use. Studies in animal models have been based on the assumption that the fastest cooling technique is the best. In clinical patient care, other factors will also influence the choice of technique. Patient access, monitoring, safety, ease of use, and availability are all considerations, in addition to speed of cooling. A technique that may not be the most rapid but allows easy patient access and is readily available may be preferable to more cumbersome (albeit more rapid, once established) cooling techniques in some clinical settings.

The cooling rates achieved in various human and animal studies of heatstroke are summarized in Table 72-2 . The relative advantages and disadvantages of various cooling techniques are outlined in Table 72-3 .

*In addition to the cooling procedures outlined below, it is imperative that the clinician institute the judicious use of sedation and/or muscle paralysis, which are required to control agitation and to make the patient receptive to sometimes unpleasant therapies.* In general, IV benzodiazepines are the easiest and safest first-line drugs used for sedation.

## Indications for Rapid Cooling

Rapid cooling should be instituted as soon as the diagnosis of heatstroke (rectal temperature >40 °C, altered mental status, history of heat stress or exposure) is made. Rapid cooling is also indicated for the treatment of malignant hyperthermia and NMS but should be instituted concurrently with discontinuation of the triggering agent or drug and administration of dantrolene. Because studies show that the degree of organ damage correlates with the degree and duration of temperature elevation above 40 °C, a reasonable clinical goal is to reduce the temperature to below 40 °C within 1 hour of the start of therapy. [53]

## Contraindications for Rapid Cooling

Rapid cooling, per se, is never contraindicated in the presence of heatstroke. Immersion cooling is relatively contraindicated when cardiac monitoring of an unstable patient is required or when limited personnel make constant patient supervision impossible. Iced gastric lavage is contraindicated in patients with depressed airway reflexes unless the airway is protected by endotracheal intubation. [57] Gastric lavage is also contraindicated by conditions that preclude placement

| TABLE 72-2 -- Cooling Rates Achieved with Various Cooling Techniques | | | |
|---|---|---|---|
| **Technique** | **Author(s)/Year** | **Model** | **Rate (°C/min)** |

| | | | |
|---|---|---|---|
| Evaporative | Weiner & Khogali/1980 [59] | Human | 0.31 |
| | Al-Aska/1987 [62] | Human | 0.04 |
| | Kielblock et al/1986 [60] | Human | 0.09 |
| | Wyndam et al/1959 [58] | Human | 0.034 |
| | White et al/1987 [76] | Dog | 0.14 |
| | Daily & Harrison/1948 [64] | Rat | 0.93 |
| | White/1993 [73] | Dog | 0.18 |
| Immersion (ice water) | Weiner & Khogali/1980 [59] | Human | 0.14 |
| | Wyndam et al/1959 [58] | Human | 0.14 |
| | Magazanik et al/1980 [67] | Dog | 0.27 |
| | Daily & Harrison/1948 [64] | Rat | 1.86 |
| | Costrini/1990 [66] | Human | 0.15 |

| | | | |
|---|---|---|---|
| Ice packing (whole body) | Kielblock et al/1986 [60] | Human | 0.034 |
| | Bynum et al/1978 [70] | Dog | 0.11 |
| Strategic ice packs | Kielblock et al/1986 [60] | Human | 0.028 |
| Evaporative strategic ice packs | Kielblock et al/1986 [60] | Human | 0.036 |
| Cold gastric lavage | Syverud et al/1985 [57] | Dog | 0.15 |
| | White et al/1987 [76] | Dog | 0.06 |
| Cold peritoneal lavage | Bynum et al/1978 [70] | Dog | 0.56 |
| | White/1993 [73] | Dog | 0.18 |

| **TABLE 72-3** -- Advantages/Disadvantages of Various Cooling Techniques | | |
|---|---|---|
| **Technique** | **Advantages** | **Disadvantages** |

| | | |
|---|---|---|
| Evaporative | Simple, readily available | Constant moistening of skin required |
| | Noninvasive | |
| | Easy monitoring and patient access | |
| | Relatively more rapid | |
| Immersion | Noninvasive | Cumbersome |
| | Relatively more rapid | Patient access and monitoring difficult |
| | Low mortality rates reported | Shivering |
| | | Poorly tolerated by conscious patients |
| Ice packing | Noninvasive | Shivering |
| | Readily available | Poorly tolerated by conscious patients |

| Strategic ice packs | Noninvasive | Relatively slower cooling |
|---|---|---|
| | Readily available | Shivering |
| | Can be combined with other techniques | Poorly tolerated by conscious patients |
| Cold gastric lavage | Can be combined with other techniques | Relatively slower cooling |
| | | Invasive |
| | | Requires airway protection |
| | | Human experience limited |
| Cold peritoneal lavage | Rapid cooling | Invasive |
| | | Human experience limited |

of an orogastric or nasogastric tube. Cold peritoneal lavage is relatively contraindicated when multiple previous abdominal surgeries make placement of a lavage catheter risky owing to potential bowel perforation (see Chapter 46) .

**Evaporative Cooling**

Evaporating water is thermodynamically a much more effective cooling medium than melting ice. Evaporating 1 g of water requires 540 kcal. Melting 1 g of ice requires only 80 kcal. In theory, therefore, evaporative cooling should be approximately 7 times more efficient than ice packing. In practice, evaporative cooling is more efficient, but not nearly to the degree thermodynamics would predict. [58] In separate human studies, Wyndam and colleagues and Weiner and Khogali found evaporative cooling to be 1.5 to

2.2 times faster than ice water immersion. [58] [59] In human volunteers with mild, exercise-induced hyperthermia, Kielblock and associates found evaporative cooling no faster than whole-body ice packing. [60] In clinical practice, ice water immersion or ice packing causes heat loss by conduction, as well as by heat consumption by the phase change of melting ice.

Despite the continued enthusiasm of some clinicians for ice water immersion, *evaporative cooling is the fastest noninvasive cooling technique in human studies*. To maximize evaporative cooling rates, several factors must be optimized. [61] Air flow rates must be high (large fans are required). The air must be warm (but *not* humid), as evaporation is decreased at lower temperatures. The entire body surface must be exposed to airflow and continuously moistened with water (ideally the patient is suspended in a mesh sling to expose the back to airflow and moisture). Finally, the temperature of the water used to moisten the skin must be tepid (15 °C). If the water is ice cold, evaporation will be slowed. Conversely, if it is hot, conductive heat gain may occur.

Weiner and Khogali have constructed a sophisticated "body cooling unit" (BCU) to maximize evaporative cooling. [59] Patients in the BCU are suspended in a mesh net. High airflow rates (30 m/min) at temperatures of 45 °C are maintained both anterior and posterior to the mesh net. Atomized water at 15 °C is continuously sprayed on all body surfaces. Using this device, Weiner and Khogali achieved rapid cooling rates under laboratory conditions. [59]

The realities of clinical practice make these conditions hard to reproduce. Half the body surface (the back) will usually be unavailable for evaporative cooling. Airflow rates and temperatures are usually limited by the ambient temperature in the treatment facility and by the size and power of the fan available. These realities are reflected in the slower cooling rates Al-Aska and coworkers achieved when treating heatstroke victims with evaporative cooling in a clinical setting (see Table 72-2) . [62]

**Procedure**

Evaporative cooling is accomplished by undressing the patient completely, positioning a fan or fans (usually at the foot of the bed or stretcher) as close to the patient as possible, and then sponging or misting the skin continuously with tepid 15 °C water. A single care provider can continue the technique and monitor the patient once cooling has been initiated. It is important to keep as much body surface as possible moist and exposed to airflow. Covering sheets or clothing will impede skin evaporation and cooling. At one center, the downdraft of a helicopter has been used as an evaporative cooling adjunct. [63]

**Complications**

Complications of evaporative cooling are rare and are more often attributable to the underlying disorder than to the cooling technique. Wet skin may interfere with electrocardiogram (ECG) monitoring, but this can usually be avoided by using electrodes on the patient's back. Shivering occurs infrequently with this technique when

compared with other cooling techniques, because the water is relatively warm (15 °C). [62] Evaporative cooling should be discontinued when the rectal temperature reaches 39 °C. Continued cooling beyond this temperature may lead to subsequent "overshoot hypothermia" due to continued core temperature drop after active evaporative cooling is discontinued. [24]

**Immersion Cooling**

It would seem obvious that the fastest way to cool a heatstroke patient would be immersion in ice water. In one of the first studies of heatstroke cooling techniques, Daily and Harrison demonstrated that rats with hyperthermia cooled faster with ice water immersion than with evaporative cooling. [64] Some contemporary sources continue to recommend ice water immersion as the cooling technique of choice for heatstroke, usually citing Daily and Harrison's work as a reference. [65]

Studies in humans comparing ice water immersion with evaporative cooling have demonstrated evaporative cooling to be considerably faster. An explanation of the discrepancy between Daily and Harrison's results and subsequent human studies can be inferred. Daily and Harrison studied rats with intact coats of fur who were heated until they collapsed in a hot, humid environment. They then compared the cooling rate observed with ice water immersion with that observed when the rat was dunked in ice water and then held in front of a fan. [64]

Clearly, cooling a rat with an intact coat of fur is not the same as cooling a human heatstroke victim. The higher body surface area-to-mass ratio of the rat would make conductive heat loss in ice water relatively more efficient than in humans. Fur impedes evaporative heat loss and is not a factor in humans. Finally, a single dunking in 0 °C water is not the optimal technique for evaporative cooling.

More recently, Costrini and colleagues reported no fatalities in 252 consecutive young marine recruits who were treated for exertional heatstroke over a 15-year period with ice water immersion within 20 minutes of diagnosis. [66] They regard ice water immersion as superior in reducing mortality rates when compared to other conventional methods described in the literature. However, these other descriptive case series reporting higher mortality rates also included older patients, some with underlying disease states, and unknown times of cooling initiation. [66]

In clinical trials, cold water immersion remains the second fastest noninvasive cooling technique available (see Table 72-2) . In situations where evaporative cooling is not available, immersion may be the cooling technique of choice.

Several factors are important in maximizing the rate of immersion cooling. Conductive heat loss is dependent on cutaneous blood flow to maintain a heat gradient from skin to water. Theoretically, contact with ice water causes skin and subcutaneous (SQ) vasoconstriction, blocking heat exchange and turning these structures into insulators. [15] Intense cutaneous vasoconstriction will impede conductive heat loss. Magazanik and associates, in a canine study, have suggested that warmer water (15 °C) may actually cool faster than ice water (0 °C) owing to this factor. [67] The optimal water temperature

for cooling human heatstroke patients has not been defined.

Regardless of the water temperature, it is clear that increasing surface area increases conductive heat loss. Maximizing the body surface area in contact with the water will increase cooling rates with immersion cooling. In clinical practice, this means that complete immersion of the trunk and extremities will cool the patient faster than partial immersion of the trunk (back only) with the extremities extended out of the bath.

**Procedure**

Immersion cooling is accomplished by undressing the patient completely prior to transfer to a tub of water of a depth sufficient to cover the torso and extremities. Various water containers have been used. A regular bathtub, if available, can be used. Most clinical reports have described tubs that can be moved to the emergency treatment area when needed. A child's plastic wading pool and a decontamination tub or stretcher with waterproof sides and drainage capability are examples of the latter approach. The patient's head must be continuously supported out of the bath. Temperature and ECG leads must be securely attached to the patient if monitoring is to be continued during immersion. The patient should be removed from the bath when rectal temperature reaches 39 °C, because core temperature will continue to drop for a short period, even after the patient is removed. An electronic temperature monitor with a long flexible rectal probe is useful for continuous temperature monitoring during immersion.

**Complications**

The common complications of immersion cooling are patient shivering, cutaneous vasoconstriction, and the loss of monitoring capability. Shivering generates considerable heat through muscle metabolism. Cutaneous vasoconstriction impedes conductive heat loss. If significant shivering does occur, it can be reduced with benzodiazepine agents such as diazepam. Although the use of phenothiazines such as chlorpromazine has been advocated for shivering in the past, their use is currently discouraged, because administration of these agents also may impair heat loss by their anticholinergic effects on sweat glands, contribute to hypotension via alpha-adrenergic blockade, lower the seizure threshold, and cause dystonic reactions. In addition, they possess central dopamine-blocking effects that may exacerbate symptoms of NMS.  Benzodiazepines are also valuable if the patient is hyperthermic secondary to sympathomimetic agents such as cocaine. Magazanik and coworkers [67] have also suggested that warmer water temperatures (15 °C) will minimize shivering and increase cutaneous blood flow, thereby increasing cooling rates. Interestingly, Costrini and colleagues reported that shivering occurred only when patient temperatures fell below 36.7 °C and was rarely problematic.

Patient monitoring is a problem under water. Electrodes can be used on the nonimmersed upper shoulders. ECG artifact often becomes a major problem during vigorous shivering. Immersion cooling is not recommended for patients with unstable cardiac rhythms or patients who are at risk for developing these rhythms. A significant change in cardiac rhythm might go undetected during the labor-intensive process of

immersion cooling.

Patient access for resuscitative procedures is also a major problem with this technique. Should the patient develop ventricular fibrillation, he or she must be removed from the bath and dried prior to defibrillation. Invasive and diagnostic procedures (e.g., IV access and radiography) cannot be performed during the cooling period. Care must be taken to avoid displacement of IV lines during placement in and removal from the bath.

As body temperature drops, mental status will improve in many heatstroke victims. When awake, most people find ice water immersion difficult to tolerate. IV sedation may be required.

Finally, this technique is labor intensive. Several caregivers must be present throughout the process. The patient's head must be maintained out of the bath. If massage is used, one or more individuals will need to immerse their own hands in water to continuously massage the patient. Medications should be given IV, and constant attention to temperature and ECG monitors is also necessary. This cooling technique should be used only if adequate personnel are available.

**Whole-Body Ice Packing**

Packing the heatstroke victim in ice may enhance conductive heat loss without the attendant logistical problems caused by water immersion (Fig. 72-1) . Constant attendance, as required for skin moistening with evaporative cooling and as described for immersion cooling, may not be necessary with ice packing. Kielblock and colleagues demonstrated in a human study of mild, exercise-induced hyperthermia that whole-body ice packing cooled just as fast as evaporative cooling (see Table 72-2) . [60] Whether these results will also be observed in heatstroke victims with much higher body temperatures remains to be determined. Further study of this technique is required before a clear recommendation can be made regarding its value in relationship to other heatstroke therapies.

**Procedure**

Whole-body ice packing is accomplished by undressing the patient completely and then covering the extremities and torso with crushed ice. As with any cooling technique, constant temperature monitoring using an electric thermometer and a long, flexible rectal probe is recommended.

The ice should be removed, and the patient should be dried off when the rectal temperature reaches 39 °C. A large supply of crushed ice will be needed whenever this technique is used. Whole-body ice packing can usually be performed on the emergency department (ED) stretcher without additional equipment. As with immersion cooling, ECG monitoring can potentially be difficult owing to shivering artifact and displacement of electrodes.

Alert patients usually do not tolerate ice packing well, and IV sedation or restraint is

usually required. Excessive shivering can be treated with benzodiazepines if the rate of cooling is decreased.

Logistically, ice packing may be problematic. Ideally the patient is placed in a container that facilitates ice contact with the skin and prevents water from dripping onto the floor. This is best accomplished by placing the patient in a child's lightweight plastic pool, which is available in toy stores. Lacking this equipment, plastic cloths or trash bags may be placed under the patient with the edges curled up to form a sling-like apparatus.

## Strategic Ice Packs

Noakes has suggested that selective placement of ice packs over areas of the body where large blood vessels run close to the skin may be an effective cooling technique. [69] Cooling in these areas would occur despite cutaneous vasoconstriction, owing to direct conductive heat loss from the blood within the vessel, across the vessel wall, SQ tissue, and skin, to the ice. The most common areas used for strategic ice packing are the anterior neck (carotid and jugular vessels), the axilla (axillary artery and vein), and the groin (femoral vessels). Kielblock and associates' study of this technique reported a cooling rate slightly slower than that seen with evaporative or whole-body ice pack cooling (see Table 72-2) . [60] In addition, application of ice packs, although easier to perform than immersion or total-body ice packing, limits the conductive cooling offered by the latter two procedures. [24] However, one clinical trial demonstrated that a combination of strategic ice packs with evaporative cooling resulted in faster cooling than either technique alone, although the relative increase achieved by adding ice packs to evaporative cooling was small. [60]

In unconscious patients or in awake patients who can tolerate ice packs without excessive shivering, this technique could be added to evaporative cooling. However, the clinical value of strategic ice packs alone or in combination with other techniques remains to be determined. Anecdotally, during the Chicago heat wave of 1995, the majority of heatstroke patients who presented to EDs survived after being effectively cooled using the evaporation method accompanied by strategic placement of ice packs.

### Procedure

This technique is best accomplished by placing large plastic bags filled with crushed ice or an ice water mixture in both axillae and over both femoral triangles. If the neck is used, the packs must be placed laterally, with care taken not to compress the trachea or apply excessive weight over the carotid arteries. The neck area should probably not be packed in the presence of carotid bruits or a history of cerebrovascular disease. Some sources advocate rubbing the body surface briskly with plastic bags containing ice after the body has been wet down with water. This is effective, provided it is combined with evaporation therapy. [52]

### Complications

Complications of strategic ice packing are limited to shivering and patient discomfort as

described for whole-body ice packing previously. The ice packs should be removed when rectal temperature reaches 39 °C to avoid excessive core temperature drop.

## External vs Core Cooling

All of the external cooling techniques described previously are noninvasive and use heat loss by evaporation or conduction across the skin as the primary cooling mechanism. With each of these techniques, dropping of the central temperature will continue even after the technique is discontinued and the skin is dried. This is due to a delay in the establishment of an equilibrium between the cold skin and the core. The amount of "core afterdrop" can exceed 2 °C. For this reason, cooling should usually be discontinued when the core temperature reaches 39 °C.

Because the sites of significant cell damage with heatstroke are centrally located (e.g., liver, kidney, heart), central cooling techniques theoretically are preferable to external techniques. Iced gastric lavage and peritoneal lavage are the two central techniques that have been studied in both animal models and human heatstroke. Cooling via the respiratory tract has been studied in animals but not investigated clinically. [74] Central cooling techniques are necessarily more invasive than external techniques and therefore have the potential for more significant complications.

## Cold Gastric Lavage

The stomach lies in close proximity to the liver, great vessels, kidneys, and heart. The gastric mucosa is not subject to the intense vasoconstriction observed on skin exposure to ice water. [75] For these reasons, lavage of the stomach might be expected to be an effective central cooling method. In a canine model, lavage with ice water at a rate of 200 mL/min produced cooling rates that were slower than those seen with evaporative cooling. [76] Human heatstroke victims have been successfully cooled with gastric lavage, but only in combination with external techniques. [71] Cold gastric lavage seems best suited for use in patients with severe hyperthermia who are cooling at a slow rate with external techniques alone. The presence of an endotracheal tube and the passage of a large-bore gastric tube make rapid lavage without aspiration possible. This technique should be reserved for patients whose airway is protected by endotracheal intubation and who do not have a contraindication to gastric tube placement (see Chapters 43 and 45) .

### Procedure

Cold gastric lavage is best accomplished by instilling 10 mL/kg of iced tap water into the stomach as rapidly as possible (usually over 30 to 60 seconds). After a 30- to 60-second dwell time, the water is removed by suction or gravity. [57] Cooling will theoretically be faster if a high temperature gradient is maintained in the stomach. To this end, the lavage should proceed quickly. A faster lavage rate is usually maintained if suction is used to withdraw instilled fluid. A large container of ice-temperature water maintained 1 to 1.5 m above the patient's body will facilitate instillation of fluid. This container should be directly connected to the lavage tubing and should ideally allow passage of water but not ice, which may occlude the tube. Since large volumes of water

are needed, it is helpful if additional ice can be added to the container without interrupting the lavage. A large syringe can be used as an alternative to gravity instillation, but this is usually slower.

A simple system that accomplishes this procedure can be devised from readily available equipment in most EDs. A standard lavage setup (for use in drug overdoses) and a large-bore gastric tube are used. The lavage bag is cut open at the top to allow water and ice to be added. It is then suspended above the patient's body and connected to the orogastric tube by Y tubing with clamps. The other arm of the Y tubing is connected to suction. Using the clamps, ice water can intermittently be instilled by gravity and withdrawn by suction.

### Complications

A major potential complication of cold gastric lavage is pulmonary aspiration. The use of a cuffed endotracheal tube should minimize the incidence of this complication. Owing to the large volume of water used and the frequent depression of airway reflexes seen with severe heatstroke, this technique should rarely be used in a patient who is not endotracheally intubated.

If tap water is used, water intoxication, hyponatremia, and other electrolyte disturbances are *potential* complications. Water is absorbed from the stomach and, with large-volume lavage, may pass the pylorus into the small intestine. In canine studies, large-volume gastric lavage with tap water did not cause electrolyte abnormalities. [57] The actual incidence of these potential complications in human heatstroke has not been determined. The use of normal saline instead of tap water would eliminate this potential problem.

Theoretically, the passage of cold water through the esophagus, located directly behind the heart, has the potential to induce cardiac dysrhythmias. Dysrhythmias have not been observed in canine studies or in case reports of human heatstroke victims cooled with this technique.

## Cold Peritoneal Lavage

The surface area and blood flow of the peritoneum greatly exceed those of the stomach. Peritoneal lavage would therefore be expected to exchange heat much faster than is possible with gastric lavage. Bynum and coworkers' study of peritoneal lavage (6 °C fluid) in dogs demonstrated the fastest cooling rate ever reported in large animal or human studies (see Table 72-2) . [70] A case report of peritoneal lavage cooling in human heatstroke also demonstrated rapid cooling. [72] As with gastric lavage, this central cooling technique offers the advantage of directly cooling the core organs that are most susceptible to thermal damage. Unlike with gastric lavage, endotracheal intubation is not required.

Peritoneal lavage is a more invasive cooling technique. Surgical placement of the lavage catheter is necessary. Since heat exchange is more efficient across the

peritoneum, smaller volumes of fluid can be used. This cooling technique is relatively contraindicated by conditions that preclude placement of a lavage catheter (e.g., multiple abdominal surgical scars) (see Chapter 46) .

Peritoneal lavage is the most rapid central cooling technique. It can theoretically be combined with other techniques to speed cooling of the heatstroke patient with refractory hyperthermia. Being the most invasive cooling technique, it requires time, proper equipment, and surgical expertise to institute. Its use is probably best suited to situations in which heatstroke patients are not responding to external cooling and adequate equipment and personnel are readily available.

In a canine investigation, White and associates demonstrated that a simple noninvasive evaporative cooling technique was just as rapid and effective as peritoneal lavage for cooling and treating heatstroke. [73]

**Procedure**

To institute peritoneal lavage cooling, 2 to 8 L of sterile saline should be immersed in an ice water bath to cool while the catheter is being placed. A standard peritoneal lavage catheter (as for diagnostic use in trauma patients) is placed using any of the techniques described in Chapter 46 . [72] Standard contraindications apply. Use of a larger peritoneal dialysis catheter may speed fluid instillation and withdrawal. Actual lavage volumes and rates have not been established. One approach is to instill and withdraw 500 to 1000 mL every 10 minutes until adequate cooling has been achieved. Rectal temperature may be falsely low during the lavage owing to the presence of cold water about the rectum at the level of the rectal temperature probe. [70] It may be preferable to monitor tympanic membrane or esophageal temperature when using this technique. The lavage should be discontinued when core temperature reaches 39 °C to avoid excessive core after-drop.

**Complications**

The potential complications of peritoneal lavage cooling are primarily related to placement of the catheter and include bowel or bladder perforation and placement into the rectus sheath rather than the peritoneum. These potential problems are discussed further in Chapter 46 .

## Other Cooling Techniques

"Rewarming" techniques are used to minimize ongoing heat loss via the respiratory tract in hypothermic patients (see Chapter 71) . [77] Although high-frequency jet ventilation (HFJV) causes core cooling in critically ill patients, [78] [79] efforts to use the respiratory tract to cool heatstroke victims have thus far been unsuccessful. In a canine model of heatstroke, the use of HFJV has shown it to be a relatively ineffective cooling technique. [74] Heat loss by convection (air transfer) is relatively inefficient compared with the conductive heat loss mechanism used by other cooling techniques. The use of dry, hot air to maximize evaporative heat loss from the lungs might cause respiratory

complications. [79]

Ice water lavage of the bladder, colon, or thorax has not been investigated as a cooling technique. Obvious logistical problems decrease the potential effectiveness of these approaches. In addition, the central cooling rate achieved would probably not be faster than the relatively slow rate seen with gastric lavage.

Hemodialysis or partial cardiopulmonary bypass could theoretically be used to cool heatstroke patients. There are no animal or clinical data in the literature evaluating these invasive central cooling techniques.

## CONCLUSION

Rapid cooling is the key step in the emergency management of heatstroke patients. Survival approaches 90% when elevated temperatures are lowered in a timely fashion. [23] The highest documented temperature in the medical literature with survival is 48.8 °C (115 °F). In this case, the patient was rapidly cooled and recovered without neurologic sequelae. [14] Evaporative cooling appears to be the technique of choice. It combines the advantages of simplicity and noninvasiveness with the most rapid cooling rates achieved with any external technique. It is also logistically easier to institute, maintain, and monitor evaporative cooling than any other cooling technique. If a patient is not cooling rapidly with evaporative cooling, other techniques can be added. Strategic ice packs can be used. If the patient is endotracheally intubated, gastric lavage can be instituted. If facilities and personnel are available, peritoneal lavage cooling can be used as a rapid central cooling technique. If muscle rigidity is present or malignant hyperthermia is suspected, dantrolene sodium should be administered. In addition, the clinician should have a heightened index of suspicion for NMS and sympathomimetic drug toxicity. Regardless of the cause, a reasonable clinical goal is to reduce the rectal temperature to 40 °C or below within 60 minutes of instituting therapy.

Immersion cooling is best limited to centers with the proper equipment and skilled medical personnel experienced in managing hyperthermic patients. This method may also be effective in conditions in which electric power for evaporative cooling is unavailable (e.g., in wilderness settings where bodies of cool water are available nearby and the victim is far from more sophisticated medical care). Other cooling techniques require further study before a clear recommendation as to their efficacy can be made.

# Chapter 73 - The Sedative-Hypnotic Interview

**Kenneth V. Iserson**

Emergency physicians occasionally encounter patients who present with complaints of sudden, nontraumatic paresis or paralysis of the extremities or even catatonia-like states. [1] Although experienced emergency physicians usually suspect an underlying primary psychiatric etiology, nagging questions about the presence of exotic or rare organic etiologies often remain. [2] This dilemma, as well as the physician's frequent inability to alleviate acute symptoms, often causes difficulty and confusion in the patient's initial disposition and treatment. The use of a sedative-hypnotic interview in carefully selected patients may quickly resolve such problems. [3] [4] The procedure can be easily accomplished in either the emergency department (ED) or the physician's office in about 20 minutes.

## BACKGROUND

Most sedative-hypnotic interviews have used amobarbital (sodium isoamyl ethyl barbiturate). Indeed the procedure is generally called the "amobarbital interview" because amobarbital is the agent of choice. Amobarbital agent was first synthesized by Eli Lilly and Co in the late 1920s. It is a moderately long-acting barbiturate with a moderately rapid induction time. [5] In 1930, W. J. Bleckwenn began using amobarbital to produce a drug-induced narcosis for the treatment of neuropsychiatric disorders. Much of the psychiatric community quickly picked up and expanded the technique under the generic term "narcoanalysis." It was generally used in institutionalized or long-term patients. World War II brought a resurgent interest in the amobarbital interview. At that time, frontline medical clinics used the technique for soldiers with acute paralysis, amnesia, aphonia, or pseudocatatonic states. At that time, Grinker and Spiegel demonstrated the technique's use for both diagnostic and therapeutic purposes. Since World War II, the psychiatric community has lost interest in narcoanalysis. Current textbooks of psychiatry give little space to narcoanalysis, and many practicing psychiatrists have little familiarity with the technique. [13] However, the emergency medicine community developed an interest in the sedative-hypnotic interview for diagnosing and treating symptoms similar to war neuroses. [14] Use of the technique has been reported for a series of ED cases of catatonic-like symptoms and conversion reactions.

## INDICATIONS

The sedative-hypnotic interview is a rapid and safe method for distinguishing and treating the functional factors that contribute to several types of symptom complexes presenting to the ED. Over the years, a variety of indications have been developed for acute outpatient use of the technique. These indications include the following:

1. To resolve conversion symptoms to avoid their "crystallization" and permanence.
2. To treat acute panic states following such traumatic events as rape, catastrophic loss, or disaster.

3. To diagnose and treat mute and unresponsive patients (benign stupor) or patients with acute hysterical amnesia.
4. To diagnose malingering.
5. To reveal suicidal ideation.
6. To gain information in criminal cases (although this is of presumed dubious merit or legal worth). [16]
7. To differentiate between organic illness or organic psychosis and functional psychosis.
8. To differentiate functional psychiatric disorders, such as multiple personality disorders or pseudodementia, from other psychiatric disorders.

Emergency physicians must have tools to differentiate organic from functional illnesses and to treat life-threatening catatonic-like states. Medical evaluation of the patient with decreased responsiveness is fraught with pitfalls. Patients who appear to be catatonic or in benign stupor have been reported to have such conditions as intracranial infections and hemorrhage, [24] endocrine abnormalities, [25] liver failure, [26] atrioventricular (AV) malformations, tumors, and drug ingestions. [27] Delay in diagnosis and therapy has led to morbidity and deaths in patients with these conditions; adverse outcomes also may be seen with acute lethal catatonia. [28]

The difficulty of differentiating organic from psychiatric findings is highlighted by the report of Gould and colleagues. [29] They found that of 30 patients with proven structural neurologic damage, all demonstrated some characteristics that are classically attributed to hysteria, including the potential for secondary gain, indifference to a serious sign or symptom ("la belle indifference"), a nonphysiologic or nonanatomic sensory or motor examination, giveaway weakness on motor testing, and improvement with reassurance. Likewise, Fishbain and Goldberg [30] described "classic conversion disorders" in three patients with limb paralysis who were subsequently found to have serious underlying organic illness, including cerebral hemorrhage. Hence, clinical examination to seek an organic cause of neurologic symptoms should precede psychiatric evaluation, even when the symptoms appear functional. However, when the clinical examination does not clearly identify an organic basis for the symptoms, a sedative-hypnotic interview may provide additional diagnostic information.

Most ED experience with the sedative-hypnotic interview has been with adults presenting with 1 of 2 specific syndromes: either hysterical conversion reactions that significantly impair the patient's functions or a catatonia-like state. The catatonia-like state is different from the common condition that Plum and Posner describe as psychogenic unresponsiveness. [31] Patients with psychogenic unresponsiveness are usually hysterical, with symptoms lasting for several minutes. They lie with their eyes closed and actively resist opening the lids. When opened, the eyelids flutter or close rapidly rather than with the smooth motion seen in coma. These patients normally respond quickly to noxious stimuli and a firm approach by the ED staff.

Patients in a catatonia-like state, however, often present either in a state of mute wakefulness without response to verbal or tactile stimuli or in a mildly stuporous condition. Patients in a state of mute wakefulness often track the observer with their eyes (coma vigil, akinetic mutism) and may show a waxy flexibility of the extremities. [25]

Those in stupor are sometimes mistakenly assumed to have a neurologic condition or to have ingested drugs.

## CONTRAINDICATIONS

Contraindications to the sedative-hypnotic interview fall into two categories: psychiatric and medical (Table 73-1) . Most psychiatric contraindications are relative rather than absolute; that is, potential benefit from the procedure must be weighed against any potential harm. Patients presenting with an overt paranoid reaction or those unwilling to passively submit to the procedure are unlikely to gain maximum benefit and may incur additional psychiatric trauma from the interview. Likewise, overeager patients may be seeking secondary gain from the procedure. Using this technique on such patients may be both dangerous and unnecessary.

Medical contraindications are both absolute (i.e., porphyria and barbiturate allergy when amobarbital is used) and relative. Relative contraindications include already being under the influence of depressant drugs and having a history of sedative-hypnotic addiction, although the interview also

| | **Medical Contraindications** | **Psychiatric Contraindications** |
|---|---|---|
| | **TABLE 73-1** -- Contraindications to an Amobarbital Interview | |
| Absolute | Porphyria Allergy to barbiturates | None |
| Relative | History of barbiturate addiction<br>Under influence of depressant drugs<br>Severe liver, cardiac, or renal disease<br>Severe hypertension or hypotension (only if >500 mg is used)<br>Pulmonary infection or edema | Paranoid reaction<br>Unwilling patient<br>Overeager patient |

can be used as a test of current addiction. However, even when there is concomitant drug ingestion, the interview may still be performed if the dose of sedative-hypnotic is very carefully titrated. Relative contraindications also include the presence of severe liver, cardiac, or renal disease. Severe hypertension or hypotension may suggest an organic cause, but use of the drug in these states poses a medical risk only if more than

the maximum safe dose (e.g., 500 mg of amobarbital sodium, IV) is given. The presence of mild pulmonary infection, pulmonary edema, or laryngitis poses only the theoretical risk of increasing the chance of laryngospasm. Generally, *in the presence of significant concomitant medical disease, it is best to forego the amobarbital interview* and to concentrate on stabilizing the condition of the patient in the ED. The interview may be performed later during the hospital stay.

## EQUIPMENT

The sedative-hypnotic interview should never be attempted unless the proper time, space, equipment, and ancillary personnel are available. A relatively quiet room, a stretcher or table with side rails up, and basic resuscitation equipment (airway, intubation equipment, bag-valve-mask setup) are the primary requirements. Intravenous access with an injection port and infusion fluid (generally 1 L of 0.9 normal saline) should be routine.

Sodium amobarbital is not the only drug that has been used in this manner. Some psychiatrists now use thiopental and mixtures of thiopental and amobarbital. [32] Others have used chloroform, *Cannabis indica*, paraldehyde, scopolamine, chloral hydrate, and most modern barbiturates for the same purpose. [33] Patients with psychogenic respiratory symptoms (often attributed to conversion disorders) have responded to benzodiazepines and other sedative-hypnotic agents. [34] However, the safety, efficacy, and dosages for optimal results using these drugs for the sedative-hypnotic interview have not been well established. Therefore, amobarbital is recommended for this procedure in the ED.

Amobarbital sodium (Amytal Sodium, Eli Lilly and Co) is supplied as a dry powder that must be reconstituted with sterile water. In reconstituting the amobarbital, it is important to rotate, not shake, the ampule. Five hundred milligrams of amobarbital sodium should be prepared as a 5%-by-weight solution by diluting 500 mg of the powder in 10 mL of sterile water. Note that because amobarbital has relinquished its role as a mainline anticonvulsant drug, many hospitals no longer keep it in stock. Its main use now is for the neurologic diagnostic Wada test (i.e., direct anesthesia of the hemicortex).

## PROCEDURE

A medical history emphasizing prior psychiatric problems, drug overdose and abuse, allergies, medications, and contraindications to the procedure must be obtained from the patient, relatives, or friends. A complete physical examination must be performed to identify any obvious organic problems. Glucose, blood urea nitrogen (BUN), electrolytes, and a complete blood count (CBC) are obtained in cases of stupor to eliminate medical causes. Appropriate specimens for toxicology should be obtained, and administration of glucose, naloxone, and thiamine should be considered for the stuporous patient. Prior records, when obtainable, should be reviewed. If a proper history, physical examination, or laboratory analysis is not possible, the procedure should not be performed. *The physician's zeal to try the procedure should not tempt him*

*or her to take shortcuts or to examine the patient hastily.*

After deciding that the symptoms are possibly of a nonorganic psychiatric origin, the clinician explains the sedative-hypnotic interview procedure to the patient or to the relatives. Reassurance should be given that amobarbital is not, in fact, "truth serum." The patient should be placed in a relatively quiet room with the patient's relative or a chaperone in attendance. It may be extremely helpful for relatives to observe the interview, because it is often difficult for them to comprehend that certain symptoms, such as paralysis, have a psychogenic basis. Successful results may further reinforce the need for the family to arrange for follow-up psychiatric care.

An intravenous (IV) line should be secured in a large peripheral vein. Sodium amobarbital (5% solution) is administered at a rate of 50 mg (0.5 mL)/min. A conversation (or monologue in the case of a stuporous patient) about benign, nonthreatening topics should be held with the patient during induction. A calm, reassuring attitude and suggestions similar to those given during hypnotic inductions are useful, because the effect of the interview may be as great as that of the medication. [35] [36]

A close similarity exists between the state produced with amobarbital and the light stages of hypnosis. [37] Some investigators have, in fact, considered it just another hypnotic medium that allows simple, direct psychotherapy with little or no analysis. [38] If a psychiatrist is present during the interview, some of the information obtained can be beneficial in future analysis. We suggest that whenever possible, the interview should be performed in consultation with a psychiatrist who will be involved in the subsequent care of the patient.

The levels of narcosis are staged using the criteria developed by Lorenz and associates (Table 73-2) . [39] The interview is conducted during stage II narcosis; stage III narcosis (absence of corneal reflexes) should be avoided. When stage II is reached, the actual interview is begun. It usually requires 250 to 500 mg of amobarbital to reach this stage, although some investigators report success with as little as 100 mg. [14]

| TABLE 73-2 -- Stages of Narcosis in the Amobarbital Interview<br>*Data from Lorenz WF, Reese HH, Washburne AC: Physiological observations during intravenous sodium amytal medications. Am J Psychiatry 13:1205, 1934.* |
|---|
| **Stage I: Induction of narcosis** |
| Patient begins to describe symptoms of the amobarbital, e.g., lightheadedness, dizziness, sleepiness, blurred vision |

| |
|---|
| **Stage II: Conduction of the interview** |
| The unresponsive patient begins to communicate or the responsive patient becomes more talkative or euphoric |
| Usually requires a total of 250-500 mg amobarbital (may occur with as little as 100 mg) |
| **Stage III: Obtundation--should be avoided** |
| Patient is unresponsive, corneal reflexes are absent |

The patient is initially questioned regarding such nonthreatening topics as identification data, current personal situation, predisposing factors, and further medical history (including drug ingestion).

The next stage of the interview is tailored to the specific problem. For example, if the patient has paralysis of an arm or leg, the interviewer suggests that the patient try to move and use the affected part. Once the paralysis is overcome, the interviewer reinforces the fact that the extremity is now back to normal and that it will continue to be normal after the patient leaves the hospital. This is analogous to the familiar "posthypnotic suggestion." It is not advisable to confront the patient with a psychiatric diagnosis at this time, even though such a diagnosis is now obvious to the family. If the patient is catatonic or unresponsive, spontaneous speech or movement will return. The clinician should emphasize to the patient that such a responsive state is normal and desirable. Patients with organic and/or toxic psychoses will not respond verbally and will merely fall asleep or become more sedated during the interview. If this occurs during the interview process, the interview can be terminated with the knowledge that the patient's symptoms should be presumed to have an organic origin. Patients who have an organic basis for paresis or paralysis will still show this deficit during the interview.

Near the end of the interview, it is suggested to the patient that he or she remember a pleasant occurrence. This helps to improve the patient's emergence from the interview. A few patients may become slightly upset during the interview. They can be given an extra 50 to 100 mg of amobarbital at the conclusion of the interview to obtain a slightly longer sleep period. [40] Respirations are the only vital sign monitored after the procedure begins. As long as sodium amobarbital is given by this protocol, there are no

significant effects on blood pressure, pulse, or respiratory rate. [39] A cardiac monitor is not required routinely, but it is advised in any patient with cardiovascular disease and in the elderly. Patients who are not hospitalized should be observed for 2 to 4 hours after the interview has been completed. Patients usually fall asleep for 2 to 3 hours following the conclusion of the interview, and when the patient is awakened, the proper disposition should be made.

Catatonic and pseudocatatonic patients who now have established psychiatric diagnoses should be admitted to a psychiatric service. Patients with resolved conversion reactions can be discharged with follow-up. When a psychiatric basis cannot be proven, the patient requires further evaluation for a presumed organic etiology.

## COMPLICATIONS

Many thousands of amobarbital interviews have been conducted with few complications, usually in settings that were not conducive to advanced life support. Nevertheless, resuscitation facilities must be readily available if this procedure is undertaken. The few complications reported have primarily been respiratory depression or apnea and were associated with too-rapid administration (i.e., >50 mg/min) or, occasionally, too much (i.e., >500 mg) of the drug. [42] Vasomotor collapse and laryngospasm also have been reported on rare occasions. (See Chapters 1 , 2 , 3 , and 4 for a discussion of the approach to laryngospasm.) The latter complications are reported to occur only in stage III (usually >700 mg amobarbital) or anesthetic levels of narcosis, and they are not reported at all in most series. Most complications are probably related to physician error or inaccurate calculation of drug doses.

## INTERPRETATION

The patient suspected of having psychiatric symptoms who does not initially respond to the amobarbital interview warrants intensive further investigation for an anatomic or physiologic basis for the deficit. Failure to alleviate symptoms with the amobarbital interview is often due to the failure of the physician to make the proper preinterview diagnosis. This usually results from an inadequate physical examination or incomplete laboratory or radiographic evaluation prior to the technique's being used. If the patient has a firm belief or delusion, such as a feeling that he or she is God or is from outer space, the amobarbital interview will not abolish that belief. Likewise, it will not render a schizophrenic normal while he or she is under the influence of the drug. [43]

Some cases of catatonia may not respond to this technique. It must be emphasized, however, that if a psychiatric diagnosis is not firmly made from the results of the interview, it is mandatory that the patient receive intensive medical care and evaluation before being referred to a psychiatric facility.

In certain organic conditions, the amobarbital also may be therapeutic. For example, drug withdrawal states or seizure disorders may be ameliorated with the technique. Generally, clinical evaluation will permit the differentiation of these "false-positive" conditions from a conversion disorder, although petit mal or partial complex seizure

disorders may be problematic. Finally, malingering may produce a "false-negative" response, as the patient may continue to deceive even an astute observer.

## ALTERNATIVE APPROACH: HYPNOSIS

For clinicians experienced in the technique, hypnosis may be a more rapid, less invasive, and less costly approach to resolving symptoms of conversion reactions and acute catatonia. The simplest induction methods involve gentle suggestions that the patient progressively relax different areas of the body, beginning with the toes. The clinician times the suggestions to correspond with the patient's breathing (i.e., with exhalation). If successful, hypnotic induction and resolution may take only minutes. Unlike stage hypnosis, the results of clinical hypnosis are obvious, if successful; the person moves or speaks. In one case, a "paralyzed" teenager who had been rushed into the ED on a makeshift backboard walked back out to greet his anxious friends <10 minutes later (on his way to his first psychiatry appointment). Hypnosis also has been demonstrated to effectively allow fracture and dislocated joint reductions in emergency medical situations. [44] Training in clinical hypnosis is widely available.

## CONCLUSION

The amobarbital interview has been shown to be a rapid and safe technique that can be readily performed by the emergency physician. It is useful for the confirmation of the psychiatric basis of stupor in patients with catatonic-like states and for diagnosis and resolution of similarly based nontraumatic paresis and paralysis. It is suggested that, when possible, the procedure be carried out in conjunction with a psychiatric consultation and (subsequent) evaluation to obtain the maximum clinical benefit.

# Chapter 74 - Bedside Laboratory and Microbiologic Procedures

Anthony J. Dean, David C. Lee

## ASSESSMENT OF URINE

### Evaluating Bacteriuria

The medical literature can be difficult to interpret with regard to urinalysis (UA) and culture for evaluation of bacteriuria and its clinical significance. Much confusion has arisen because of failure to standardize meatal cleaning procedures or collection techniques, mixing symptomatic patients with asymptomatic ones, and generalizing conclusions across varied patient groups (e.g., men vs women, boys vs girls, circumcised vs uncircumcised males). Although several studies have failed to demonstrate a statistical difference in contamination rate with various urine collection techniques, most studies show trends in favor of more meticulous and/or invasive collection methods. In general, potential contamination is most problematic when low-level bacteriuria is demonstrated in symptomatic, rather than asymptomatic, patients.

Patients with lower urinary tract infection (UTI) symptoms (e.g., urgency, frequency, dysuria, in the absence of fever) can have a UTI with $<10^5$ colony forming units (CFU)/mL in the urine. [1] [2] This important fact had been obscured by studies on *asymptomatic* patients, which demonstrated that the threshold for "significant bacteriuria" in this group is $10^5$ CFU/mL. [3] [4] *Symptomatic* patients with low-count bacteriuria should not be erroneously diagnosed as uninfected, because infection can indeed be present with as few as $10^2$ CFU/mL. [2] Furthermore, specimens are not necessarily contaminated if multiple pathogens are isolated. Up to 50% of symptomatic women may have polymicrobial infections. [5] [6]

### Obtaining a Urine Specimen

Several methods are available for obtaining a urine specimen. These can be listed in order of increasingly precise collection techniques, which tend to come at the cost of increasing difficulty and/or patient discomfort:

1. *Random voided:* Any specimen provided by the patient.
2. *Midstream voided:* No skin preparation, container placed in urinary stream 2 to 3 seconds after the initiation of micturition.
3. *Clean catch:* Same as item 1, with antiseptic cleansing of the urethral area.
4. *Midstream clean-catch* specimen (MSCC): Cleansing as in item 3, with midstream collection as in item 2.
5. *Catheterized:* Obtained from a newly placed catheter.

6. *Suprapubic aspiration* (SPA) (see Chapter 59) .

Since the mid-1950s there have been proponents and opponents for each technique, with arguments that vary according to the clinical context. When the clinician is interpreting findings of tests for bacteriuria and pyuria and culture results, it is important to be cognizant of the method of urine collection used.

The low levels of bacteriuria induced in 2% to 8% of patients after straight catheterization [7] are generally clinically insignificant. Catheterization does cause minor local injury, as reflected by bacteriuria and low-level hematuria (1 to 3 red blood cells [RBCs]/high-powered field [hpf]) in 15% of patients. [7] [8] SPA in neonates continues to be advocated by some in cases in which accurate diagnosis is essential and the risk of infection must be minimized. SPA (see Chapter 59) , has a 10% "failure rate" (i.e., no urine obtained after 3 attempts) in most series and may actually take longer than most methods, especially when multiple attempts are considered. SPA can also cause spuriously lowered leukocyte or bacterial colony counts due to the necessity of maximally filling the bladder prior to performing the procedure. [9]

**Recommendations for Urine Collection**

A selective approach (Fig. 74-1) can be taken in obtaining a urine specimen to evaluate for UTI. The first issue to address is whether the patient is symptomatic. If no symptoms of UTI are present, the urine examination can be considered as a "screening" test. Routine screening for asymptomatic bacteriuria in the general population has been shown to be unwarranted in all but 2 clinical situations: pregnant women and all patients for whom urologic surgery has been scheduled. [10] [11] Although neither of these is a major concern of the emergency physician, testing of urine frequently occurs in the emergency department (ED) in the setting of undifferentiated abdominal pain or fever.  If only a urine culture is to be performed, some would argue that any spontaneously voided specimen would suffice, since the diagnosis of asymptomatic bacteriuria depends on $10^5$ CFU of a single pathogen per milliliter of urine, and with such criteria, contaminants are usually easily identified. [12] However, performing cultures in all such patients is prohibitively costly, so that dipstick and/or UA is usually acceptable for screening. [13] [14] With these screening tests, however, contamination by bacteria, leukocytes, or erythrocytes can still cause diagnostic confusion. Thus, the advantages of a less contaminated specimen are worth the minimal extra effort of asking the patient to provide an MSCC sample. For female patients who are physically capable, the ideal position is sitting astride a toilet, facing backward. This helps to separate the labia and position the cup for a midstream specimen.

The typical ED patient is in some way "symptomatic." This includes patients with classic signs and symptoms of UTI as well as those with systemic signs of infection (e.g., fever and chills) who are unable to accurately report on symptoms. To this group should be added all patients in whom one cannot afford to miss asymptomatic bacteriuria (e.g., the immunocompromised, neonates and infants, pregnant patients, diabetics), or for whom urine cultures are going to be necessary because of a history of relapsing, recurrent, complicated, or childhood UTI. [15] In cooperative, motivated males and females with symptoms of uncomplicated lower UTI or pyelonephritis who are capable of diligently

performing the necessary maneuvers, an MSCC specimen is as accurate as a catheterized specimen, especially when the possibility of urethral and/or prostatic trauma and patient discomfort are considered. [16] In females in whom symptoms are equivocal, and in all patients unable to provide an MSCC specimen, a catheterized specimen is usually warranted.

The above approach covers the vast majority of situations. A few circumstances and techniques deserve special mention.

**Bladder Percussion and the Midstream Specimen in Infant Males**

The emergency physician is familiar with how frequently a urine stream is generated in infants who are confronted by the alarming ED environment and a cold stethoscope. Such responses can be exploited by the application of cold povidone-iodine to the genitalia with an open sterile urine container at hand. Such an approach has been shown to generate a urine sample in a median time of 10 minutes. [17] This is much less than the time needed for an average straight catheterization or suprapubic aspiration, especially since it can be performed during the history and physical examination and avoids the potential for iatrogenic complications. If the urine specimen is not immediately forthcoming, a parent can be instructed to catch the specimen in the sterile container, freeing up ED staff for other tasks.

Two techniques to actively induce voiding in infants have been described. The first, which is useful in newborns, exploits the Perez reflex. [18] After cleansing of the genitalia (with povidone-iodine or some equivalent non-irritative antibacterial agent), the infant is held in one hand while the paraspinal muscles are stroked, cephalad to caudad. This causes extension of the back, and flexion of the hips and induces micturition in <5 minutes in the majority of cases. [18]

The second technique is known as "bladder tapping." After urethral cleansing, if there is still no urine, 2 fingers may be tapped on the suprapubic area at a rate of approximately once per second for a full minute, followed by a minute's rest. The cycle is then repeated until urine is produced. The mean time prior to the production of urine is about 5 minutes. [19]

**Bag Collection in Non-toilet-trained Children**

The incidence of unsuspected UTI in the febrile neonate or infant is about 3 to 5%. Because a true UTI in an infant or child requires a subsequent evaluation for urinary tract pathology, and the disease may produce significant morbidity (e.g., hypertension, renal disease), one must be certain of the presence or absence of infection in this subgroup. Numerous studies demonstrate the disutility of urine specimens obtained for culture from a collection bag stuck to an infant's perineum. Bag specimens obtained for a routine UA in suspected UTI are equally unreliable, with white blood cell (WBC) concentrations routinely 25-fold higher and bacterial counts 1000-fold higher [21] than in a catheterized specimen. In the occasional situation in which urine is needed exclusively for chemical analysis (e.g., glucose, ketones, urobilinogen, pH, or specific gravity), a bag specimen will suffice. The utility of obtaining a bag specimen to "screen" the UA

prior to deciding on antibiotic treatment or whether to send a formal culture is only defensible if the suspicion of UTI is low, as the sensitivity for markers of a UTI (see below), is only 50 to 85%. [20] [22] In summary, an MSCC or catheter or SPA specimen is required for an accurate UA or urine culture in neonates and infants.

**Urine Specimens from Patients with Chronic Urinary Drainage Systems**

Urine obtained from any part of a chronic urinary drainage system is highly inaccurate for bacteriologic purposes. If UTI is suspected, a new catheter should be inserted and fresh bladder urine specimens subsequently obtained. [23] A small study advocating replacement of a chronically applied "Texas sheath" catheter with a fresh one was performed on subjects who did not have symptoms of UTI. [24] Such a method might be sufficiently accurate for screening of asymptomatic patients, but a Foley catheter should be used to obtain urine from patients with sheath catheters who have signs or symptoms of acute UTI.

## Urine Dipstick

Urine dipstick tests are now available to test for 10 separate parameters. Each colored square on a urine dipstick represents a complex technologic assay. It is essential to *meticulously follow the manufacturer's instructions for storage and use.* [25] Even with optimal storage and testing conditions, the false-negative and false-positive rates of these tests are problematic. In addition, most of the tests are susceptible to interference from a variety of substances (Table 74-1) .

**Method**

Urine specimens should be tested as soon as possible after they are collected. If urine has been standing, it should be stirred or shaken well, because cells rapidly sink in a container. The test strip should be completely immersed for 1 second or less. The edge of the strip is drawn along the rim of the specimen container and can also be lightly tapped to remove excess urine, thus avoiding mixing of the reagents between different test patches. Hold the strip horizontally or place it on a clean gauze pad until the recommended time has elapsed. Most strips are designed so that all of the tests can be read together after 1 to 2 minutes.

**Interpretation**

**Glucose**

The urine glucose test is normally negative. Urine glucose testing has limited usefulness in quantitative testing because the serum glucose level at which spillage occurs varies (although in most patients it starts at between 180 and 200 mg/dL). [26] Changes in urine glucose lag behind changes in blood glucose by approximately half the interval between voids. [27] Glycosuria in the absence of hyperglycemia suggests renal tubular dysfunction. Glycosuria may occur in hypothermic patients in the absence of hyperglycemia, and indeed may mask true hypoglycemia in alcoholic patients who are

hypothermic. [28]

**Ketones**

Ketones are found in the urine during starvation, inadequate carbohydrate intake, diabetic and alcoholic ketoacidosis, isopropyl alcohol poisoning, or glycogen storage disease. Urine testing for ketones is not sensitive to all forms of ketonemia or ketonuria, and the absence of a positive ketone test by dipstick should not categorically dismiss a significant ketotic state. Tests for urine ketones are 5 to 10 times as sensitive to acetoacetate as to acetone. Similar to the serum "acetone test," dipsticks do not detect 5-hydroxybutyrate, which comprises 80 to 95% of the 3 "ketone bodies," and is the predominant form in the setting of ketoacidosis. However, urine ketone testing is significantly *more* sensitive than serum ketone testing. [29] Indeed, there is generally no need to obtain "serum acetones" to diagnose or manage diabetic ketoacidosis (DKA) when urine ketone monitoring is coupled with blood gas and anion gap analyses. When an evaluation for an individual ketone body is required, serum should be specifically analyzed for acetone (the major ketone produced by isopropyl alcohol poisoning) or 5-hydroxybutyrate (the predominant ketone of alcoholic or diabetic ketoacidosis).

**Nitrites**

Normally, urine does not contain nitrites. Nitrites are specific (approximately 95%), but not sensitive (approximately 45%) indicators of UTI. Urinary nitrates are converted to nitrites most strongly by enteric coliform bacteria, explaining the nitrite test's 90% sensitivity in detecting UTI caused by *Escherichia coli*. [32] *Enterococcus*, a moderately frequent urinary pathogen, as well as *Pseudomonas* spp. and *Acinetobacter*, do not convert nitrates to nitrite and therefore are not detected by the nitrite test. False-negative results also occur because of lack of dietary nitrate, frequent voiding, and/or diuresis. [9] Early morning-voided specimens are ideal (allowing time for conversion of nitrate to nitrite), but they are rarely available in the ED; if possible, a specimen obtained 4 hours after the last voiding is preferred.

**Protein**

Proteins with molecular weight below 50,000 to 60,000 daltons can pass through the glomerulus to be reabsorbed in the proximal tubule. Normal protein passage in the urine is <150 mg/24 hours, or approximately 10 mg/dL of urine. About 10 to 33% of urinary protein is albumin, 33% is Tamm-Horsfall glycoprotein (secreted by renal tubular cells), and the balance is made up of a variety of immunoglobulins and other proteins. Proteinuria is a finding noted in about 5% of routine urine screens in men. [33] Whether this represents a normal variant is unclear, as 3 to 5% of young healthy adults have postural proteinuria (proteinuria when

**TABLE 74-1** -- Overview of Urine Dipstick Tests

|  | **Sources of Error and Artifact** | **Comments** |
|---|---|---|
| Glucose | False-negative with ascorbate, ketones, and increased SG | Hypothermia may cause glycosuria despite hypoglycemia |
|  | False-positive with peroxide and hypochlorite |  |
| Ketones | False-positive with ascorbate, levodopa, valproate, pyridium, *N*-acetyl cysteine, high-protein diet, phenylketones, phthalein compounds | Very susceptible to deterioration with humidity, causing false-negative |
| Nitrites | False-positive with contamination, pyridium | 75% false-negative rate when dipsticks exposed to air for 15 days |
|  | False-negative with high SG, ascorbate, low urine pH, dilute urine, large urobilinogen, urine standing in specimen cup >2 hr |  |
| Protein | False-positive with pH >7, and chlorhexidine | Only reliable for albumin (glomerular proteinuria) |
|  | False-negative with low pH, very dilute urine |  |

| Blood | False-positive with povidone-iodine, certain (peroxidase- producing) bacteria, hypochlorite (bleach) False-negative with high SG, and high concentrations of urinary nitrites, ascorbate, or captopril | Positive test with speckles or dots implies nonhemolyzed blood Positive test with diffuse pattern implies hemolyzed RBC or myoglobin |
|---|---|---|
| Bilirubin | False-positive with iodine, stool contamination, chlorpromazine, mefenamic acid False-negative after prolonged standing | Hard to read with agents causing marked urine discoloration |
| Urobilinogen | False-positive with pyridium, gantrisin, sulfonamides, porphyrin, methyldopa, procaine, aminosalicylic acid, 5-hydroxyindolacetic acid | Use fresh specimen; rapidly broken down by light and in acid urine |
| | False-negative with gantrisin and pyridium | |
| Leukocyte esterase | False-positive with vaginal contamination (commonest cause), oxidizing agents, eosinophils in the urine, and *Trichomonas* | |
| | False-negative with high glocuse, ketones, protein (especially albumin), pH, or SG and male sex; cephalexin, tetracycline, pyridium, oxalates, ascorbic acid, neutropenia | |

| pH | Urea splitting bacteria elevate pH | Use fresh specimen: standing raises pH by loss of $CO_2$ |
|---|---|---|
| | Run off from protein strip can falsely lower pH | |
| Specific gravity | Overestimates true SG with low pH, ketoacidosis, and protein | Not reliable at SG >1.025 |
| | Underestimates true SG due to glucose, urea, or with pH >7 | |
| S G, specific gravity. | | |

standing, but not when recumbent). [26] The urine dipstick detects negatively charged proteins much more strongly than positively charged ones; it is therefore most sensitive to albumin.

Proteinuria can be due to either systemic or renal causes. The likelihood that a positive urine protein dipstick reflects renal disease increases with an increasingly positive result, and in patients with conditions such as hypertension, diabetes, connective tissue diseases, peripheral edema, gout, or elevated serum creatinine. Mildly elevated proteinuria in patients without systemic disease or renal insufficiency who have an otherwise normal urine sediment and are asymptomatic is likely of no clinical significance. [34] In assessing a patient with proteinuria, it is helpful to divide the list of causes into those associated with hematuria and those not so associated.

The urine dipstick test is positive for protein with pyuria of >6 WBCs/hpf. [35] This is technically a "false-positive" finding but is useful when the urine dipstick is being used to screen for a UTI. Hematuria only slightly elevates urine protein levels. A 2+ to 3+ urine protein dipstick reading with only mild or microscopic hematuria should suggest a glomerular or tubular cause. [36]

**"Blood"**

The blood section of the urine dipstick shows a positive result in the presence of RBCs, hemoglobin, or myoglobin. Guidelines for distinguishing these are outlined in Table 74-2

. Moderate intravascular hemolysis does not cause hemoglobinuria, as the hemoglobin is tightly bound to haptoglobin and is therefore not filtered. Massive intravascular hemolysis gives rise to free plasma hemoglobin with a molecular weight of 32,000 daltons, which can pass through the glomerulus. Myoglobin has a molecular weight of 17,000 daltons, permitting easy passage through the glomerulus.

Urine in young, healthy volunteers contains <7 RBCs/muL (RBCs/mm3 ). [37] Studies have shown that although the

| | **Hematuria** | **Myoglobinuria** | **Intravascular Hemolysis** |
|---|---|---|---|
| Serum findings | Color: clear | Color: clear | Color: pink |
| | | Haptoglobin: normal | Haptoglobin: low |
| Urine appearance | Color: Clear to brown, clears with centrifugation | Color: Clear to red/brown; no clearing with centrifugation | Color: Clear to brown; no clearing with centrifugation |

**TABLE 74-2** -- Aids in Distinguishing Hematuria, Intravascular Hemolysis, and Myoglobinuria

| Urine microscopy | RBC, RBC casts, and protein implies glomerular source | Possible occasional RBC and tubular cells secondary to rhabdomyolysis-induced renal damage | Usually unremarkable |
|---|---|---|---|
| | No RBC casts, tubular cells, small protein: nephron source | | |
| | Just RBC: source distal to nephron | | |

urine dipstick is only 93% sensitive compared to microscopy of unspun urine at the very low level of 1 RBC/hpf, it generally is very sensitive to 10 RBCs/mm3 . [37] Hence, false-negative results are confined to clinically insignificant hematuria. [38] [39] In asymptomatic men >50 years old, significant disease can be signaled by intermittent positive dipstick hematuria, mandating careful follow-up of patients with this incidental finding. [40]

Dipsticks are vitiated by humidity and air and give false-negative results after improper storage. [41] Because RBCs lyse rapidly in urine with high pH or low specific gravity, delays in performing a microscopic UA may suggest myoglobinuria or hemoglobinuria. Microscopy or dipstick testing of a *freshly* obtained specimen can clarify this issue. The dipstick pad should be inspected for discreet positive "dots," indicative of nonhemolyzed RBCs. Conversely, urine with high specific gravity or low pH can inhibit the lysis of erythrocytes, which is necessary for the dipstick chemical reaction to occur, thus causing a false-negative result. [42] Povidone-iodine was reported to cause false-positive results in vitro, but a study reproducing typical clinical conditions demonstrated that this does not happen in vivo. [43] Iatrogenically caused trace positive results may occur after straight catheterization in 15% of cases, but at such low levels that they should not be a source of confusion with emergency urologic conditions. [8]

**Urine bilirubin**

Urine bilirubin represents the filtered, soluble, conjugated form of bilirubin. Unconjugated bilirubin is protein-bound, and does not pass through the glomerulus. Bilirubinuria is therefore due to intrahepatic or extrahepatic cholestasis. Bilirubinuria will be detected significantly earlier than clinical jaundice and thus is a sensitive test for early liver disease. Urinary bilirubin excretion is enhanced by alkalosis. A fresh sample of urine should be tested, because bilirubin glucuronide is hydrolyzed when exposed to light. Ascorbic acid and high levels of urinary nitrites decrease the sensitivity of the test

to bilirubin.

In a healthy person, conjugated bilirubin is excreted in bile. In the colon it is broken down into a number of compounds, which includes urobilinogen. Most of these compounds are excreted in the stool, giving the characteristic color. A small amount of urobilinogen is absorbed from the colon, and if it is not taken up on the first pass through the liver, it enters the circulation. Ultimately, some of this urobilinogen is filtered by the glomerulus to enter the urine, so that it is normal to have zero to moderate levels of urinary urobilinogen on dipstick testing. Most diseases causing hepatocyte dysfunction (viral or drug-induced hepatitis, cirrhosis, congestive heart failure with passive liver congestion, or cholangitis) also cause increased urinary urobilinogen excretion. This is because there is greater impairment of hepatic uptake of urobilinogen than there is of hepatic excretion of conjugated bilirubin. As a qualitative test with a wide range of normal values, it is rarely helpful, but in evaluating a patient with jaundice, it can have diagnostic significance (Table 74-3) .

**Leukocyte esterase**

This portion of the dipstick test is designed to detect enzymes from the azurophilic granules in neutrophils. Normally the test is negative. Studies report a wide and clinically important range of thresholds of sensitivity of dipstick testing, from 10 to 100 WBCs/mm3 urine. Studies suggest that the test is between 50 and 96% sensitive in detecting infection. The specificity is between 91 and 99%. The most common cause of a false-positive leukocyte esterase test is vaginal contamination.

**pH**

Average daily excretion of 50 to 100 mmol of H+ in the urine gives rise to a typical urine pH of approximately 6. Dietary protein lowers the urinary pH, whereas fruit and vegetables, especially citrus fruits, tend to increase it. The normal urinary pH ranges from 4.5 to 8. The significance of pH testing is in the assessment of normal renal function. In most states of alkalosis and acidosis, the healthy kidneys maintain homeostasis by conserving or excreting H+ . Failure

| **TABLE 74-3** -- Relationship Between Urinary Bilirubin, Urobilinogen, and Stool Color in the Jaundiced Patient | | | | |
|---|---|---|---|---|
|  | **Healthy Normal** | **Complete Biliary Obstruction** | **Intravascular Hemolysis** | **Hepatocellular Disease** |

| Urinary bilirubin | None | Elevated | None | Elevated |
|---|---|---|---|---|
| Urinary urobilinogen | None or present | None | Present, sometimes large | Normal early, Increased late |
| Stool color | Normal | Acholic | Normal | Normal |

 to do so suggests renal disease, especially renal tubular acidoses (an exception is the "paradoxical aciduria" of hypokalemic alkalosis secondary to volume contraction, hypercorticism, or diuretics). In these circumstances, the highest priority of the renal tubule is to conserve sodium. Note that pH is falsely elevated by the action of urea-splitting bacteria, especially *Proteus* spp. This can occur with "stasis" of urine, either in the bladder or in specimen cups awaiting processing in the laboratory. A persistently alkaline urine is seen in patients with struvite (triple phosphate) urolithiasis.

**Specific gravity**

The dipstick test for urine specific gravity assays for the primary urinary cations, sodium and potassium. True specific gravity, which is also dependent on anions, albumin, proteins, urea, and glucose, is therefore not measured. Artifactually low specific gravity readings are obtained in alkaline urine. Acid urine and albumin falsely elevate the specific gravity reading. [49] Some investigators believe that these strips on the dipstick test are of marginal clinical utility at best. [50] Other clinical indicators of a patient's hydration status are probably equally reliable. If necessary, a refractive specific gravitometer or a hygrometer should be used.

## Microscopic Urinalysis

Microscopic UA is done by 1 of 5 methods, considered individually below. Traditionally, the most common laboratory technique of microscopic UA for clinical purposes has been the examination of unstained centrifuged urine (technique 3). It has the advantage of concentrating formed elements that might otherwise be missed. In addition to cells and bacteria, casts, crystals, and other microbes can be recognized (Fig. 74-2) (Figure Not Available) . The disadvantage is that an individual specimen shows different amounts of formed elements after centrifugation, depending on many uncontrolled factors: the volume of the specimen, the time and speed of centrifugation, the fragility of the formed elements, the volume of the "drop" in which the pellet is resuspended after removal of the supernatant, and the size of the microscope's "high-powered field."

1. *Examination of unspun urine in a hemocytometer counting chamber* is a specialized laboratory technique. The hemocytometer uses a specially milled slide

etched with precisely measured squares, allowing for the exact enumeration of cells in each square. Because the distance between the etched surface and the coverslip is exactly known, it is possible to determine the number of cells per unit volume of specimen. Enough fresh unspun urine is placed on the slide to fully cover the counting area, and the cells are counted. There should usually be <1 WBC/mm$^3$ , [54] [55] although >10 WBCs/mm$^3$ and >5 RBCs/mm$^3$ are unequivocally abnormal counts. *Examination of unspun, unstained urine placed on a regular microscope slide* is a qualitative method sometimes used in the diagnosis of UTI. Using 1 organism/hpf as a positive result, the sensitivity and specificity in detecting $10^5$ CFU/mL are between 60% and 90%. [57] Others have used >1 WBC/*low*-powered field as a criterion for infection. [54]

2. *Examination of unstained, centrifuged urine* is performed by centrifuging 10 mL of urine at approximately 450 *g* (1000 to 4000 rpm) for 3 to 5 minutes. Roughly 9 mL of supernatant is poured off, and the pellet is resuspended in the remaining fluid. This suspension is placed on a slide with a coverslip and examined. The larger formed elements, especially casts, tend to migrate to the edge of the coverslip, and they can be seen with low magnification. One or 2 casts, depending on the clinical context, may be normal; more are not. The morphology of the more commonly encountered formed elements of urine sediment is shown in Figure 74-2 (Figure Not Available) . The significance of each is beyond the scope of this text, but this information can be found in a standard textbook of clinical laboratory procedures and diagnostic testing. [26] [27] When examining centrifuged urine, >5 WBCs/hpf seen in the middle of the coverslip has traditionally been taken as being indicative of abnormal pyuria. Most authors have estimated that 10 WBCs/mm$^3$ are equivalent to approximately 1 WBC/hpf, although this is subject to wide variations.  Various numbers of bacteria per high-powered field have been used as criteria for the diagnosis of UTI. A threshold of 10 to 20 organisms/hpf has been recommended. [57] Obviously, the larger the number of organisms seen, the greater the positive predictive value of the test. This test is also only useful in ruling out bacteriuria at the $10^5$ CFU/mL level, and it therefore does not exclude infection in symptomatic patients.

3. *Examination of Gram-stained, noncentrifuged urine* is also a semiquantitative measurement. It is estimated that 1 bacterium/hpf is equivalent to $10^5$ CFU/mL in bacterial culture. This technique is therefore also unable to rule out infection in symptomatic patients and is only 80% to 90% sensitive for $10^5$ CFU/mL.

4. *Gram stain of centrifuged urine* is probably the optimal technique, short of culture, in the assessment of bacteriuria. It is >95% sensitive and >60% specific to $10^4$ CFU/mL: an order of magnitude lower concentration of bacteriuria than the previously described methods. [57] [60] Detection of 1 organism/oil-immersion field constitutes a positive result. ] Specificity is increased to 95% if 5 organisms/hpf are seen. [57]

## Summary

Many clinicians treat female patients who present with signs of uncomplicated lower UTI without obtaining a culture, because treatment is generally benign, and about 95% of positive urine cultures grow either *E. coli* or *Staphylococcus saprophyticus.* Generally, urine dipstick and/or microscopic UA is used to confirm a UTI in this patient group. If the tests are negative, alternative diagnoses should be considered, although some

clinicians will treat empirically while awaiting the results of a urine culture. The rationale for obtaining a microscopic UA is primarily for the detection of formed elements such as cells, casts, or crystals.

In febrile infants, and chilren, pyuria less consistently accompanies infection, rendering the dipstick and UA less sensitive than in adults. Hence, as with adults, there should be a low threshold for sending cultures and/or giving empirical treatment in the presence of a negative test when clinical suspicion is high. Cultures are recommended for febrile infants younger than 2 months.

The extent to which asymptomatic patients and those in whom symptoms are poorly defined (e.g., those being evaluated for nonspecific abdominal pain) are worked up for a UTI depends on the degree of clinical suspicion. When the clinical suspicion is low, a urine dipstick analysis may be sufficient.

## TESTING FOR PREGNANCY

Pregnancy tests are based on the detection of beta-human chorionic gonadotropin (beta-hCG) in serum or urine. beta-hCG is secreted by trophoblastic cells of the placenta starting from the time of implantation of the blastocyst. Qualitative serum tests and the urine test detect beta-hCG levels of between 15 and 25 mIU/mL. [62] The concentration of hCG is usually lower in urine than in serum, which accounts for the slight advantage of serum tests in detection of early pregnancy. Optimal urine pregnancy tests are obtained on first-voided concentrated morning specimens. Home urine pregnancy test kits detect levels of beta-hCG of about 50 mIU/mL in the urine.

beta-hCG levels of 5 to 8 mIU/mL correspond to the 9th to 11th day after ovulation (23 to 25 days after the first day of the last normal menstrual period). In a viable intrauterine pregnancy, the beta-hCG level doubles approximately every 1.5 days during the first 4 weeks of gestation, reaching a serum level of 25 to 300 mIU/mL, detectable by virtually all beta-hCG-based pregnancy tests in urine or blood, on or around the first day of the missed menstrual period. The doubling rate declines to every third day thereafter, [63] [64] with the quantitative beta-hCG reaching a peak of around 100,000 to 200,000 mIU/mL between the 10th to 14th gestational weeks and declining to around 10,000 to 20,000 mIU/mL for the rest of the pregnancy (Table 74-4) . There is a wide range of beta-hCG levels among different women at the same stage of gestation, making definite clinical determinations on the basis of a single quantitative test impossible.

The rate of decline of quantitative beta-hCG after gestation varies depending on the reason for the conclusion of the pregnancy. After a term delivery, beta-hCG falls to zero in 2 weeks. After surgery for an ectopic pregnancy the range is 1 to 31 days, with a median of 8.5 days; after a first-trimester spontaneous abortion, the range is 9 to 35 days (median, 19 days); and after first-trimester elective abortion the range is 16 to 60 days (median, 30 days). [26]

Although a previous quantitative beta-hCG level is rarely available to the emergency physician, doubling rates are an important part of the assessment of a healthy first-trimester pregnancy. Fetal nonviability, ectopic pregnancy, and intrauterine demise

are signaled by abnormalities in the predicted rise in quantitative beta-hCG. A serum quantitative beta-hCG level that does not increase by 66% every 48 hours has a 75% chance of being due to a nonviable pregnancy. [67] [68] The beta-hCG levels in a healthy intrauterine pregnancy (IUP) and associated sonographic findings are listed in Table 74-5 .

The association between beta-hCG levels and gestational dates has led to the concept of a "discriminatory zone" of quantitative beta-hCG for guiding interpretation of pelvic ultrasound testing. Serum beta-hCG levels of >1000 to 1500 mIU/mL should be associated with *transvaginal* ultrasonographic signs of IUP (6500 mIU/mL for transabdominal ultrasonography), or the pregnancy is definitely abnormal, with a significant possibility of ectopic pregnancy. However, the converse--that ectopic pregnancy will always be accompanied by a beta-hCG level 1000 mIU/mL--is *not* true. Only 15% of ectopic gestational sacs examined pathologically have evidence of an embryo. This observation, combined with a variety of pathologic characteristics and sites of implantation, leads to highly variable quantitative beta-hCG levels in ectopic pregnancies. [69] In general, an ectopic pregnancy elaborates quantitatively small amounts of beta-hCG, with 1% of ectopic pregnancies having a quantitative beta-hCG of <10 mIU/mL

| TABLE 74-4 -- Quantitative Immunometric Assay for beta-hCG | |
| --- | --- |
| | **mIU/mL (IU/L)** |
| Males and nonpregnant females | <5 |
| Pregnant females (wk gestation) | |
| 1: | 5-50 |
| 2: | 50-500 |

| 3: | 100-10,000 |
|---|---|
| 4: | 1000-30,000 |
| 5: | 3500-115,000 |
| 6-8: | 12,000-270,000 |
| 12: | 15,000-220,000 |

**TABLE 74-5** -- Relationship Between Gestational Age, Quantitative beta-hCG Levels, and Ultrasound Findings in Viable Intrauterine Pregnancy

| Time Elapsed from 1st Day of Last Normal Menstrual Period | Quantitative beta-hCG Level (mIU/mL) Using the IRP | Ultrasound Findings |
|---|---|---|
| <28 days | 5-50 | |
| 4-5 wk | 50-500 | From 4.5-5 wk and beta-hCG 1000, TVU can show DDS to confirm IUP |
| 5-6 wk | 100-10,000 | Definitely abnormal if TVU does *not* show DDS after 5 wk or if beta-hCG >1500 |

| | | |
|---|---|---|
| 6-7 wk | 1000-30,000 | TAU should show DDS after 6 wk or if beta-hCG >6500 |
| 7-8 wk | 3500-115,000 | Yolk sac 5-6 wk or beta-hCG >7200; fetal pole/heart 5.5-7 wk or beta-hCG >11,000-17,000 |
| 8-10 wk | 12,000-270,000 | |

DDS, double decidual sac; IRP, international reference preparation; IUP, intrauterine pregnancy; TAU, transabdominal ultrasound; TVU, transvaginal ultrasound. Values for beta-hCG vary somewhat from Table 74-4 since dates are based on last normal menstrual period and different patient set.

and up to 8% having beta-hCG levels of <25 mIU/mL. [70] About 10% of ruptured ectopic pregnancies are associated with a beta-hCG level 100 mIU/mL. [69] In 1 series, at the time of operation, >50% of women with unruptured ectopic pregnancies and 40% of those with ruptured ectopic pregnancies had serum beta-hCG levels of <1000 mIU/mL. [69] Furthermore, patients with abdominal and/or pelvic pain or vaginal bleeding and a beta-hCG level 1000 mIU/mL have a 4-fold increased risk of ectopic pregnancy compared with those with the same symptoms and a beta-hCG level 1000 mIU/mL. [71]

Thus, it can be seen that the "discriminatory zone" can only be used to identify patients in whom ultrasound should definitely demonstrate signs of healthy IUP: a transvaginal ultrasound that fails to show an IUP with a beta-hCG level 1000 is >86% specific for ectopic pregnancy. [72] *Because of the low beta-hCG titers in ectopic pregnancy, the "discriminatory zone" cannot be used as a basis for deciding which first-trimester patients with pelvic pain and/or vaginal bleeding should receive ultrasound evaluation.*

## BLOOD CULTURES

### Indications

Blood cultures are indicated in the ED when there are clinical findings suggestive of a bacteremic state (Table 74-6) . It should be noted that 25% of patients with documented bacteremia have periods without fever. [73] In the elderly the proportion is even higher, with 50% of bacteremic patients over the age of 65 years having a temperature between

| |
|---|
| **TABLE 74-6** -- Summary of Indications for Obtaining Blood Cultures |
| **Patients with fever and:** |
| Unexplained alterations in mental status, functional status, or autonomic status in a previously healthy patient between the ages of 5 and 65 yr |
| No source, if <2 yr of age, >65 yr, or immunocompromised |
| All infants <3 mo |
| **Patients with or without fever and:** |
| Rigors |
| Toxic or "septic" appearance (e.g., unexplained hypotension, altered mental status, shock) |
| Suspicion of infectious endocarditis |
| Serious focal infections (e.g., meningitis, septic arthritis, osteomyelitis), pneumonia, or pyelonephritis with signs of toxicity |

> Unexplained alterations in mental status, functional status, or autonomic status at extremes of age and in the immunocompromised

°C (97.1 °F) and 38.3 °C (100.9 °F). Up to 13% of elderly bacteremic patients have no documented temperature, >37.3 °C (99.1 °F) at any time. [74] [75] In the elderly, vomiting, altered mental status, increasing age, and >6% band forms reported on a WBC differential cell count are predictive of positive blood culture results. [74] The subjective impression of "having fever" in adults is not a reliable indicator of the presence of fever, although the subjective impression of "no fever" is much more likely to be accurate. [76]

In children, blood cultures have been advocated to identify "occult bacteremia" in patients <2 years of age with fever >38.6 °C (> 101.5 °F) and no identifiable source. In infants <3 months with temperatures >38 °C (> 100.5 °F), many authorities recommend blood cultures regardless of the presence or absence of a source, although this approach is controversial. A child with a normal temperature in the ED and a history from the parents of tactile fever ("felt hot") should be approached in the same way as a patient with documented fever, since parents' tactile impression has been shown to be highly reliable. [77] Bacteremic children, like adults, have intermittent fever, with up to 50% afebrile rates in children with demonstrated bacteremia. [78] Children with significant clinical findings, such as lethargy or nuchal rigidity, should have blood cultures taken regardless of fever history.

**Controversy Regarding "Outpatient Blood Cultures"**

There is a long-standing debate in emergency medicine as to the utility of outpatient blood cultures (i.e., blood sent for cultures on patients subsequently released from the ED pending culture results). Arguments for and against outpatient blood cultures are summarized in Table 74-7 . The paucity of data on the subject of outpatient blood cultures leaves the issue in dispute. Opponents cite medicolegal issues, problems with follow-up, high contamination rates (an often correctable error in technique), low rates of positive cultures, and even lower rates of frequency of patients in whom therapy is changed because of culture results. Proponents also cite medicolegal concerns, positive rates similar to those seen with inpatient blood cultures, cost savings, and the benefit of diagnosing significant, yet subtle, bacteremic states (such as endocarditis).

On the basis of current data, it would seem to be fiscally extravagant to admit all patients in whom a bacteremic state is possible, and injudicious to deny blood cultures solely on the basis of a patient not appearing "toxic enough" to warrant admission. Outpatient blood cultures, with due attention to collection technique, patient selection, and diligent follow-up, would therefore seem to be an appropriate component of emergency medicine practice.

**Technique of Obtaining Blood Cultures**

Studies have demonstrated possible sources of contamination at every stage of the process of obtaining and processing blood cultures. In addition to obvious sources of contamination from the patient's and phlebotomist's skin, both antiseptic agents [82] [83] and gloves [84] have been implicated. Some authorities have argued that the primary source of contamination is in the laboratory processing of specimens. [85] However, the consensus is that the commonest source of contamination is the process of phlebotomy and inoculation of blood culture bottles. Obviously this is the single step over which the emergency physician has control. Contamination rates in most large series are around 3%; if quality assurance review reveals significantly higher rates, it would be prudent to provide special in-service training for ED personnel. [88]

A high degree of sensitivity is required of blood cultures. Many significant bacteremic illnesses have been documented with as little as 1 CFU/10 mL of blood. Human skin has a bacterial concentration between $10^3$ and $10^6$ CFU/cm$^2$ on the forearm and groin, respectively. [89] Designed to detect vanishingly low concentrations of bacteria, blood cultures are clearly susceptible to false-positive results (impaired specificity)

| **TABLE 74-7** -- Summary of Arguments for and Against the Performance of Outpatient Blood Cultures |
| --- |
| **Arguments *against* outpatient blood cultures:** |
| 1. Low true-positive rates |
| 2. True-positives are rarely clinically significant |
| 3. High false-positive rates are expensive and time-consuming for both patient and institution |
| 4. Difficulty of ED follow-up makes positive blood culture results a medicolegal liability |
| **Arguments *for* outpatient blood cultures:** |

| 1. Facilitates outpatient management of some patients, sparing them financial, psychological, and nosocomial costs |
| --- |
| 2. Allows initiation of antibiotics without fear of a potentially irrevocably lost opportunity for blood cultures (e.g., in a patient with a urinary infection, mild constitutional symptoms, and a heart murmur, infectious endocarditis is always possible, but one would want to start treatment of the UTI as the much more likely cause) |
| 3. May give useful etiologic information and antibiotic susceptibility profiles regarding infectious diseases that are being treated on an outpatient basis |

when they must necessarily be obtained by passing a needle through inherently contaminated skin. Most (80%) of the skin flora are transient, superficial, and removable; 20% inhabit the sebaceous ducts and hair follicles, and are not removable without destroying the skin. [89] [90] The former group are predominantly gram-positive and gram-negative aerobes and are the target of skin disinfectants.

The primary agents for skin disinfection are iodine compounds, alcohols, chlorhexidine, and hexachlorophene. Povidone-iodine (Betadine) 10% solution has a much lower free iodine concentration than iodine solution and is therefore less potent. The most effective cleansing agent is tincture of iodine, which is a mixture of 2% iodine solution and 70% alcohol. [92] The iodine-containing agents are superior to hexachlorophene and chlorhexidine in killing gram-negative bacteria. However, iodine, like other antiseptic agents, is inhibited by the presence of organic matter, which emphasizes the need for thorough skin cleansing prior to the application of any skin disinfectant.

Ethyl or isopropyl alcohol should be used in 60 to 80% solution. Alcohol prep pads generally contain 70% isopropanol. Alcohol is a less powerful germicide than iodine in vitro, and only kills 90% of surface bacteria after a full 2 minutes, with reapplication to prevent drying. [91] Alcohol is inactive against fungi, spores, and viruses; however, in vivo studies of blood culture contamination rates have shown it to compare favorably with iodine. [92] [93] Since tincture of iodine solution is often not available, and povidone-iodine solutions are less potent, alcohol still has an important place in skin antisepsis. In addition, alcohol is an excellent solvent, so that alcohol pads are a good tool for skin preparation prior to the application of iodine compounds.

Chlorhexidine (Hibiclens) and hexachlorophene (pHisoHex) are antiseptics that are more effective against gram-positive than gram-negative bacteria. Both agents have intradermal absorption, which causes prolonged antimicrobial activity and is the basis of their popularity as surgical scrub and operative site preparations. This also makes them preferable agents where indwelling lines, especially central lines, are being placed. [94] For routine blood culture phlebotomy, they are not as effective as alcohol and iodine

combinations. Most studies show chlorhexidine to be more potent than hexachlorophene. Prolonged exposure to hexachlorophene has been associated with seizures in infants.

The results of studies of contamination rates with various skin preparation agents are not unanimous. Table 74-8 presents a consensus skin preparation protocol. Optimal results seem to be obtained by alcohol-iodine mixtures. [89] [95] [96] The most important concept in skin disinfection is that bacteria do not die at the instant of contact with disinfectant agents. Iodine (2%), which is twice as potent as 10% povidone-iodine, requires at least 90 seconds in contact with the skin to kill 90% of surface bacteria. [91] In many ED patients it will be necessary to use alcohol prep pads to remove gross dirt and debris from phlebotomy sites prior to initiating the steps of the formal skin preparation.

## Special Considerations

### "Changing the Needle" After Phlebotomy

In considering this issue, it is important to emphasize the distinction between *needle changing* and *needle recapping.* [97]

---

**TABLE 74-8** -- Skin Preparation and Technique for Drawing Blood Cultures

1. Cleanse the skin with alcohol swabs 3 times, or until the swabs appear entirely free of surface dirt after application

2. Allow to dry

3. Apply 10% povidone-iodine, or (preferably) 2% iodine solution, or (ideally) 2% tincture of iodine in 70% alcohol, 3 times, in centrifugal circles from the anticipated site of venipuncture

4. After 3rd swab, allow to dry at least 60 sec

5. During this period, prepare lids of blood culture bottles with iodine and/or alcohol; lay out sterile gloves, and use paper wrapper as a sterile field for the needle and syringe (not necessary if using a Vacutainer system)

| |
|---|
| 6. Wipe off dry iodine at venipuncture site with alcohol; substitute chlorhexidine if placing an indwelling catheter |
| 7. Obtain 20-30 mL ( *at least* 20 mL) of blood; place up to 10 mL in each bottle (these bottles constitute 1 blood culture *set*) |
| 8. Inoculate bottles without changing needles |
| 9. Repeat at different sites for requisite number of sets for *series* |

The latter is a well-established risk to health care workers; it contravenes standard recommendations for universal precautions, *and should not be performed*. Needle replacement using the standard needle removal device on "sharps" containers is an unquantified risk, but it is clearly much less dangerous than recapping.

Based on little scientific data, [98] it was long considered essential to change the phlebotomy needle prior to inoculating blood culture bottles. With increasing awareness of the risk of bloodborne infections associated with needle-stick injuries, this practice has come under scrutiny. One large, well-controlled study showed no difference in contamination rates with or without needle changing. [99] Other studies each show a clear trend *toward* a lower contamination rate with a needle change, but no single study reaches statistical significance. In conclusion, not changing needles prior to blood culture bottle inoculation is acceptable practice for obtaining routine blood cultures. However, in those situations in which the results of the blood culture are of paramount importance (e.g., suspected infectious endocarditis, for which empiric antibiotic coverage will be started immediately), it is suggested that needles be changed prior to inoculation of culture bottles, *but not recapped*.

**Special Access Sites**

Newly placed IV catheters are a reliable source of blood culture specimens, providing that the appropriate measures are taken in skin preparation. Previously placed IV catheters (central and peripheral) and arterial lines have been compared with fresh percutaneous phlebotomy for blood cultures in several studies. Some show clearly increased contamination rates with the use of a chronic line, whereas other authors report a trend toward the same conclusion.In the aggregate, chronically indwelling vascular lines are not an adequate source of blood for culture.

**Miscellaneous Issues**

Although arterial blood may be used for blood cultures, venous blood is usually drawn for culture, even in subacute bacterial endocarditis. Because it is difficult to disinfect the groin skin, the femoral vein should be avoided whenever possible. Clotted blood or citrated blood should not be used for cultures.

**Heel Stick in Neonates**

This technique results in recovery rates of bacteria equivalent to phlebotomy in 2 studies. [110] [111] Because approximately 25% of bacteremic infants have <25 CFU/mL of blood, this proportion (25%) will be missed if 0.2 mL is obtained for culture. For this reason, heel stick should be considered as a source of last resort for blood culture. *Intraosseous specimens* may also be used in circumstances in which phlebotomy is impossible. [112]

## Timing of Blood Cultures

In most circumstances, the timing of blood cultures is a moot issue in the ED. Most patients are either so sick that the expeditious administration of antibiotics is mandated or are well enough for release home, so that 2 or more sets of blood need to be drawn immediately. The situation in which the timing of blood cultures might become a consideration is in a patient who requires admission but for whom the diagnosis of bacteremia is sufficiently in doubt that antibiotic therapy is not to be initiated empirically.

Contrary to the time-honored tradition of obtaining blood cultures during a fever spike, data show a trend toward a higher proportion of true-positive culture results in patients whose blood is drawn in the 12 hours *prior* to a fever spike. [113] [114] Furthermore, excepting infectious endocarditis, most clinically significant bacteremia is thought to be intermittent, so that multiple sets of cultures obtained at one time would heighten the risk of missing the period of bacteremia. [87] Therefore, for patients admitted to the hospital with the tentative diagnosis of bacteremia, it is theoretically advantageous not to draw all 3 sets of blood cultures in the ED, but to draw them at intervals over the following 12 to 24 hours. [115] However, if administration of antibiodies is clinically indicated, 2 or 3 sets of cultures separated by 20 to 30 minutes are obtained in the ED prior to initiation of antibiotic therapy.

## Blood Culture Volumes

**Volumes in Adults**

Multiple studies demonstrate that the sensitivity of blood cultures is directly related to the volume of blood cultured. [116] [117] [118] One representative study [119] showed that culturing a total volume of 20 or 30 mL of blood yielded, respectively, 38% or 62% more true-positive results than did a volume of 10 mL of blood. Another study showed that each additional milliliter of blood yields an average of 3% more true-positive results. [120]

This finding is also consistent with the fact that 40% of adults with bacteremia have <1 CFU/mL of blood, and that 20% have <1 CFU/10 mL. [121] Alternatively expressed, if only 10 mL of blood is obtained for culture, 20% of patients with continuous bacteremia will be missed. Because most bacteremia is intermittent, and because endogenous factors in blood will cause some inhibition of bacterial growth even with modern lysis- and filtration-centrifugation techniques, the false-negative rate in clinical practice will always be significantly higher. On purely mathematical grounds, 10 mL per set of blood cultures (5 mL per bottle) is an absolute bare minimum for culture. In adults, most authorities recommend that at least 30 mL of blood be taken per culture site and/or set and that each bottle be inoculated with 10 mL.

To ensure dilution of the blood's inherent antibacterial properties (e.g., immunoglobulins, complement, WBCs), blood should be placed in the culture medium with a concentration <10% (i.e., not >1 part of blood to 10 parts of medium). [123] Thus, if 30 mL of blood is obtained from 1 site, it should be equally divided into three of the usual 100 mL broth bottles.

**Volumes in Children**

A blood volume of 30 mL drawn from a 70 kg adult is equivalent to 1.5 mL of blood in a 3.5 kg neonate. It has been shown that levels of bacteremia are typically 10-fold higher in infants than in adults. Generally, the sicker the child, the greater the likelihood of a high level of bacteremia. [125] Although one study [127] failed to show any effect on the rate of detection of bacteremia with increasing volumes of blood specimens, many studies have suggested that small culture volumes are at increased risk of false-negative results. Furthermore, the studies showing high rates of colony-forming units per milliliter were performed on neonates. As the immune system matures during infancy, levels of bacteremia might be expected to fall toward those seen in adults. In 2 recent reviews, it was recommended to obtain a similar volume of blood with respect to body mass as would be drawn in adults: approximately 1 mL per 2.5 kg (4 mL per 10 kg of body mass). These recommendations are summarized in Table 74-9 .

## How Many Sets of Blood Cultures Should Be Tested?

A single *set* of blood cultures is the total volume of blood obtained from a single site that is inoculated into media. A 1 mL specimen from a neonate placed in an aerobic bottle, and a 30 mL specimen from an adult divided equally (10 mL per bottle) between fungal, aerobic, and anaerobic bottles, are both a *single set* of blood cultures. Two or more *sets* of blood cultures make up a *series*. [115] [133] The information derived from the blood culture sets is pooled in such a way as to make both the sensitivity and specificity of the series greater than that of the component sets. Sensitivity is enhanced because even with continuous bacteremia, an individual

**TABLE 74-9** -- Optimal Specimen Volumes to Be Drawn per Blood Culture Set D

| Age Group/Weight (kg) | Ideal Volume of Specimen per Set (mL) |
|---|---|
| Neonates | 1-2 |
| Infants/5-10 | 2-4 |
| Children/7-20 | 3-8 |
| Children/>20 | 10 |
| Children/>40 and adults | 20 |
| | 30 |

Do not place more than 10 mL of blood in each 100 mL broth bottle.

set is usually only 80% sensitive. [133] Specificity is improved by determining whether pathogens that are also frequently contaminants are found in >1 set of the series.

While this conceptual process is applied to all blood culture series, the focus of inquiry varies depending on the infectious process being ruled in or out. For example, in an elderly patient with fever and purulent urine, it is extremely unlikely that the causative organism is a typical skin contaminant. The usual causes of "false-positive" blood cultures will therefore be easily recognized, thus lowering the false-positive rate for the series and making for a test with intrinsically higher specificity. At the same time, with typical pathogens in this clinical context being nonfastidious organisms, sensitivity is typically around 99% with 2 sets of 20 mL blood per set. [122] Conversely, in a patient with a prosthetic heart valve, fever, and signs of septic emboli, many likely pathogens are also skin contaminants (this phenomenon lowers the specificity of each individual blood culture set), so at least 2 *sets* of cultures will need to be *positive* with such organisms before the overall test result (i.e., of the *series*) is considered positive. At the same time, this clinical picture indicates a very high pretest probability of disease (diminishing the negative predictive value of a negative set), so that an extremely sensitive overall test (i.e., *series*) will be needed to adequately rule out disease. Thus, in this clinical context, most authorities would recommend four sets of blood cultures, with good volumes in each.  Except in infants, single sets of blood cultures are of insufficient sensitivity or specificity to be of any utility, and they should not be drawn. Recommended numbers of sets of blood cultures as they relate to the pretest probability of disease and causative organism are summarized in Table 74-10 .

**Aerobic vs Anaerobic (vs Other) Bottles**

Anaerobic infections, by their nature, tend to occur in poorly perfused tissues or locations, frequently evolving into abscesses, which further isolate them from the bloodstream, decreasing the likelihood of bacteremia, and making them intrinsically elusive to blood cultures. In addition to these pathophysiologic considerations, a significant decrease in the proportion of positive blood cultures due to anaerobic

organisms has been widely reported over the past 15 years.

| No. of Sets (Minimum) | Clinical Context |
|---|---|
| **TABLE 74-10** -- Numbers of Blood Culture Sets to Be Obtained for an Adequate Series in Various Clinical Situations in Adults | |
| 2 sets | Etiology is likely to be easily distinguished from contaminants *and* pretest probability of bacteremia is low to moderate |
| 3 sets | Skin contaminants are possible causes of infectious process, *or* pretest probability of bacteremia is high, *or* infectious endocarditis is a consideration, but with low to moderate pretest probability |
| 4 sets | Possible infectious endocarditis where *either* the pretest probability is moderate to high *or* the patient has recently been on antibiotics |

| **TABLE 74-11** -- Infectious Processes That Can Cause Anaerobic Bacteremia |
|---|
| Odontogenic head and neck infections |
| Aspiration pneumonia |
| Abdominal/pelvic infections |

| | |
|---|---|
| Deep soft tissue infections (e.g., myofasciitis) | |
| Sepsis with decubitus ulcers or necrotic tissue | |

In recent series, anaerobic pathogens account for only 1 to 5% of positive blood cultures. Clinically significant isolates--those that could not have been predicted on the basis of the clinical picture, or that alter management--are much rarer. [136] Thus, with a typical true-positive blood culture rate of 7% and an anaerobic true-positive rate of 3% of those, approximately 500 *series* of blood cultures need to be drawn for every significant anaerobic blood culture. Since, as noted above, the true-positive rate for each blood culture set is increased by approximately 3% per additional milliliter of blood, and at least 95% of these true-positive findings will be caused by aerobic pathogens, [138] there is a strong argument to be made for devoting the entire specimen to aerobic culture unless there are clinical grounds to suspect anaerobic infection. The vast majority of anaerobic bacteremias occur in one of the clinically identifiable situations listed in Table 74-11 .

The issue of selective use of anaerobic blood cultures is controversial, with some authors still advocating their routine use. [124] [140] However, the arguments for a selective approach are compelling. Based on these considerations and analysis of a number of recent review articles, Table 74-12 suggests guidelines for inoculation of blood obtained for culture.

**Identifying Contaminants**

The emergency physician must be prepared for telephone calls from the laboratory with positive results of cultures obtained on previous shifts. "False-positive" blood cultures

| TABLE 74-12 -- Blood Culture Bottle Type to Be Used in Various Clinical Situations | |
|---|---|
| **Clinical Situation** | **Bottles to Be Obtained** |
| Children <12 yr | Aerobic bottles only unless patient has peritonitis or fasciitis (in which case draw standard aerobic and anaerobic cultures) |

| Adults and children >12 yr | If anaerobic infection is unlikely, use aerobic bottles only unless the patient is immunocompromised |
|---|---|
| | In the immunocompromised, consider a bottle for fungal culture (usually effective in aerobic bottles, but consult the laboratory) |
| | If anaerobic infection is possible, each set consists of 1 aerobic and 1 anaerobic bottle |

can be due to true contamination, but they also may be caused by the intermittent bacteremia that occurs in normal, healthy people. In either case, distinguishing these from clinically significant bacteremia is based on both microbiologic information and correlation with the patient's clinical condition. The following features are often helpful. [122] [133] Notwithstanding these guidelines, it is prudent to contact patients with positive blood cultures, even when contamination is suspected on a microbiologic basis, to confirm improvement in their clinical condition.

Features of false-positive blood cultures are as follows:

1. Coagulase-negative staphylococci ( *S. epidermidis*) and *S. viridans* in a single bottle in patients not suspected of infectious endocarditis and without chronic indwelling IV access catheters are usually contaminants.
2. Corynebacteria (previously known as "diphtheroids"), *Propionibacterium acne*, and *Bacillus* spp. are usually contaminants, but they can be pathogenic in immunocompromised patients.
3. Multiple organisms in a series suggests contamination.
4. Late-appearing growth: species that grow out after prolonged culture have a higher likelihood of being contaminants.
5. The patient's symptoms have resolved or are inconsistent with sepsis (beware of infectious endocarditis, which can have an indolent course).
6. A primary infected source (e.g., sputum or urine) yields a different pathogenic isolate.

**Fungal Cultures**

Generally, fungi are difficult to isolate in blood cultures, and it may take 4 to 6 weeks to obtain a positive yield. If a fungemia is suspected, it is best to discuss culture media and technique with the laboratory before cultures are taken. Cultures of bone marrow are occasionally positive in deep mycoses when blood cultures are negative.

Occasionally, blood cultures are positive in cases of disseminated histoplasmosis or

candidiasis, but other fungi, such as *Cryptococcus* and *Aspergillus* or fungus-like bacteria such as *Nocardia* and *Actinomycosis*, are rarely isolated from the blood.

## DETECTION OF BLOOD IN THE STOOL

The fecal blood test makes use of the peroxidase-like activity of hemoglobin. The test card is impregnated with a compound that exhibits a blue color reaction when oxidized. The original test used guaiac, but current tests use more sensitive and more reliable dyes. The addition of hydrogen peroxide developer solution will oxidize the impregnated dye in the presence of a peroxidase (e.g., hemoglobin).

Testing for occult blood in the stool is associated with false-positive and false-negative results, but in its primary role in emergency medical practice the test is usually reliable in detecting significant acute gastrointestinal (GI) hemorrhage. [27] Low pH, heat, dry stools, reducing substances (e.g., ascorbate), and antacids can cause false-negative findings. Slow bleeding in the upper GI tract in which heme can be converted (denatured) to porphyrin during transit through the gut may not be identified by stool testing. False-positive results have been attributed to the ingestion of partly cooked or large quantities of meat (dietary sources of myoglobin and hemoglobin), and peroxidase-rich food. [141] Most vegetables contain peroxidase, including (in decreasing order) broccoli, turnips, cantaloupe, red radish, horseradish, cauliflower, parsnip, Jerusalem artichoke, bean sprouts, beans, lemon rind, mushrooms, parsley, and zucchini. [141] However, a simple in vivo study convincingly calls into question the possibility of peroxidase passing through the stomach without being denatured. [142] False-positive fecal occult blood tests are uncommon, and a positive test should be considered evidence of the presence of blood until proven otherwise. Routine iron supplementation should *not* be considered as a cause for a false-positive Hemoccult test,  although iron does (like bismuth preparations) cause the stools to appear black on gross examination. Despite this, early in vitro studies demonstrating an artifactual false-positive effect of iron are still frequently cited.

Normal GI blood loss is limited to <2.5 mL/day, which translates to <2 mg of hemoglobin per gram of stool (0.2% by weight). [145] The Hemoccult test is 37 or 95% sensitive to stool containing 2.5 or 20 mg of hemoglobin per gram of stool, respectively. [26] These figures reveal that low to moderate levels of blood may be missed, especially if breakdown of hemoglobin occurs during passage through the gastrointestinal tract or dilution occurs due to diarrheal illness. The test is much more likely to detect lower GI hemorrhage than an identical rate of bleeding from the upper GI tract due to the 100-fold diminution of peroxidase activity of blood during transition through the GI tract. [147]

### Method

The stool specimen is smeared onto the reagent area on the card and a drop of developer is added. A drop of water should be added to very dry specimens and allowed to moisten them prior to addition of developer, because the reaction needs to occur in an aqueous medium. Formation of a blue color on the paper anywhere around

or under the specimen within 60 seconds should be considered a positive result.

## TESTING FOR GASTRIC BLOOD

Heme tests designed for use on stool specimens can be unreliable when applied to gastric juices, with an increasingly high false-negative rate (low sensitivity) as pH decreases. [148] With these limitations in mind, the Gastroccult card was designed to test for blood in gastric juice. The modified guaiac developer contains buffers to neutralize gastric acid, thereby facilitating accurate hemoglobin detection. The test uses the same properties of hemoglobin as a peroxidase as does the fecal guaiac test. In product testing, the Gastroccult card was 100% sensitive in detecting specimens of 500 parts per million of blood by volume, equivalent to 0.05%, or 0.25 mL of blood in 500 mL of gastric contents (Smith Klein Diagnostics--package insert).

### Method

Apply a drop of gastric aspirate to the test area. Apply 2 drops of developer to the sample. Look for formation of a blue dye within 1 minute. Do *not* use fecal blood test developer. In a specimen that is already a bilious green, the test is only considered positive if new *blue* color is formed. The Gastroccult card also contains a pH testing strip located close to the occult blood testing area, which might be useful in testing emesis after an acid or alkali ingestion. False-positive results might be expected to occur (although studies to investigate this have not been performed) with meats and peroxidase-rich foods. False-negative reactions are likely in the presence of reducing substances, such as ascorbic acid. The accuracy of Gastroccult should not be affected by the presence of cimetidine or sucralfate.

## DIAGNOSTIC AND THERAPEUTIC TOXICOLOGIC BEDSIDE PROCEDURES

Patients who present to the ED with drug overdoses or poisoning can be quite difficult to manage if the clinician does not know the ingested drug or toxin. These patients often present with no available history, an inaccurate history, or a change in mental status that can obscure a significant part of the history. [150] Therefore, clinicians must rely heavily on physical examination findings and other sources of information to diagnose or confirm their clinical suspicions of poisoning or overdose. [151]

The hospital toxicology laboratory can be valuable in select cases. Limited screening tests for commonly ingested drugs are available, and ascertaining levels of specific drugs (e.g., acetaminophen, lithium, digoxin, phenytoin) can be helpful. However, most hospital laboratories are not equipped to perform timely analytic procedures for the thousands of possible drugs or toxins. In fact, the results obtained from use of limited drug screening have been shown to rarely influence medical management of patients in the ED.

Diagnostic bedside testing has the advantage of being cost-effective and timely. Selected tests provide immediate information to the clinician and can significantly

influence medical management in a timely manner. This section discusses bedside diagnostic and therapeutic toxicologic procedures.

## Noninvasive Diagnostic Procedures

### Amatoxin: Meixner Test

The ingestion of several types of mushrooms (e.g., *Amanita phalloides*) can be fatal. The most poisonous of these are the mushrooms containing amatoxins. Patients who have ingested these mushrooms often present 6 to 8 hours after ingestion with symptoms consisting of nausea, vomiting, diarrhea, and abdominal cramping. They often bring in specimens of the mushrooms chopped, crushed, cooked, or mixed with stool or gastric contents. Standard hospital laboratories cannot confirm or exclude the diagnosis of amatoxin poisoning; therefore, treatment decisions must be made on clinical grounds. [155]

Meixner reported a simple spot test for detecting amatoxins that can be used on gastric contents, stool, or actual mushroom samples. The basis of this test is the acid-catalyzed color reaction of amatoxins with lignin, a complex organic compound found in wood pulp. Cheaper grades of paper (e.g., newsprint) contain high amounts of lignin. Although there have been no extensive reports of in vivo studies, in vitro tests have shown this method to be highly sensitive and relatively specific for amatoxins.

The *procedure* for a qualitative detection of amatoxin consists of squeezing a drop of liquid from a fresh mushroom sample or squashing a piece of fresh mushroom onto a piece of newspaper. If a stool or a gastric sample is the only available specimen, the sample is mixed with reagent grade methanol (99.8%). The methanol will extract the amatoxin. If the samples are mixed with methanol, they should then be centrifuged and filtered. A drop of the liquid extract should then be placed on newspaper. Gently air dry all specimens at room temperature and avoid direct sunlight. Add 2 to 3 drops of concentrated hydrochloric acid (37%) to the dried specimen. Use an adjacent area for a control. High amounts of amatoxin in the dried samples exhibit a blue color in 1 to 2 minutes. Small amounts of amatoxin show a blue color in the sampled area in 10 to 20 minutes. This procedure has *not* been proven effective using other bodily secretions, such as blood or urine. [156]

### Mothball Identification

Presently, commercial mothballs are composed of either nontoxic paradichlorobenzene or possibly toxic naphthalene. Naphthalene can cause a significant hemolytic reaction in patients with glucose-6-phosphate dehydrogenase (G6PD) deficiency and in neonates. [157] In the past, mothballs have also been produced from camphor, which can cause central nervous system (CNS) depression and seizures in overdoses. Fortunately, these mothballs are not commercially available, although they may still exist in some households. A rapid differentiation between these groups of mothballs can expedite patient management and disposition. Several bedside tests have been reported to

facilitate this.

1. Paradichlorobenzene is heavier than naphthalene. In turn, naphthalene is heavier than camphor. [158] In lukewarm tap water, camphor will float and naphthalene and paradichlorobenzene will sink. In a solution of 3 tbsp of table salt thoroughly dissolved in 4 oz of lukewarm water, camphor and naphthalene will float and paradichlorobenzene will sink.
2. Paradichlorobenzene has a lower melting point than naphthalene. Paradichlorobenzene mothballs will melt in a water bath at 53 °C whereas naphthalene requires a water bath of >80 °C. [159]
3. Paradichlorobenzene is described as "wet and oily," whereas naphthalene is described as having a "dry" appearance. Paradichlorobenzene is familiar to many people as a cake of disinfectant used in urinals and diaper pails. [160]

**Body Secretion Analysis**

Careful analysis of bodily secretions, the odor emanating from poisoned patients, and the color of their urine can help identify certain toxins. Some characteristic smells and urine colors are noted in Tables 74-13 (Table Not Available) and 74-14 (Table Not Available) .

**Bedside Toxicologic Tests on Urine**

**Ethylene Glycol.**

Evaluation of the urine of patients who may have been exposed to ethylene glycol can be helpful. Urine should be tested for fluorescence under an ultraviolet light (caused by an additive to antifreeze) and presence of calcium oxalate crystals (a metabolic by-product of ethylene glycol metabolism).

Microscopic inspection of urine for *calcium oxalate crystals* may be helpful in the diagnosis of ethylene glycol exposure. The presence of either envelope-shaped calcium dihydrate crystals or needle-shaped calcium monohydrate indicates high oxalate levels in the serum (Fig. 74-3) (Figure Not Available) . Calcium monohydrate crystals can be easily confused with sodium urate crystals; therefore, the presence of the dihydrate crystal tends to be more specific for ethylene glycol ingestion. The lack of these crystals does not rule out a significant ethylene glycol ingestion, because the excretion of these may occur late in the ingestion (>6 hours) and occasionally does not occur at all.

Visual inspection of urine under a Wood's lamp or ultraviolet light to ascertain *fluorescence* may also be helpful in the diagnosis of ethylene glycol exposure. Antifreeze is the most common source of ingested ethylene glycol. Fluorescein, the actual fluorescing material, is often placed in commercially available antifreeze to enable mechanics to detect radiator leaks with a Wood's lamp or other ultraviolet light source. Fluorescein is a nontoxic inert vegetable dye that is eliminated unchanged in the urine. Therefore, high levels of fluorescein in urine suggest a significant ethylene glycol ingestion. However, a lack of fluorescein does not rule out a significant exposure,

because not all antifreezes contain fluorescein or high concentrations of fluorescein in relation to ethylene glycol. False-positive findings can occur if certain plastic urine containers are used. [168] To perform the test, take the urine sample in a glass test tube and a control urine sample into a dark room and inspect for fluorescence under a Wood's lamp.

**Salicylates.**

Several bedside tests have been developed to qualitatively detect salicylates in urine. These include the addition of 10% ferric chloride solution or Trinder solution to urine or testing with Phenistix reagent strips. All of these are rapid, inexpensive, sensitive tests that give a qualitative rather than a quantitative result. Therefore, a positive result requires a confirmatory quantitative serum salicylate assay.

The *ferric chloride test* is a commonly used rapid, qualitative, urinary screening procedure. To perform this test, several drops of 10% ferric chloride are added to 1 or 2 mL of urine that has been collected in a test tube. The immediate appearance of a bluish purple color signifies that salicylates are present in urine. Acetoacetic acid, acetone, and phenylpyruvic acid will test false-positive for salicylates. Thus this test may be falsely positive in patients with diabetic, alcoholic, or starvation ketoacidosis. This test is very sensitive, and as few as 2 aspirins taken within 24 hours will give a positive result. It will require 90 to 120 minutes from time of ingestion for this reaction to become positive in patients with normal renal function, so early test results may be misleading.

The *Trinder test* uses a mixture of mercuric chloride and ferric nitrate in deionized water. To perform this test, 1 mL of urine is mixed with 1 mL of Trinder solution. A violet or purple color signifies the presence of salicylates. Acetoacetic acid and high levels of phenothiazines may give false-positive results.

*Phenistix reagent strips* were originally developed to detect phenylketonuria. However, Phenistix strips also turn brown in presence of salicylates. False-positive findings for salicylates can occur if phenothiazines are present.

**Bedside Toxicologic Tests on Blood**

**Methemoglobinemia.**

Patients with methemoglobinemia will often have a normal partial pressure of oxygen ($pO_2$ ) on routine arterial blood gas analysis, a normal *calculated* hemoglobin saturation, a nondiagnostic pulse oximeter reading, and cyanosis that does not clear with $O_2$ administration. Bedside visual inspection of venous or arterial blood may be helpful in the diagnosis of methemoglobinemia. Methemoglobinemia occurs when normal hemoglobin is exposed to an oxidant stress ($Fe^{2+}$ converted to $Fe^{3+}$ ). If the erythrocytes are not able to handle such stress, such as in the presence of G6PD deficiency, hemoglobin remains in a oxidized state (methemoglobin), causing a color change in the molecule. Methemoglobin levels >15% are reported to cause a cyanotic

appearance in a patient. [172]

The procedure for evaluating for methemoglobinemia is to place a drop of sample blood on a white background (a white coffee filter is appropriate) in a well-lit environment. Next to this, place a drop of normal blood as a comparison control sample. Blood with methemoglobinemia appears "darker" or "chocolate-brown." [173]

This method relies on the ability of the examiner to distinguish color changes and therefore may have a degree of interobserver variance. Methemoglobin levels of <10% may only slightly alter the color of blood and thereby cause a false-negative finding. Methemoglobin levels of between 12 and 14% may cause a false-negative reading 50% of the time. However, at methemoglobin levels of 35%, the identification of methemoglobinemia by visual inspection is quite accurate. [173] At this level, most patients are obviously cyanotic and quite symptomatic.

## Invasive Diagnostic Procedures

Several invasive diagnostic bedside procedures can be useful in the assessment of possible drug overdoses. The basic premise of these procedures is that patients who have been exposed to a certain drug or poison will respond in a particular fashion if given a diagnostic challenge dose of another particular drug or true antidote.

### Naloxone

Naloxone hydrochloride (Narcan) is an opioid receptor antagonist that has the ability to reverse the effects of chemical agents affecting all opioid receptor sites, particularly respiratory and CNS depression. Because of this, a trial of naloxone has been recommended for all patients with CNS depression. [174] Certain clinical findings such as miosis, decreased respiratory rate, and evidence of illicit drug use can predict many patients who will respond to a diagnostic challenge dose of naloxone. [175]

If a patient's mental status improves significantly after a dose of naloxone, the patient should be considered to have been exposed to an opioid substance. This is true even if a laboratory drug screen is negative for opioids. This is due to the fact that many of the synthetic opioid agents, such as fentanyl, propoxyphene, meperidine, methadone, and pentazocine may not be detected by the routinely used immunoassay drug screen. [176] Although cases have been reported of patients with other non-opioid overdoses (such as alcohol or phencyclidine) responding to naloxone, those single observations have not been confirmed in controlled animal or human studies.

The traditional challenge dose of naloxone in an adult or child is 2 mg every 2 minutes IV until a response is achieved or 10 mg is given. [176] Some clinicians prefer to use much smaller doses (0.1 to 0.2 mg) and titrate to effect. This may partially reverse opioid overdose-related symptoms and confirm the diagnosis without precipitating the opioid withdrawal syndrome seen in patients with opioid dependency. Most patients with an opioid overdose will exhibit some response to 1 to 4 mg of naloxone, but some massive overdoses may require larger amounts. A patient who does not respond at all

to 10 mg of naloxone probably does not have a pure opioid overdose. The high doses of naloxone presently recommended are needed to reverse many synthetic narcotic agents, such as propoxyphene and methadone. Lower doses can be given (0.4 to 0.8 mg in adults or 0.01 mg/kg in children) to reverse known opioid-induced respiratory depression without reversing analgesia. [177] Because naloxone has a half-life between 30 and 60 minutes, a continuous drip of naloxone can be used to avoid resedation. A reasonable choice is to use two thirds of the initial bolus dose that achieved the desired reversal effect as the hourly IV dose. For example, a patient who satisfactorily responded to 1.5 mg of naloxone might receive a naloxone solution of 10 mg of naloxone in 500 mL of normal saline at a rate of 1 mg (50 mL)/hour IV. [178] Nalmefene, a new long-acting opioid receptor antagonist that has a terminal half-life of roughly 11 hours, can also be given to patients with suspected overdoses. Theoretically, a single dose of nalmefene will be effective longer than the effects of heroin or most abused opiate substances. The initial recommended dose is 1.0 to 1.5 mg IV.

Naloxone and nalmefene have minimal significant side effects, other than precipitating withdrawal from patients addicted to opioids. Unlike alcohol withdrawal, naloxone-induced opioid withdrawal in the adult is short-lived and is usually not life-threatening. Withdrawal can be avoided if lower initial doses of naloxone or nalmefene are given and then are slowly titrated upward to the desired effect. [177]

**Flumazenil**

Flumazenil is a competitive benzodiazepine receptor antagonist that has the ability to reverse the CNS and respiratory depression caused by all currently commercially available benzodiazepines. The use of flumazenil as a *routine diagnostic bedside challenge in all obtunded patients is discouraged,* and its use in the setting of possible benzodiazepine overdose is controversial. Unlike naloxone, flumazenil can have significant side effects in certain subsets of patients. [179] These include precipitating seizures or a withdrawal syndrome in benzodiazepine-dependent patients. To minimize the chance of seizures, flumazenil should be avoided in patients who may have ingested epileptogenic drugs (e.g., cyclic antidepressants, cocaine, theophylline, lithium, carbamazepine, or isoniazid). [180]

In overdose settings in which there is obtundation and respiratory depression and no history of seizures or suspicion of involvement of epileptogenic agents, flumazenil can be administered IV at a dose of 0.2 to O.5 mg/min. Most benzodiazepine-overdosed patients show mental status improvement with 1 mg of flumazenil and almost all respond to 3 to 5 mg. It is prudent to use small, escalating doses given very slowly (maximally, 0.5 mg/min). Larger doses can be given at one time as a bolus, although this increases such side effects as anxiety, agitation, and emotional lability; it also increases the chances of precipitating withdrawal in benzodiazepine-dependent patients. [181] Fortunately, seizures that occur after flumazenil use are usually transient and can usually be controlled with additional benzodiazepines. In rare cases, higher doses of benzodiazepines, barbiturates, and phenytoin may be required. [180]

If a patient responds to flumazenil with an improvement in depressed mental status, this only suggests that the patient is under the influence of a benzodiazepine. Flumazenil

can partially reverse the effects of many other agents or conditions that affect the gamma-aminobutyric acid (GABA) pathway, such as zolpidem and hepatic encephalopathy, [182] but it does not have any significant effect on alcohol, barbiturates, and other non-benzodiazepine sedative-hypnotics. [183]

**Physostigmine**

Physostigmine is an acetylcholinesterase inhibitor that can penetrate into the CNS and thus can reverse both the central and peripheral effects of anticholinergic agents. The use of physostigmine as a diagnostic challenge can be helpful in select situations. In the majority of patients with anticholinergic toxicity, no laboratory tests are available to rapidly confirm the diagnosis, and testing for specific drugs is limited. A clinical picture that may consist of mydriasis, dry and flushed skin, dry mucous membranes, urinary incontinence, absent bowel sounds, tachycardia, hyperthermia, hallucinations, agitation, and seizures, suggests an anticholinergic toxicologic syndrome. A rapid and dramatic response to physostigmine often confirms a diagnosis of anticholinergic toxicity. In these patients, physostigmine reduces much of the CNS toxicity of the agents and decreases the degree of agitation and confusion. [184] [185]

As a diagnostic challenge or therapeutic intervention, physostigmine can be administered IV under constant cardiac monitoring at a dose of 1 to 2 mg in adults and 0.02 mg/kg in children, over 5 minutes. Some clinicians empirically pretreat with a benzodiazepine to militate against seizures, but this practice has not been proven effective or necessary. Because the half-life of physostigmine is 30 to 60 minutes, a repeat dose of 2 mg also can be given as clinically indicated. Continuous infusions or multiple repeat doses are strongly discouraged.

Similar to flumazenil, physostigmine has been reported to interact detrimentally with cyclic antidepressants, often causing life-threatening dysrhythmias. Physostigmine also can cause an excess of acetylcholine and a resultant cholinergic crisis. This syndrome includes salivation, lacrimation, urination, defecation, bradycardia, bronchorrhea, and seizures. [186] Dysrhythmias, including asystole, have also been reported. [187] For this reason, 1 mg of atropine, IV, should be readily available to reverse a potential cholinergic excess when using physostigmine.

**Deferoxamine**

Deferoxamine is an organic compound derived from the bacterium *Streptomyces pilosus*. Deferoxamine can chelate iron and can be used as therapy or as a diagnostic challenge in patients with iron overdoses. Patients who have unstable vital signs or significant GI or CNS symptoms usually require therapeutic doses of deferoxamine. *Asymptomatic* patients with a history of iron overdose usually require supportive care only. Patients with persistent but mild GI symptoms, such as vomiting and diarrhea, may be given a diagnostic challenge dose of deferoxamine. The use of iron levels and total iron binding capacity to dictate ED management can be inaccurate, misleading, and time-consuming.

A diagnostic challenge dose of deferoxamine is administered IM or IV over 45 minutes

at doses of 40 to 90 mg/kg up to a maximum of 1 g in children and 2 g in adults. Deferoxamine can also be administered IV as a constant drip of 15 mg/kg/hour. [191] A positive result occurs when chelated iron in the form of ferrioxamine appears in the urine. This usually causes the urine to turn a reddish orange or "vin rose" color in 2 to 3 hours after initiation of treatment. The color change is qualitative only and has no prognostic significance. Color change caused by ferrioxamine is pH and concentration dependent, and false-negative test results occur. [192]

Chronically administered deferoxamine has been reported to have multiple adverse effects, such as adult respiratory distress syndrome (ARDS), visual defects, and enhancement of *Yersinia enterocolitica* infections. [183] In the setting of the single challenge dose, flushing, erythema, tachycardia, urticaria, and hypotension caused by rapid administration of deferoxamine are the most serious side effects. [193]

**Invasive Therapeutic Procedures**

The indications and rationale for use of certain therapeutic procedures in toxicology are often misunderstood.

| **TABLE 74-15** -- Drugs That Have Increased Elimination with Urinary Alkalinization |
|---|
| Chlorpropamide |
| 2,4-Dichlorophenoxyacetic acid |
| Formate |
| Methotrexate |
| Phenobarbital |
| Salicylate |

**Alkalinization of Urine and Blood**

Alkalinization of urine consists of manipulating the pH of urine to enhance excretion of certain drugs (Table 74-15) . Weak acids remain in ionic form in a basic milieu. The ionic form often prevents reabsorption of that drug in the proximal tubule, and urinary alkalinization can therefore promote elimination in the urine. For certain drugs, this can play a significant role in their elimination. For example, salicylate elimination increases proportionately to the urinary flow rate, but it increases *exponentially* with increases in the urinary pH.

Recommendations differ on the actual method or formula to achieve urinary alkalinization. No body of literature exists that supports one method of urinary alkalinization over another. [197] In general, this procedure should be titrated to the patient's fluid and acid-base status to achieve a urinary pH of 7.5 to 8.0. Many authors recommend the use of a constant infusion of a relatively isotonic solution consisting of 3 ampules of sodium bicarbonate (44 mmol/ampule) added to 1 L of 5% dextrose in water ($D_5$ W). Another reasonable formula is to begin with a bolus of 2 ampules of IV sodium bicarbonate, or 1 to 2 mmol/kg of body weight. The bolus is followed with a constant infusion of 3 ampules of sodium bicarbonate in 1 L of $D_5$ W solution with 20 to 40 mmol of potassium (if the patient has normal renal function) infused at 100 to 300 mL/hour. Although repetitive boluses of sodium bicarbonate ampules also can be used, this may increase the chances of hypernatremia, hypokalemia, relative hypocalcemia, fluid overload, and alkalemia. All of these are potential adverse effects of aggressive urinary alkalinization. The actual amount of fluids and bicarbonate administered requires titration to the patient's clinical condition. Therefore careful monitoring of electrolyte, pH, and fluid status is encouraged. [198] [199] [200]

Urinary alkalinization can sometimes be difficult to achieve or maintain. Hypovolemia is probably the leading cause of an inability to achieve an alkaline urine. Other theoretical causes are hypokalemia and hypochloremia. Several authors have suggested that in patients with severe salicylate poisoning, urinary alkalinization may be difficult if not impossible to achieve with reasonable doses of bicarbonate.

## Ethanol Infusion

Ethanol can be used as a therapeutic intervention in patients with methanol or ethylene glycol poisoning due to ethanol's much greater affinity for alcohol dehydrogenases. These enzymes metabolize methanol and ethylene glycol to even more toxic by-products. However, with serum ethanol levels of

**TABLE 74-16** -- Ethanol in Methanol or Ethylene Glycol Poisoning [N]

**Intravenous Ethanol: Loading Dose (using a 10% ethanol solution)** [1]
**(A 10% volume/volume concentration yields approximately 100 mg/mL)**

| | Volume of Loading Dose (given over 1-2 hr as tolerated) [1] | | | | | |
|---|---|---|---|---|---|---|
| | 10 kg | 15 kg | 30 kg | 50 kg | 70 kg | 100 kg |
| Loading dose of 1000 mg/kg of 10% ethanol (infused over 1-2 hours as tolerated); *assumes a zero ethanol level to start* | 100 mL | 150 mL | 300 mL | 500 mL | 700 mL | 1000 mL |
| Aim is to produce a serum ethanol level of 100-150 mg/dL | | | | | | |

**Oral Ethanol: Loading Dose**
**(A 20% volume/volume concentration yields approximately 200 mg/mL)**

| | Volume of Loading Dose | | | | | |
|---|---|---|---|---|---|---|
| | 10 kg | 15 kg | 30 kg | 50 kg | 70 kg | 100 kg |
| Loading dose of 1000 mg/kg of 20% ethanol, [b] diluted in juice; may be administered orally or via nasogastric tube; *assumes a zero ethanol level to start* | 50 mL | 75 mL | 150 mL | 250 mL | 350 mL | 500 mL |

| Aim is to produce a serum ethanol level of 100-150 mg/dL | | | | | | |

**Intravenous Ethanol: Maintenance Dose (using a 10% ethanol solution)** ı
**(A 10% volume/volume concentration yields approximately 100 mg/mL. Infusion to be started immediately following the loading dose.**
**Aim is to maintain serum ethanol level of 100-150 mg/dL )**

| Normal Maintenance Range | Infusion Rate (mL/hr for various weights) ı | | | | | |
| --- | --- | --- | --- | --- | --- | --- |
| | 10 kg | 15 kg | 30 kg | 50 kg | 70 kg | 100 kg |
| 80 mg/kg/hr | 8 | 12 | 24 | 40 | 56 | 80 |
| 110 mg/kg/hr | 11 | 16 | 33 | 55 | 77 | 110 |
| 130 mg/kg/hr | 13 | 19 | 39 | 65 | 91 | 130 |
| Approximate maintenance dose for chronic alcoholic | | | | | | |
| 150 mg/kg/hr | 15 | 22 | 45 | 75 | 105 | 150 |
| Range required during hemodialysis | | | | | | |

| | 25 | 38 | 75 | 125 | 175 | 250 |
|---|---|---|---|---|---|---|
| 250 mg/kg/hr | 25 | 38 | 75 | 125 | 175 | 250 |
| 300 mg/kg/hr | 30 | 45 | 90 | 150 | 210 | 300 |
| 350 mg/kg/hr | 35 | 53 | 105 | 175 | 245 | 350 |

**Oral Ethanol: Maintenance Dose**
**(A 20% volume/volume concentration yields approximately 200 mg/mL; infusion to be given each hour immediately following a loading dose; aim is to maintain serum ethanol level of 100-150 mg/dL; each dose may be diluted in juice and given orally or via nasogastric tube)**

| Normal Maintenance Range | Infusion Rate (mL/hr [§] for various weights [¶]) | | | | | |
|---|---|---|---|---|---|---|
| | 10 kg | 15 kg | 30 kg | 50 kg | 70 kg | 100 kg |
| 80 mg/kg/hr | 4 | 6 | 12 | 20 | 28 | 40 |
| 110 mg/kg/hr | 6 | 8 | 17 | 27 | 39 | 55 |
| 130 mg/kg/hr | 7 | 10 | 20 | 33 | 46 | 66 |
| Approximate range for chronic alcoholic or for patient receiving continuous oral activated charcoal | | | | | | |

| | | | | | | |
|---|---|---|---|---|---|---|
| 150 mg/kg/hr | 8 | 11 | 22 | 38 | 53 | 75 |
| Range required during hemodialysis | | | | | | |
| 250 mg/kg/hr | 13 | 19 | 38 | 63 | 88 | 125 |
| 300 mg/kg/hr | 15 | 23 | 46 | 75 | 105 | 150 |
| 350 mg/kg/hr | 18 | 26 | 52 | 88 | 123 | 175 |

100 mg/dL, minimal amounts of ethylene glycol or methanol are metabolized by alcohol dehydrogenases. [201] [202] Ethanol infusions are not useful in the treatment of isopropyl alcohol poisoning.

Ethanol can be administered orally or IV (Table 74-16) . Intravenous ethanol has the advantages of obtaining therapeutic levels rapidly, ensuring complete absorption, limiting chances of aspiration, and avoiding gastritis. A 5% concentration of ethanol, which can be given in a peripheral vein, requires the use of large fluid volumes. In a 70-kg patient, a loading dose requires 1.4 L of 5% solution, with a maintenance dose of 700 mL/hour. In contrast, oral loading can be achieved using much lower volumes. However, oral loading can be difficult in the uncooperative or unconscious patient or if vomiting or GI hemorrhage is present. A therapeutic level is reached more slowly with oral loading.

Ethanol metabolism can vary widely, and ethanol is dialyzable. Therefore, it may be difficult to maintain appropriate ethanol levels during dialysis therapy of ethylene glycol or methanol. Frequent measurements of ethanol should be obtained and the infusion adjusted accordingly. [203] When patients are given ethanol infusions, CNS depression and hypoglycemia are common adverse effects (the latter is particularly true in diabetics and children). [204] Serial levels of ethanol and glucose should be obtained. If IV ethanol is given, careful attention to fluid status should be maintained.

# Chapter 75 - Universal Precautions

**Kevin Rodgers**

The concept of "universal precautions" implies that health care workers consider all blood and body fluids (B/BF) as potentially infectious and take appropriate precautions to protect themselves and, as a consequence, their patients. This concept is especially relevant to emergency medicine, because emergency patients may present with acute and chronic infectious diseases, and clinical procedures performed in the emergency department (ED) often involve exposure to B/BF. Infectious pathogens of particular concern to emergency physicians include human immunodeficiency virus (HIV), hepatitis B and C viruses (HBV and HCV), *Mycobacterium tuberculosis,* cytomegalovirus, *Clostridium tetani* (tetanus), herpes simplex virus, and *Plasmodium* (malaria). Each year, health care workers suffer considerable morbidity, mortality, and health care costs from occupationally acquired infections.

In 1985, the Centers for Disease Control and Prevention (CDC) first recommended guidelines for the protection of health care workers from B/BF-borne diseases. [1] They asked that hospitals adopt an infection control policy embracing universal precautions. The guidelines delineated methods for minimizing mucous membrane, cutaneous, and percutaneous exposure to B/BF from all patients, especially during the performance of invasive procedures. These guidelines were meant to supplement standard infection control procedures such as hand washing. Although the initial recommendations dealt with the emerging problem of HIV, subsequent updates have recommended universal precautions for protection against transmission of hepatitis and other B/BF-borne pathogens.  In 1991, these recommendations became law under a mandate from the Occupational Safety and Health Administration (OSHA). [5]

In general, the OSHA requirements involve hand washing and barrier protection (gloves, eye protection, gowns, shoe covers, patient barriers) to prevent exposure to B/BF. Although these precautions prevent the mucocutaneous exposures responsible for transmission of most B/BF-borne diseases, including hepatitis, they do little to prevent the typical percutaneous exposure from sharps and needlesticks associated with HIV transmission. Indeed, most of the documented occupational HIV transmissions reported by the CDC have been related to percutaneous exposures. In a study of 1201 health care workers exposed to B/BF of HIV-infected patients, needlestick injuries accounted for 80% of the exposures and sharp instruments accounted for 8%. [6] With this in mind, the original idea of universal precautions has been modified to include methods for prevention of needlestick and sharps injuries. These efforts are aimed at educating health care workers in the proper handling of sharps, the avoidance of needle recapping, provision of easily accessible disposal containers, and the re-engineering of needles and sharps to decrease the likelihood of percutaneous exposure.

The OSHA mandate requires that health care facilities provide their employees with the education and equipment necessary to prevent occupational exposures. Additionally, because health care workers have a 3- to 6-fold greater seropositivity for HBV as compared with the general population and they contract approximately 12,000 new

cases of HBV per year, OSHA also required employers to offer their employees the HBV vaccine. [5] [7] In complying with the OSHA requirements and the CDC recommendations, each health care facility must develop a formal, comprehensive plan for preventing exposures to occupational health hazards. Gerberding has outlined a 3-pronged approach for preventing exposures. [8] The first step is use of equipment with inherent safety features. Second, standardized protocols and techniques designed to maximize safety during work should be used. Personal protective gear is used when hazards cannot be successfully avoided through equipment and work practice controls. Unfortunately, because of the frequent exposure to bloody procedures on patients with unknown health status in the ED, some degree of personal protective equipment is always required. Third, administrative controls, which include personnel assignment, training requirements, and enforcement procedures, are used in implementing safety interventions.

Unfortunately, numerous studies reveal that compliance with universal precautions is far from universal. Kelen and colleagues found only a 44% compliance rate with a comprehensive barrier protection approach by health care workers caring for critically ill and injured ED patients. [9] More disturbing was the fact that compliance was worse during interventions with the highest risk of potential exposure. At another center, ED compliance with various forms of barrier protection as dictated by protocol ranged from negligible to incomplete: gloves were worn with a frequency of 75%; gowns, 25%; eyewear, 19%; and masks, 2%. [10] Stated reasons for noncompliance included lack of time, perception that patients were low-risk exposures, interference with technical skills, lack of necessary supplies, and finally the fact that the protective equipment was uncomfortable. It is clear that mandatory, continuing education centering on the risks of B/BF exposure and the prevention thereof, in conjunction with re-engineering, is critical for the reduction of occupational exposures.

## OCCUPATIONAL DISEASE EXPOSURE

Health care workers are at risk for occupational exposure through contact with B/BF and aerosols for a myriad of diseases, especially HIV infection, hepatitis, and tuberculosis. Factors that affect the risk of exposure include inoculum concentration, quantity of inoculum, duration of exposure, portal of entry, loss of infectivity during transfer, and the immunologic status of both the source patient and the exposed health care worker. Emergency care workers are at increased risk because of the sheer number of exposures and the inability to predict high-risk patients even with rigorous risk factor assessment. [12] [13] Indeed, in a study of 2523 patients presenting to an ED in Baltimore, 24% were infected with either HIV, hepatitis B, or hepatitis C.

Another concern for emergency care workers is the inability to predict which bodily fluids contain blood. The CDC recommends universal precautions for exposure to blood, semen, vaginal secretions, and serous (cerebrospinal, synovial, pleural, peritoneal, pericardial, amniotic) fluids. Previously, feces, nasal secretions, sweat, tears, urine, and vomitus were not included as high-risk exposures unless they contained "visible" blood. However, the CDC subsequently issued specific recommendations for the use of universal precautions during all uncontrolled emergency circumstances because of the inability to predict which fluids will be tainted with blood. [15] Because of their significant

morbidity and mortality and frequency of exposure in the ED, factors associated with the transmission of HIV, hepatitis B and C, and tuberculosis will be reviewed.

## Hepatitis B

Although most attention is focused on HIV with regard to B/BF-borne pathogens, because of its inherent mortality, the risk of developing hepatitis B after a B/BF exposure is significantly greater. Percutaneous exposure to HBV carries a much greater risk of contagion than does similar exposure to HIV (i.e., 10 [8] vs 10 [3] free viral particles/mL). [16] The risk of infection with HBV in a nonimmune person after percutaneous exposure to an HBV surface antigen-positive patient ranges from 6 to 30%. This results in approximately 12,000 cases of HBV in health care workers each year and 200 to 250 deaths. Seroprevalence of HBV antibodies in emergency care workers ranges from 12 to 30%, reflecting a significant exposure rate. Additionally, HIV-positive patients are 4 times more likely to be HBV positive than HIV-negative patients. Although the epidemic nature of HBV among health care workers has declined since the introduction of the HBV vaccine, an estimated 23 to 58% of health care workers remain unvaccinated.

## Hepatitis C

Hepatitis C, formally known as non-A, non-B hepatitis, is no longer simply a disease associated with transfusions. Occupational exposure is firmly established, with a 3 to 4% seroconversion risk after percutaneous exposure to blood positive for the HCV antibody. [19] Several studies have documented an 18% seroprevalence rate among ED patients. The lower transmission rates for HCV as compared with HBV are due to a smaller concentration of viral particles. However, this disease is increasing in prevalence; and with a 10% risk of developing chronic active hepatitis, it cannot be ignored.

## HIV Infection

Through July of 1993, a total of 115 U.S. health care workers had been reported to the CDC for occupational transmission of HIV. [20] Of these, 37 had documented seroconversion associated with documented occupational exposure. [21] Included in this group were 34 percutaneous exposures, 2 mucous membrane exposures, and 2 exposures associated with contact to an open wound or nonintact skin. The combination of statistics from multiple studies has predicted the risk of infection from a single percutaneous exposure to be 0.3% and 0.05% for a single mucous membrane exposure. Although the risk is certainly not zero, Fahey and associates report almost 8000 cutaneous exposures to blood presumably infected with HIV without evidence of HIV transmission. [22]

Thus far, casual contact has not been associated with the transmission of HIV. In one case-control study by the CDC, the risk for developing postexposure HIV infection was associated with *deep injury to the health care worker, visible blood on the injuring device, a device placed in the source patient's artery or vein, and a source patient who died of AIDS within 60 days of the exposure, consistent with a high HIV titer*. [23] Clearly,

there is an occupational risk for transmission of HIV infection; however, the magnitude is small when compared with the risk for transmission of other B/BF-borne diseases.

However, the cumulative risk of HIV conversion due to transmission from patient to physician may not be trivial. Using several theoretical assumptions about emergency physician exposure to HIV-infected patients, Wears and coworkers estimated the *cumulative career risk* of occupational HIV conversion, depending on the use or nonuse of universal precautions, to be from 0.08 to 0.1% in low-risk EDs (i.e., patient seroprevalence of 1%) and from 1.1 to 1.4% in high-risk EDs (i.e., patient seroprevalence 7%). [24]

**Tuberculosis**

Tuberculosis is included in this discussion because of its resurgence especially in HIV-positive and other immunocompromised patients. These patients commonly require acute respiratory procedures in the ED that expose emergency care workers to aerosolized contamination. Risk factors for tuberculosis transmission include a large number of contacts, duration of exposure, poor air circulation, cough-inducing procedures, degree of infectivity, ineffective therapy, and a delay in disease diagnosis. An additional risk, with regard to universal precautions, is the fact that health care workers were least compliant with the wearing of masks. Thus, a low index of suspicion must be exercised in identifying potential tuberculosis patients. They must be quarantined in a room possessing a high rate of air exchange with non-recirculated air. Also, high efficiency particulate air (HEPA) masks capable of screening droplet nuclei 1 to 5 mum in diameter should be worn by health care workers and a surgical mask should be worn by the patient.

## UNIVERSAL PRECAUTIONS

The goal of universal precautions is the prevention of all occupational exposures to B/BF-borne diseases. In the ED where bloody procedures are commonplace and the health status of the patients is unpredictable, appropriate precautions must be viewed as a way of life. It is clear that noncompliance with the guidelines set forth by the CDC and OSHA is actually the greatest risk factor associated with occupational exposure. The guidelines listed below are recommended when performing invasive procedures, or when handling blood, body fluids, or contaminated equipment:

1. Wash hands before and after patient contact. Hands and skin surfaces should be washed immediately and thoroughly if contaminated by blood or body fluids.
2. Use barrier precautions to limit skin and mucous membrane exposure. Gloves should be worn whenever the potential exists for any contact with or handling of B/BF. Gloves should be changed after each patient contact and removed carefully to avoid skin contamination. The added protection gained from wearing double gloves is controversial. Latex gloves seem to be less porous to B/BF than vinyl gloves. [25] However, quality of production, [26] intensity and duration of use, [27] and chemical exposure (including alcohol) [28] are major determinants of leakage. Furthermore, latex allergy affects a sizable minority of health care workers [29] ; hypoallergenic latex and nonlatex gloves may be required. Masks and protective

eyewear should be worn during procedures that generate B/BF droplets (e.g., wound irrigation) whereas gowns and shoe covers should be worn during situations likely to generate exposures to large volumes or splashes of B/BF (e.g, during resuscitation of penetrating thoracic trauma).

3. Use precautions with sharps (needles, scalpels, and other sharp instruments). Ideally, needles should not be recapped, bent, broken, or removed from disposable syringes. Immediately after use, they should be placed in a puncture-resistant container located at the point of use. Whenever possible, re-engineered needles and sharps designed to prevent percutaneous exposures should be used.

4. Use mechanical ventilation devices. Mouthpieces, resuscitation bags, or ventilation devices should be strategically located in areas where resuscitation is predictable, to avoid mouth-to-mouth resuscitation.

5. Use special caution to avoid cutaneous B/BF contact if the health care worker has exudative lesions or weeping dermatitis. When possible, refrain from direct patient contact in these circumstances.

6. Wear a snugly fitting HEPA mask capable of filtering 1- to 5-mum particles when coming in contact with patients who potentially have tuberculosis or immunocompromised patients receiving aerosolized treatments. If possible, the patient should be treated in a high-circulation, negative-pressure room with external exhaust.

7. All health care workers should receive mandatory annual education about infection control and safe work place practices.

## SPECIFIC CLINICAL PROCEDURES AND EXPOSURE RISK

With regard to guidelines for specific procedures, little is known about potential B/BF contact associated with specific procedures performed in the ED. Kelen and coworkers studied 2529 procedures in 1025 patients and found that all 18 procedures examined except IM injections resulted in B/BF contact with the hands. [31] Thus, virtually all procedures performed in the ED warrant glove use.

The potential for health care worker-transmitted cutaneous infectious exposure of immunocompromised patients in the ED also exists. Hand washing or the use of nonsterile examination gloves between patient evaluations appears to represent useful means to reduce the risk of patient exposure and an important rationale for general examination glove use during even minor patient examinations and procedures. [25] [32]

In the study by Kelen and coworkers, chest tube placement, lumbar puncture, and examination of the bleeding patient (from any source including the gastrointestinal tract and during wound care) resulted in B/BF contact with the facial area, suggesting that a mask and eye protection be worn. [31] The study also revealed B/BF contact with the body for many procedures not commonly considered to be at risk for exposure to that area. This study suggests gown protection for all but the simplest procedures. Shoe covers should be worn for all situations associated with large volumes or splashes of blood.

Procedures involving sharps (needles, scalpels, sharp instruments) are particularly

problematic and the most common cause of high-risk exposure. Percutaneous injury may be reduced by modifying specific clinical practices. For example, the use of fingers rather than an instrument to hold tissue being sutured has been associated with health care worker punctures in the operating room setting. [33] The practice of recapping needles also is a common mechanism for health care worker punctures (Fig. 75-1 *A*). Although it is preferred to dispose of syringe needles (and other sharps) immediately after their use, this practice is not always possible in the middle of the procedure. Placement of the sharp in a protected area of the sterile field or Mayo instrument stand is commonly practiced, but this practice may be hazardous when several operators are performing the procedure. Close communication between operators regarding sharps placement is encouraged.

If a needle must be recapped, the protective cap should be placed on a flat surface free of the operator's hand while the operator guides the needle into the cap (Fig. 75-1 *B*). That is, the cap is *not* held by the operator during *placement of the needle into the cap*. Only after the needle is fully enclosed in the cap should the operator secure the cap to the syringe. It is advised that even this final step be performed on the flat surface to enhance control of the needle-syringe-cap unit. It is critical that the operator *personally take responsibility to dispose of all sharps promptly* in puncture-proof containers after a procedure--thus avoiding exposure of other health care workers to these items. [34]

Equipment involved in direct patient care should also receive special attention in accordance with universal precautions. Reusable equipment and devices that enter a patient's bloodstream or body cavities should be washed thoroughly and sterilized between patient uses. Devices that touch intact mucous membranes but do not penetrate the body should be washed thoroughly and undergo high-level disinfection or sterilization if possible. Devices that only touch intact skin should be cleaned with a hospital-grade disinfectant.

## POSTEXPOSURE MANAGEMENT

Unfortunately, universal precautions and our compliance with them are not perfect. When occupational exposures to B/BF do occur, each health care facility must have a protocol that clearly defines the evaluation and management of the health care worker at risk. Because it is well documented that exposures are underreported, the first step in a successful occupational exposure program is encouraging health care workers to report any and all exposures.

Immediately after any exposure, the area should be thoroughly cleaned and clinically evaluated. The HIV, HBV, HCV, and other B/BF-borne disease status of the source patient and the exposed health care worker should be established. If there is no record of a recent negative serologic test for the source patient and health care worker, a hepatitis panel (HBV, HCV) should be done and permission for HIV testing obtained. If the source patient is unknown or refuses to be tested, the health care worker should be treated as though the exposure occurred unless there is strong clinical and epidemiologic evidence to the contrary. The health care worker should be counseled on postexposure prophylaxis, and follow-up testing should be done at 6 weeks, 3 months, and 6 months. During this follow-up period, the CDC recommends that the exposed

person defer pregnancy; refrain from breast feeding and donation of blood, semen, and tissue; and practice safe sex or abstinence.

## HIV Exposure

For known HIV exposure, the role of chemoprophylaxis should be discussed with the health care worker. Current information related to the transmission of HIV and use of zidovudine (ZDV; formerly known as azidothymidine or AZT) and other retroviral agents should be reviewed.

Recommendations for prophylactic use of ZDV are based on the case-controlled study by the CDC, which showed a 79% reduction in the risk for HIV infection among health care workers who used ZDV. [35] If the worker decides to take postexposure prophylaxis, it should begin preferably within 1 to 2 hours of exposure and no later than 24 hours post exposure. The dose of ZDV is 200 mg 3 to 5 times a day for 4 weeks. Lower doses are generally well tolerated whereas higher doses are associated with gastrointestinal symptoms, fatigue, and headache. [36]

This is an area of rapid treatment protocol evolution; multiple antiretroviral therapy may become the norm with high-risk exposures. The emergency physician is encouraged to work closely with his/her infectious disease consultant(s) to maintain a current therapeutic protocol. Other agents (Table 75-1) that may be added to prophylaxis with ZDV include lamivudine [3TC] (150 mg, orally, twice daily), indinavir [IDV] (800 mg, orally, 3 times a day), and saquinavir (600 mg, orally, 3 times a day). [23]

## Hepatitis Exposure

The treatment of hepatitis B exposure depends on the immunization status of the health care worker. Fully immunized (adequate antibody response) health care workers need no treatment. Nonimmunized workers need hepatitis B immune globulin (HBIG) in a dose of 0.06 mL/kg IM. Their initial dose of hepatitis B vaccine (1 mL IM) is given at a separate site. The vaccine is repeated at 1 month and 6 months. Health care workers with an inadequate response to the HBV vaccine (i.e., low antibody level to hepatitis B surface antigen; generally <10 mIU/mL) require a booster dose. Factors associated with an inadequate immunologic response include increasing age, smoking, and obesity. [37] [38] Those who have received a prior vaccination series with good antibody response should have their hepatitis B titer checked and further therapy guided by the current level.

The only treatment for proven hepatitis C exposure is

---

**TABLE 75-1** -- Provisional Public Health Service Recommendations for Chemoprophylaxis After Occupational Exposure to HIV, by Type of Exposure and Source Material--1996

| Type of Exposure | Source Material * | Antiretroviral Prophylaxis | Antiretroviral Regimen |
|---|---|---|---|
| Percutaneous | Blood § | | |
| | Highest risk | Recommend | ZDV plus 3TC plus IDV |
| | Increased risk | Recommend | ZDV plus 3TC, ± IDV |
| | No increased risk | Offer | ZDV plus 3TC |
| | Fluid containing visible blood, other potentially infectious fluid, ¶ or tissue | Offer | ZDV plus 3TC |
| | Other body fluid (e.g., urine) | Not offer | |
| Mucous membrane | Blood | Offer | ZDV plus 3TC, ± IDV |
| | Fluid containing visible blood, other potentially infectious fluid, ¶ or tissue | Offer | ZDV, ± 3TC |
| | Other body fluid (e.g., urine) | Not offer | |

| Skin, increased risk* [*] | Blood | Offer | ZDV plus 3TC, ± IDV |
|---|---|---|---|
| | Fluid containing visible blood, other potentially infectious fluid, [¶] or tissue | Offer | ZDV, ± 3TC |
| | Other body fluid (e.g., urine) | Not offer | |

From Centers for Disease Control and Prevention: Provisional Public Health Service recommendations for chemoprophylaxis after occupational exposure to HIV. MMWR 45:468, 1996.

immune globulin administered in a dose of 0.06 mL/kg IM as soon as possible after the exposure. [39] However, the effectiveness of this regimen has not been established. Currently, there is no vaccine for HCV.

**Tuberculosis Exposure**

The health care worker exposed to tuberculosis who has a negative skin test within the previous year requires interval testing (generally in 8 to 12 weeks) to determine potential conversion during the exposure. Those without a prior conversion, but >1 year since their last negative skin test, also require an immediate skin test to identify any prior interval conversion. Treatment after skin test conversion using antituberculous agents depends on the patient's age, underlying health, and other factors. The health care worker who is exposed, but has a previous positive tuberculosis skin test, would be considered for treatment depending on any subsequent signs or symptoms of the disease.